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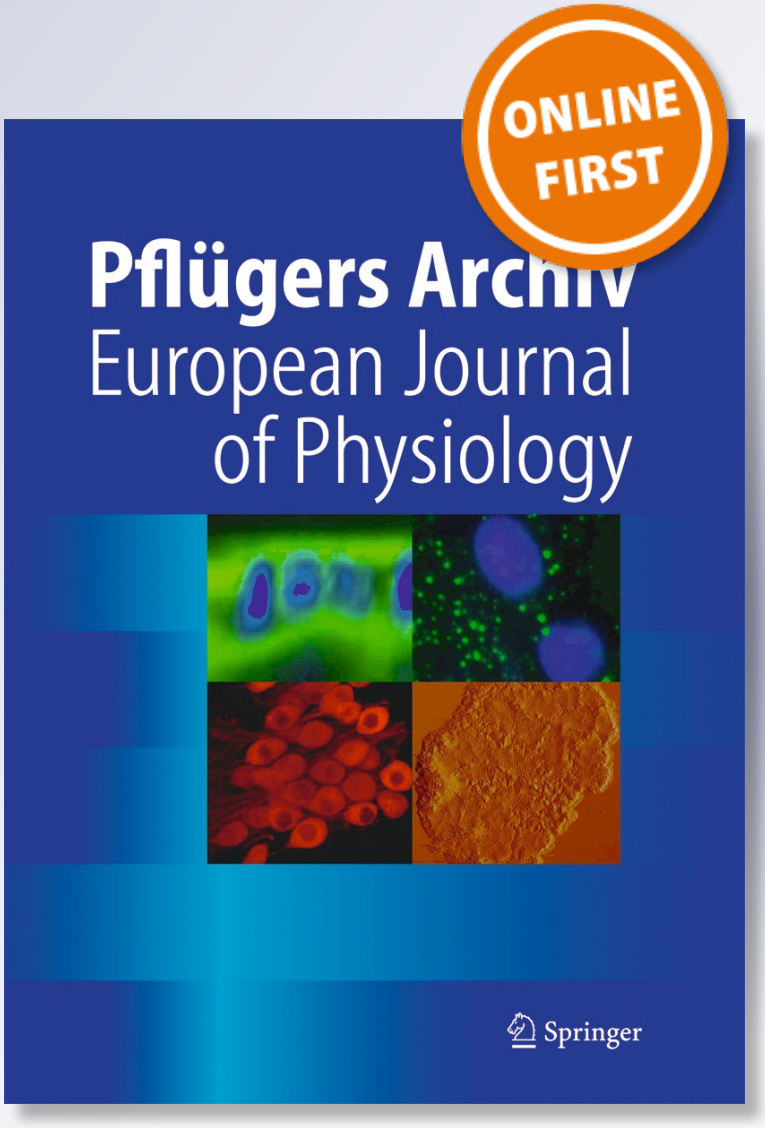

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INVITED REVIEW

Salt and sugar: their effects on blood pressure

Feng J. He · Graham A. MacGregor

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Abstract Both dietary salt and sugar are related to blood pressure (BP). The evidence for salt is much stronger, and various types of studies have consistently shown that salt is a major cause of raised BP, and a reduction from the current intake of ≈9–12 g/day in most countries of the world to the recommended level of 5–6 g/day lowers BP in both hypertensive and normotensive individuals, in men and women, in all age groups and in all ethnic groups. Countries such as Finland and the UK that have successfully reduced salt intake have demonstrated a reduction in population BP and cardiovascular mortality, with major cost savings to the health service. The mechanisms whereby salt raises BP are not fully understood. The traditional concepts focus on the tendency for an increase in extracellular fluid volume. Increasing evidence suggests that small increases in plasma sodium may play an important role. There are several other factors that also increase BP, one of which is added sugars. The current high intake of added sugars increases obesity which, in turn, raises BP. Recent studies also suggest that added sugars, particularly those in soft drinks, may have a direct effect on BP. However, the relationship between soft drink consumption and BP could be, at least partially, mediated by the effect of salt intake on increasing soft drink consumption. Actions to reduce salt and sugar intake across the whole population will have major beneficial effects on health along with major cost savings.

Keywords Salt intake · Added sugar consumption · Blood pressure · Cardiovascular risk

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Added salt and sugar are not part of mammalian or human diet. Indeed, there is absolutely no requirement for adding either to human diet. Salt was only added to food about 5000 years ago, when the Chinese discovered that salt could be used to preserve food. Salt then became of great economic importance. It became the most taxed and traded commodity in the world, with intake reaching a peak around the 1870s [55]. Salt intake then declined with the invention of the deep freezer and the refrigerator as salt was no longer required as a preservative. However, with the recent large increase in the consumption of processed, restaurant and fast food, salt intake is now increasing again.

Added sugar in human diet is a very recent phenomenon (c. 200 years) and only occurred when sugar obtained from sugar cane became very cheap to produce. This added sugar is a totally unnecessary source of calories and gives no feeling of fullness. It is well known that most soft drinks are high in sugar; however, many processed foods also contain large amounts of hidden sugars.

The added salt and added sugars have many harmful effects on health [35, 56]. For example, they increase blood pressure (BP) and obesity, both of which increase the risk of cardiovascular disease (CVD), the leading cause of death and disability worldwide. The evidence that relates salt to BP is much stronger than sugar. In this article, we will provide an update on evidence that relates salt to BP and CVD, as well as the potential mechanisms whereby salt increases BP, in particular, the role of plasma sodium. Additionally, we will briefly review the recent studies that relate added sugars to BP.

Salt and BP

Various types of studies including animal experiments, human genetics, epidemiology, migration, population-based intervention studies and treatment trials have consistently shown that

dietary salt intake is a major cause of raised BP [1, 24, 34, 35, 45, 66]. Several large epidemiological studies have shown that salt intake is directly related to BP [45, 49, 92]. The INTERSALT study [45] which enrolled 10,079 individuals from 52 centres around the world also demonstrated a highly significant positive relationship between salt intake and the increase in BP with age. It was estimated that an increase of 6 g/day in salt intake over 30 years would lead to an increase in systolic BP by 9 mmHg [45]. 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 four communities consuming lower salt were excluded, there was no overall relationship remaining between salt intake and BP. The INTERSALT's investigators re-analysed their data and showed that the highly significant within-population association between salt intake and BP across all 52 centres was virtually unchanged when the four low-salt populations were excluded, and the association between salt intake and the rise in BP with age persisted across 48 centres [22, 23, 45].

Population-based intervention studies have shown that when salt intake decreased, there was a reduction in population BP [24, 79]. One of the most successful intervention studies was conducted in two similar villages in Portugal [24] where salt intake was very high (≈21 g/day) and the prevalence of hypertension was also very high. During 2 years' intervention through vigorous, widespread health education to reduce the consumption of salt especially from foods that had previously been identified as the major sources of salt, there was a difference of ≈50 % in salt intake between the two villages (i.e., intervention vs. control). This was associated with a difference of 13/6 mmHg in BP. Another community-based intervention trial in two rural villages in northeastern Japan reduced salt intake by 2.3 g/day through dietary counselling. This reduction in salt intake was associated with a decrease of 3.1 mmHg in systolic BP [76]. Several other studies [71, 80] showed no significant change in BP; however, these studies failed to achieve a reduction in salt intake, and such results are therefore not surprising.

There have been a large number of clinical trials looking at the effect of salt reduction on BP. Recent meta-analyses of randomised trials demonstrate that a longer-term modest reduction in salt intake, as currently recommended, results in significant and, from a population viewpoint, important falls in BP in both hypertensive and normotensive individuals, in men and women, in all age groups and in all ethnic groups although there is a variation in the extent of the fall in BP among different groups [1, 34]. Importantly, there is a dose-response relationship and, within the range of 12 to 3 g/day, the greater the reduction in salt intake, the greater the fall in BP [34].

The most persuasive evidence on the dose-response relationship comes from rigorously controlled trials with multiple levels of salt intake [57, 67]. One of such trials was the randomised double-blind crossover study in 20 individuals with untreated essential hypertension, where salt intake was reduced from 11.2 to 6.4 and to 2.9 g/day, each for 1 month [57]. BP was 163/100 mmHg with a salt intake of 11.2 g/day and reduced to 155/95 mmHg when salt intake decreased to 6.4 g/day (i.e. a decrease of 8/5 mmHg). BP fell further to 147/91 mmHg when salt intake reduced to 2.9 g/day (i.e. a further fall of 8/4 mmHg) [57]. This well-controlled study in hypertensive individuals clearly showed that the lower the salt intake achieved, the lower the BP. The Dietary Approaches to Stop Hypertension (DASH)-sodium trial [67] which studied 412 individuals with normal or mildly raised BP also demonstrated a clear dose-response relationship when salt intake was reduced from 8 to 6 to 4 g/day. The fall in BP was greater at a lower level of salt intake, i.e. from 6 to 4 g/day compared with that from 8 to 6 g/day. It is well established that the DASH diet which is rich in fruits, vegetables and low-fat dairy products lowers BP [3]. However, the dose-response relationship between salt intake and BP was observed both on the normal American diet and the DASH diet (Fig. 1) [67]. The lowest BP

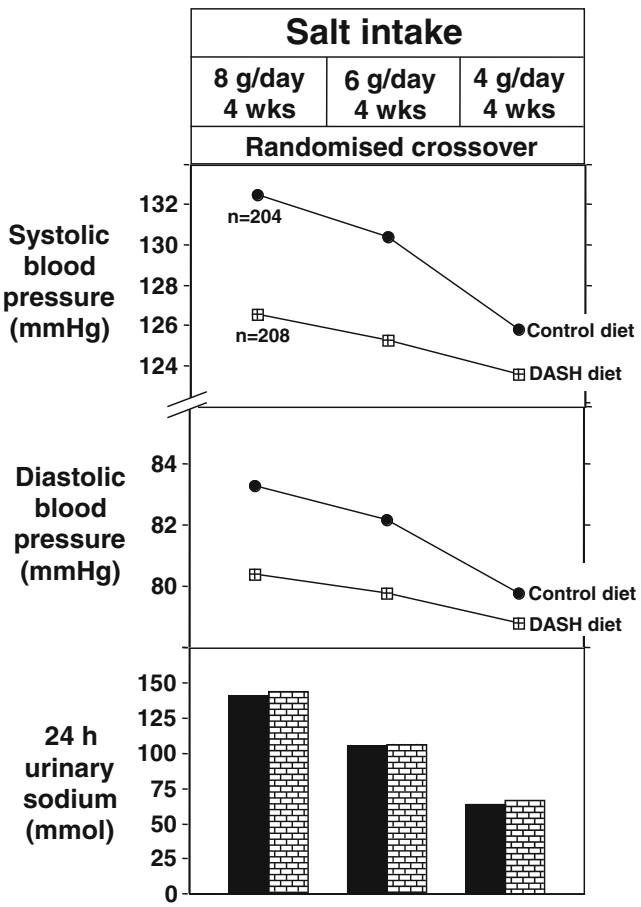

Fig. 1 Changes in blood pressure and 24-h urinary sodium excretion with the reduction in salt intake in all participants (hypertensives, N=169; normotensives, N=243) on the normal American diet (i.e. control diet) and on DASH diet. Redrawn from ref. [67]

occurred in those who were on the DASH diet with the lowest level of salt intake, indicating an additive effect of lower salt and higher fruit and vegetable intake on BP.

From the well-controlled trials, it is clear that the current recommendations to reduce salt from 9–12 g/day in most countries of the world to the recommended level of 5–6 g/day will have a major effect on BP, but are not ideal. A further reduction to 3 g/day will have a much greater effect. Therefore, 3 g/day should become the long-term target for population salt intake. It is important to note that the current recommendation is based on the feasibility of reducing population salt intake to 5–6 g/day, but not on the potential maximum beneficial effects of salt reduction. Recently, the UK government's health advisory agency, the National Institute for Health and Care Excellence (NICE) has recommended a reduction in the population's salt consumption to 3 g/day by 2025 [61]. In the USA, it is recommended that salt intake should be reduced to less than 6 g/day for adults, with an even further reduction to 4 g/day for about half the population, including African Americans, all adults 51 and older, and those with hypertension, diabetes or chronic kidney disease [82].

It has been shown that, for a given reduction in salt intake, the fall in BP was larger in individuals of African origin, in older people and in those with raised BP compared to whites, young people and individuals with normal BP, respectively [83]. These differences in the fall in BP were, at least in part, due to the differences in the responsiveness of the renin-angiotensin system [41]. 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. Such studies are irrelevant to the public health recommendations of more modest reduction in salt intake for a prolonged period of time. There is strong evidence that a modest reduction in salt intake should be carried out universally in the entire population [35, 37]. 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 cardiovascular disease [88].

Salt and cardiovascular disease

There is much evidence that BP throughout its range starting at 115/75 mmHg is a major cause of CVD [53]. A modest reduction in salt intake lowers BP and, therefore, would reduce cardiovascular risk. Based on the fall in BP from a meta-analysis of randomised salt reduction trials [38], it was estimated that a reduction of 6 g/day in salt intake would reduce stroke by 24 % and ischaemic heart disease (IHD) by 18 %. This would prevent ≈35,000 stroke and IHD deaths a year in the UK [39] and ≈2.5 million deaths worldwide. A recent modelling study of global salt consumption and deaths showed that approximately 1.65 million CVD deaths that occurred in 2010 were attributed to salt consumption above the WHO recommended level of 5 g/day. Four of every five deaths (84.3 %) occurred in developing countries, and two of every five deaths (40.4 %) were premature (before 70 years of age) [59].

Meta-analyses of prospective cohort studies have shown that a lower salt intake is related to a reduced risk of CVD [73]. However, a few more recent cohort studies have reported a U-shaped association, with a lower or higher salt intake both being associated with higher CVD or all-cause mortality [27, 62, 72]. These studies have created substantial controversy, particularly as they were publicised by the Salt Industry public relations body, the Salt Institute. However, there are severe methodological flaws with these studies. Two recent papers from the Science Advisory of the American Heart Association (AHA) [12, 87], along with several other papers [9, 13, 32], have provided detailed analysis of the methodological issues in cohort studies, e.g. reverse causality, residual confounding, errors in salt assessment, particularly, the inherent problem of estimating individuals' usual salt intake as it varies by a large amount from day to day. Due to the methodological problems, these studies cannot be used to inform public health policy on salt.

Evidence from outcome trials of long-term salt reduction is very limited due to the innate difficulty in conducting such trials. Six publications in patients with severe heart failure on multiple drug treatments claimed that randomised trials showed that salt reduction had no benefits or increased mortality or rehospitalisation. All of these papers were from the same group of researchers, and the integrity of their data has now been seriously undermined with a recent meta-analysis of these studies being retracted from the journal *Heart* after an investigation by BMJ Publishing Ethics Committee [20], and another meta-analysis by Taylor et al. has also been withdrawn from *The Cochrane Library* [77] due to the inclusion of the trial in heart failure as well as other methodological problems.

A re-analysis of Taylor's meta-analysis by excluding the trial in heart failure and combining hypertensives and normotensives together shows that there is a significant reduction in cardiovascular events by 20 % ($P<0.05$) (Fig. 2) and a non-significant reduction in all-cause mortality (5–7 %), in spite of the small reduction in salt intake of 2.0–2.3 g/day. These results provide further support for a reduction in population salt intake [36].

Several cost-effective analyses have shown that a reduction in salt intake not only saves lives but also saves money. Indeed, it is one of the most cost-effective measures to reduce CVD in both developed and developing countries [4, 6, 47, 60, 64, 69, 70]. For instance, a recent study in the US showed that even a very modest reduction in salt intake of only 10 %

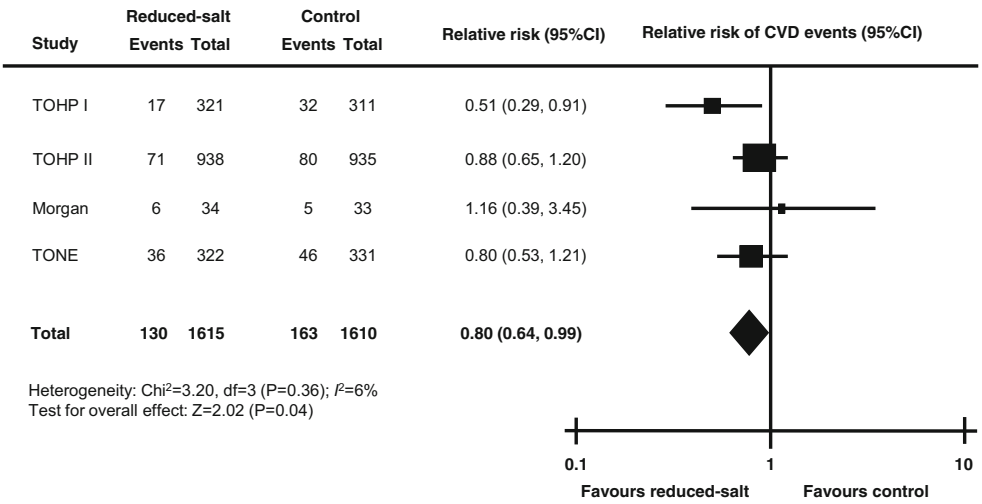

Fig. 2 Relative risk of cardiovascular disease (CVD) events in a meta-analysis of salt reduction trials. *TOHP I*, trial of hypertension prevention, phase 1. *TOHP II*, trial of hypertension prevention, phase 2. *TONE*, trial of nonpharmacologic interventions in elderly

which could be easily achieved, as demonstrated in the UK [21], would prevent hundreds of thousands of strokes and heart attacks over the lifetimes of adults aged 40–85 years who are alive today and could save more than $32 billion in medical expenses in the US alone [70]. A larger decrease in salt intake would result in a larger health improvement and greater cost savings [6]. The UK salt reduction campaigns, which have been successful in reducing population salt intake, have prevented ≈9000 stroke and IHD deaths per year and resulted in annual healthcare savings of ≈£1.5 billion [61].

Asaria et al. estimated the effects and cost of strategies to reduce salt intake and control tobacco for 23 developing countries that account for ≈80 % of chronic disease burden in the developing world [4]. They demonstrated that, over 10 years, 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 programmes was estimated to be US$0.09 per person per year. The cost for tobacco control including both price and non-price measures was US$0.26 per person per year [4]. 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 terms of reducing CVD.

Successful experience of reducing population salt intake

So far, there are only three countries (i.e. Japan, Finland and the UK) that have successfully reduced salt intake. Japan, in the late 1960s, carried out a government-led campaign to reduce the amount of salt used by households as it was realised that the high stroke mortality in Japan was directly related to the high salt intake in the population. Over the following decade, salt intake was reduced, particularly in northern areas from 18 to 14 g/day. Paralleling this reduction in salt intake, there were falls in BP and an 80 % reduction in stroke mortality [68] in spite of large increases in fat intake, cigarette smoking, alcohol consumption and obesity that occurred during that period. Finland, in the late 1970s, initiated a systematic approach to reducing salt intake through mass media campaigns, co-operation with the food industry and implementing salt-labelling legislation [48, 51, 65]. This led to a significant reduction in the average salt intake of the Finnish population [48, 51] from ≈14 g/day in 1972 to less than 9 g/day in 2002 [48]. The reduction in salt intake was accompanied by a fall of over 10 mmHg in systolic and diastolic BP and a decrease of 75–80 % in both stroke and IHD mortality [48]. Although these results were attributable to several factors, the reduction in salt intake is likely to have played a major role, particularly in the fall in BP as both body mass index and alcohol consumption increased during that time.

More recently, the UK, through Consensus Action on Salt and Health (CASH) [10], a nongovernmental organisation, and the Food Standards Agency (FSA), a quasi-government organisation, has successfully developed and implemented a programme of voluntary salt reduction in collaboration with the food industry [33]. The main components of the UK salt reduction programme are summarised in Fig. 3 [33]. The key element is the rigorous setting of progressively lower salt targets for over 80 categories of foods, with a clear timeframe and independent monitoring programme. Significant progress has been made since the start of the salt reduction programme in 2003/2004. The salt content in many food products have been reduced by 20 to 40 % [7, 33]. These reductions have been made slowly, and there have been no loss of sales to the food industry, and the public are largely unaware of these

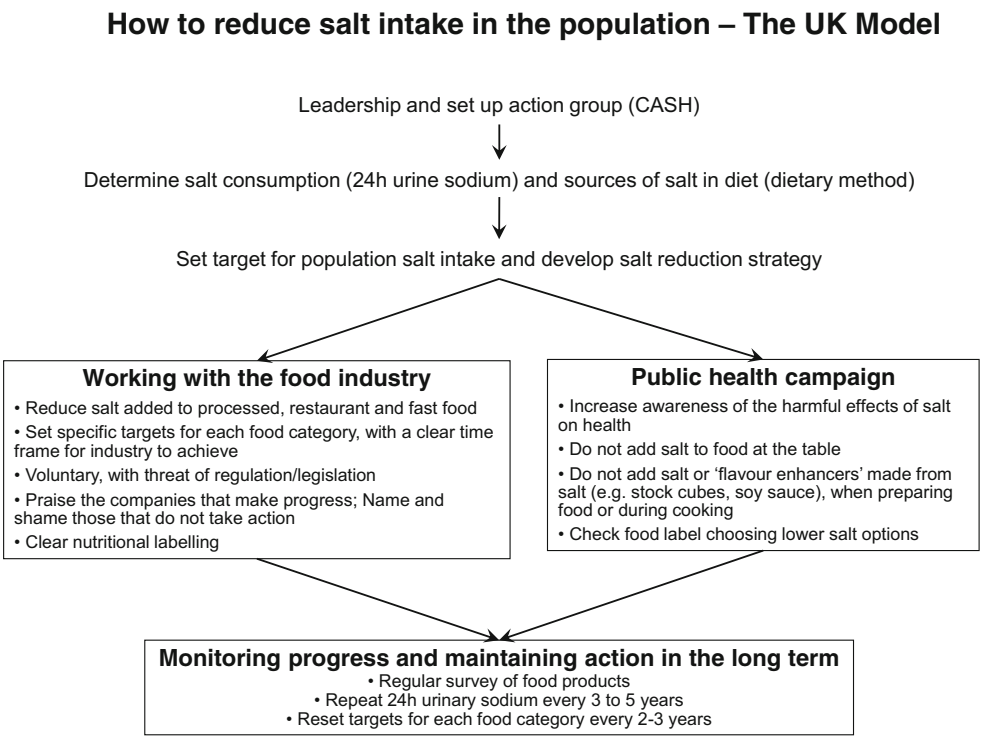

Fig. 3 An action framework of reducing salt intake in the population—the UK model

reductions. The average salt intake as measured by 24 urinary sodium excretion in a random sample of the adult population has been reduced by 15 %, i.e. from 9.5 g/day in 2003 to 8.1 g/day in 2011 [21]. This was accompanied by a significant fall in population BP and mortality from stroke and IHD (Fig. 4) [44].

Several developed countries such as the US, Canada and Australia are following the UK's lead and setting their own targets. Many developing countries, however, are lagging behind. In most developing countries, the major sources of salt consumption are additions during cooking and in sauces, spice mixes, seasonings, pickles, etc. rather than prepackaged prepared foods [2]. Public health campaigns are needed to encourage people to use less salt in their own food preparations.

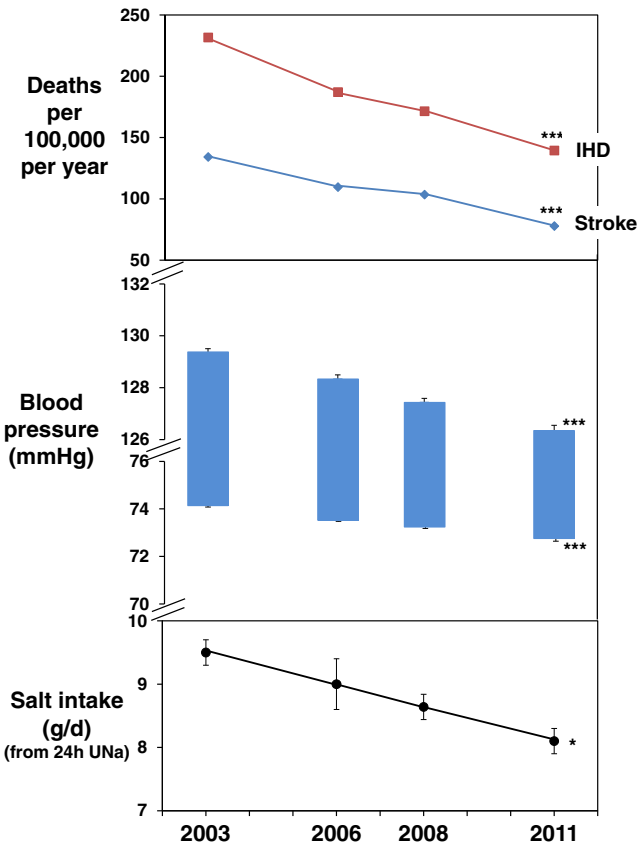

Fig. 4 Changes in salt intake as measured by 24-h urinary sodium (UNa) excretion, blood pressure (BP), stroke and ischemic heart disease (IHD) mortality in England from 2003 to 2011. *$P<0.05$, ***$P<0.001$ for trend

Potential mechanisms whereby salt increases BP

Role of extracellular volume

The mechanisms by which salt increases BP are not fully understood. There is much evidence that the kidney plays an important role [14, 15]. In individuals who develop high BP, there is an underlying defect in the kidneys' ability to excrete sodium. This 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 which in turn helps overcome the kidneys' difficulties in excreting sodium. Based on experiments in 70 % nephrectomised dogs given large amounts of saline intravenously daily for 2 weeks, Guyton suggested that volume expansion raises BP by the autoregulatory effect on resistance vessels [31].

Role of plasma sodium

There is increasing evidence that small changes in plasma sodium may be an important mechanism for the changes in BP with changing salt intake. Several epidemiological studies have shown a significant positive association between plasma sodium and BP [8, 50, 84]. In a study of 3578 London civil servants, a 1 mmol/L increase in plasma sodium was associated with a 1 mmHg increase in systolic BP after adjusting for confounding factors [8]. In another study of a Japanese population (3222 normotensives and 741 individuals with essential hypertension), serum sodium distribution was shifted by ≈2 mmol/L towards higher values in the hypertensives [50]. The Framingham Heart Study (2172 nonhypertensive Framingham Offspring Study participants) showed that serum sodium was not associated with BP cross-sectionally or with the development of hypertension during 4 years follow-up [52]. However, the sample size of the Framingham Heart Study was smaller compared with the other two epidemiological studies [8, 50].

Plasma sodium is a major determinant of extracellular volume and thereby influencing BP. At the same time, small changes in plasma sodium may have a direct effect on BP, independent of extracellular volume [16]. Using peritoneal dialysis in rats, Friedman et al. were able to change plasma sodium in an opposite direction to extracellular volume by altering sodium concentration of the dialysis fluid [26]. When plasma sodium was increased by 10–15 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. suggested that increases in intracellular sodium may affect vascular smooth muscle tension and thereby increasing BP [25, 26]. In humans, it is difficult to study the effects of changes in plasma sodium without there being an associated change in extracellular volume. A recent study in patients on haemodialysis indicated that acute changes in plasma sodium could have an immediate and direct effect on BP [75]. Ten individuals were studied, in random order, on two separate haemodialysis sessions, one with dialysate sodium set at 145 mmol/L and the other at 135 mmol/L, each for 2 h with no ultrafiltration [75]. This reduction in dialysate sodium concentration resulted in a significant decrease in plasma sodium of 3.4±0.3 mmol/L at 1 h ($P<0.01$) and 4.8 ±0.3 mmol/L at 2 h ($P<0.001$). This was associated with a fall in systolic BP of 8±3 mmHg ($P<0.05$) at 1 h and 12±5 mmHg ($P=0.05$) at 2 h. During the study, there was no fluid removed and there was no significant change in haematocrit which is a crude index of extracellular fluid volume. These results suggest that changes in plasma sodium could have a direct effect on BP although small changes in extracellular volume due the movements of fluid between intracellular and extracellular compartment could not be ruled out.

A number of studies have shown that an increase or a decrease in salt intake causes parallel changes in plasma sodium in both hypertensive and normotensive individuals [40, 74], e.g. a decrease of ≈3 mmol/L ($P<0.001$) in plasma sodium when salt intake was reduced from 20 to 1 g/day for 5 days. In a well-controlled double-blind trial of 1 month, plasma sodium fell by 0.4 mmol/L ($P<0.05$) when salt intake decreased from ≈10 to 5 g/day in 118 hypertensive individuals. The decrease in plasma sodium was weakly but significantly correlated with the fall in systolic BP that occurred with the reduction in salt intake [40].

Tissue culture experiments demonstrated that increasing bath sodium concentration within the physiological range caused marked cellular hypertrophy in both arterial smooth muscle and cardiac myocytes [30]. 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 to 157 mmol/L) [54]. Using cultured human endothelial cells, Oberleitner et al. demonstrated that an increase in the sodium concentration of the culture medium from 135 to 145 mmol/L stiffened the endothelium and reduced nitric oxide release [63].

Recent studies in humans have shown that a reduction in salt intake improves endothelial function [17, 19, 81]. A randomised crossover trial in 25 overweight and obese normotensive individuals showed that when salt intake was reduced from 9 to 6 g/day for 6 weeks, there was a significant improvement in endothelial function as measured by brachial artery flow-mediated dilatation [17]. Other studies showed that a high salt meal has an immediate adverse effect on endothelial function in normotensive individuals [18].

Added sugars and BP

It is well established that high sugar consumption increases obesity which, in turn, increases BP. There is now an emerging but inconclusive body of evidence that added sugars, particularly those in soft drinks, may have a direct effect on BP independent of obesity [11, 78]. A recent systematic review and meta-analysis of randomised trials showed that a higher sugar intake was associated with a higher systolic BP, but this effect was only significant in the three trials with duration of more than 8 weeks and there was no significant effect of higher

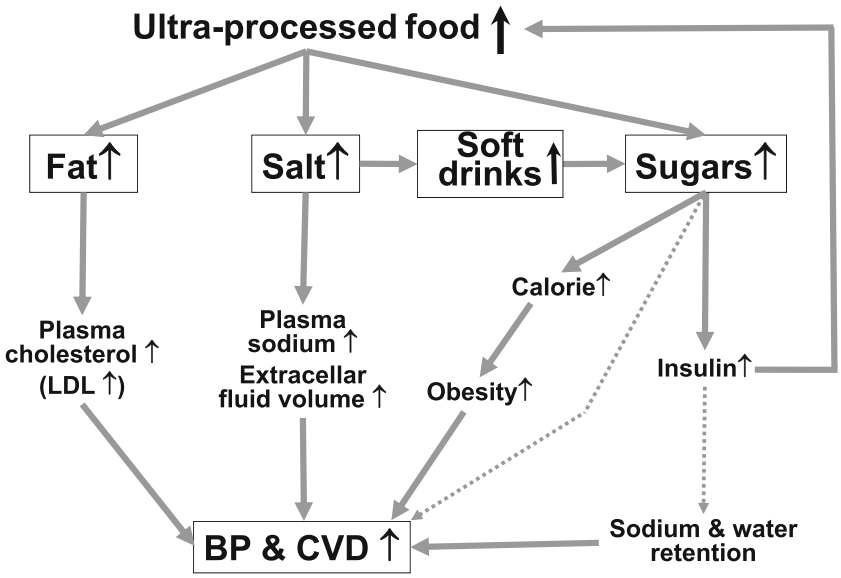

Fig. 5 Possible links between salt, soft drinks, sugars and blood pressure (BP)

sugar intakes on systolic BP when all of the 12 trials were included [78]. A meta-analysis of three large prospective cohort studies of US health professionals showed that total fructose intake was not associated with an increased risk of high BP [46].

Soft drinks and other sugar-sweetened beverages are the major source of added sugars in the diet. A recent review of 12 studies with 409,707 participants (6 were cross-sectional studies, and the rest were prospective cohort studies) [58] showed that sugar-sweetened soft drink consumption was associated with increased BP. However, the underlying mechanism for this association could be, at least partially, attributable to salt consumption. There is clear evidence for a causal relationship between salt intake and thirst and thereby total fluid consumption [42], including sugar-sweetened soft drinks [43]. A carefully controlled metabolic study in adult humans where salt intake was changed has quantified the relationship between the change in salt intake and the subsequent change in fluid consumption [42]. A study in 10,074 free-living individuals across the world showed an identical relationship between usual salt and fluid consumption [42]. An analysis of the National Diet and Nutrition Survey dataset showed that in free-living children in Great Britain, salt intake was significantly related to total fluid, as well as sugar-sweetened soft drink consumption [43]. Similar findings have also been reported in the USA and Australia [28, 29]. Taking together, these studies suggest that in humans, like other mammals, salt is a major drive to thirst, and an increase in salt intake increases the amount of fluid consumed, and if part of this fluid is in the form of soft drinks, they will be increased proportionately. It is therefore likely that the observed association between sugar-sweetened soft drink consumption and BP is, at least, partially mediated by salt intake. At the same time, an increase in sugar intake stimulates insulin secretion, which in turn increases the consumption of food. A greater intake of ultra-processed food will increase salt, fat and sugar consumption and thereby increasing BP and CVD risk. The suggested mechanism for the links between salt, soft drinks, sugars and BP, as well as a possible vicious circle between salt and sugar intake, are summarised in Fig. 5.

Conclusions

Both added salt and sugars have an effect on BP. However, the evidence for salt is much stronger [35]. Indeed, salt reduction is one of the most cost-effective measures to prevent hypertension and CVD worldwide [4, 6, 61]. At the 2011 UN high-level meeting on non-communicable diseases (NCDs), salt reduction was recommended as one of the top three priority actions (along with a reduction in smoking and alcohol consumption) to reduce premature mortality from NCDs by 25 % by 2025 [5, 89]. The WHO in its recent guideline recommends a 30 % reduction in salt intake by 2025 with an eventual target of 5 g/day for all adults worldwide and lower levels for children based on calorie intake [90]. Following on from this, member states at the 66th World Health Assembly formally adopted these WHO salt targets as part of an omnibus resolution to tackle NCDs. Most countries are now adopting a policy of reducing salt intake by persuading the food industry to reformulate food with less salt, as is occurring successfully in the UK with an accompanying fall in population BP and CVD mortality [44]. Several countries such as the US, Canada and Australia are following the UK's lead and setting their own targets [86]. The major challenge now is to spread this out to all other countries. In most developing countries where the majority of salt in the diet is added by the consumers, a public health campaign plays a major role. World Action on Salt and Health (WASH) [85], a similar group to CASH with

over 500 members in 98 countries, is encouraging action groups to be formed in each country. All countries should adopt a coherent and workable strategy to reduce salt intake.

A reduction in the consumption of added sugars will provide additional beneficial effects on BP and also have many other health benefits [56]. The WHO in its recently released draft guidelines on sugar intake for consultation (March 2014) stated that a reduction in added sugars from the current level of ≈13 % of total energy intake per day to below 5 % (i.e. below 25 g or six teaspoons) would be very beneficial to health. An action group (Action on Sugar) [56] was recently set up with a mission to achieve a gradual and sustained reduction in the amount of sugars added to foods and drinks, following a similar model to salt reduction pioneered by Consensus Action on Salt and Health (CASH) [10]. This model has become one of the most successful nutritional policies in the UK since the Second World War [91], by setting targets for the food industry to reduce the amount of salt added to all of their products, over a period of time [33]. As this is done slowly, people do not notice the difference in taste. A reduction in population salt and sugar intake, even by a small amount, will have a major beneficial effect on health along with major cost savings.

Conflict of interest FJH is a member of Consensus Action on Salt and Health (CASH) and World Action on Salt and Health (WASH). Both CASH and WASH are non-profit charitable organisations, and FJH does not receive any financial support from CASH or WASH. GAM is the chairman of the Blood Pressure UK (BPUK), chairman of CASH, WASH and Action on Sugar. BPUK, CASH, WASH and Action on Sugar are non-profit charitable organisations. GAM does not receive any financial support from any of these organisations.

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