Reduced dietary salt for prevention of cardiovascular disease (Unknown)

Hooper L, Bartlett C, Davey SG, Ebrahim S

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Reduced dietary salt for prevention of cardiovascular disease

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\section*{A B S T R A C T}

\textbf{Background}

Restricting sodium intake in elevated blood pressure over short periods of time reduces blood pressure. Long term effects (on mortality, morbidity or blood pressure) of advice to reduce salt in patients with elevated or normal blood pressure are unclear.

\textbf{Objectives}

To assess in adults the long term effects (mortality, cardiovascular events, blood pressure, quality of life, weight, urinary sodium excretion, other nutrients and use of anti-hypertensive medications) of advice to restrict dietary sodium using all relevant randomised controlled trials.

\textbf{Search strategy}

The Cochrane Library, MEDLINE, EMBASE, bibliographies of included studies and related systematic reviews were searched for unconfounded randomised trials in healthy adults aiming to reduce sodium intake over at least 6 months. Attempts were made to trace unpublished or missed studies and authors of all included trials were contacted. There were no language restrictions.

\textbf{Selection criteria}

Inclusion decisions were independently duplicated and based on the following criteria: 1) randomisation was adequate; 2) there was a usual or control diet group; 3) the intervention aimed to reduce sodium intake; 4) the intervention was not multifactorial; 5) the participants were not children, acutely ill, pregnant or institutionalised; 6) follow-up was at least 26 weeks; 7) data on any of the outcomes of interest were available.

\textbf{Data collection and analysis}

Decisions on validity and data extraction were made independently by two reviewers, disagreements were resolved by discussion or if necessary by a third reviewer. Random effects meta-analysis, sub-grouping, sensitivity analysis and meta-regression were performed.

\textbf{Main results}

Three trials in normotensives (n=2326), five in untreated hypertensives (n=387) and three in treated hypertensives (n=801) were included, with follow up from six months to seven years. The large, high quality (and therefore most informative) studies used intensive behavioural interventions.

Deaths and cardiovascular events were inconsistently defined and reported; only 17 deaths equally distributed between intervention and control groups occurred. Systolic and diastolic blood pressures were reduced at 13 to 60 months in those given low sodium advice as compared with controls (systolic by 1.1 mm Hg, 95\% CI 1.8 to 0.4, diastolic by 0.6 mm hg, 95\% CI 1.5 to -0.3), as was urinary 24
hour sodium excretion (by 35.5 mmol/24 hours, 95% CI 47.2 to 23.9). Degree of reduction in sodium intake and change in blood pressure were not related. People on anti-hypertensive medications were able to stop their medication more often on a reduced sodium diet as compared with controls, while maintaining similar blood pressure control.

Reviewer’s conclusions

Intensive interventions, unsuited to primary care or population prevention programmes, provide only minimal reductions in blood pressure during long-term trials. Further evaluations to assess effects on morbidity and mortality outcomes are needed for populations as a whole and for patients with elevated blood pressure.

Evidence from a large and small trial showed that a low sodium diet helps in maintenance of lower blood pressure following withdrawal of antihypertensives. If this is confirmed, with no increase in cardiovascular events, then targeting of comprehensive dietary and behavioural programmes in patients with elevated blood pressure requiring drug treatment would be justified.

**PLAIN LANGUAGE SUMMARY**

**Synopsis**

This review set out to assess the long term effects of advice to cut down on salt in food on deaths, cardiovascular disease and blood pressure in adults.

Intensive support and encouragement to reduce salt intake did lead to reduction in salt eaten. It also lowered blood pressure but only by a small amount (about 1 mmHg for systolic blood pressure, less for diastolic) after more than a year. This reduction was not enough to expect an important health benefit. It was also very hard to keep to a low salt diet. However, the reduction in blood pressure appeared larger for people with higher blood pressure.

There was not enough information to assess the effect of these changes in salt intake on health or deaths.

Evidence from a large and small trial showed that advice to reduce salt helps to maintain lower blood pressure following withdrawal of antihypertensive medication. If this is confirmed, with no increase in cardiovascular events, then comprehensive dietary and behavioural programmes in patients with elevated blood pressure requiring drug treatment would be justified.

See also the Cochrane review of short-term salt reduction trials: Jurgens 2003.

**BACKGROUND**

There is evidence from published systematic reviews that restricting sodium intake in people with elevated blood pressure leads to reductions in blood pressure of about 4 mmHg systolic and 2 mmHg diastolic (Law 1991; Midgley 1996; Cutler 1997; Graudal 1998; Alam 1999; Jurgens 2003). However, within these reviews many included trials are short term, neither allowing for complete adjustment of blood pressure to altered sodium intake or to reduced motivation for following dietary restrictions over time. Also, some trials increased sodium intake in one arm and compared this with a reduced sodium intake in the other arm and so do not estimate likely effects of cutting down on sodium in a normal diet.

In addition, some reviews suggest that the level of blood pressure reduction achieved over a longer period in free-living adults is less impressive than in the short term (Ebrahim 1996; Ebrahim 1998; Graudal 1998).

A decrease in blood pressure is only important if it results in a decrease in cardiovascular events and deaths. The published systematic reviews on the effect of salt restriction on blood pressure and other risk factor outcomes have expressed different interpretations with regard to the significance of these changes in relation to cardiovascular events and deaths. This systematic review and meta-analysis aimed to draw together information on the effect of long-term dietary salt reduction on health outcomes.

**OBJECTIVES**

This systematic review aimed to study the effects of restricting
sodium intake over at least six months in free-living adults, compared with a normal or usual sodium intake.

The specific objectives were to assess, in people with normal and elevated blood pressure, the effect of advice and/or support to reduce dietary sodium intake, on deaths and cardiovascular events; number and dose of anti-hypertensive medications used; quality of life; weight; systolic and diastolic blood pressure; and urinary sodium excretion and other nutrient intakes in free-living adults at least six months after the initial intervention was commenced.

The effects of potential modifiers of salt restriction (i.e. initial level of blood pressure, categorization into normal or elevated blood pressure, degree of sodium reduction, gender, race and age) were also investigated.

RESULTS

Mortality and cardiovascular events.

These outcomes were inconsistently reported in trials (see Data - total deaths and cardiovascular events (including cardiovascular deaths)). No differences in periods of hospitalisation were seen between intervention groups in the HPT study (no further data were provided). Morgan 1978 reported that three control participants were treated for cardiac failure, as were two on low sodium diets, with four cardiovascular deaths in the low sodium group and two in the control group. TONE recorded cardiovascular events (including stroke, transient ischaemic attack, myocardial infarction, angina, congestive heart failure, arrhythmia and ‘other’ events) of participants. Cardiovascular events occurred in 46 control participants and 36 of those on low sodium diets. Pooling the two studies suggests no significant difference in cardiovascular morbidity between low sodium and control groups (relative risk 0.82, 95% CI 0.56 to 1.21).

The trials report few deaths, altogether only 9 deaths in control groups and 8 in low sodium groups (relative risk 0.90, 95% CI 0.36 to 2.24). The available data are shown in the metaview.

Blood pressure.

Changes in blood pressure and urinary sodium excretion at intermediate and late assessments are given in Data - BP & urinary sodium (‘mean (sd)’ for control / ‘mean (sd)’ for low salt) and meta-analysis results in Meta-analysis, subgrouping and sensitivity analysis results. Systolic blood pressure was reduced on a low salt diet at both intermediate (by 2.5 mm Hg, 95% CI 3.8 to 1.2) and late follow up (by 1.1 mm Hg, 95% CI 1.8 to 0.4). Diastolic blood pressure was also reduced at intermediate follow up (by 1.2 mm Hg, 95% CI 1.8 to 0.7), less so later (by 0.6 mm Hg, 1.5 to -0.3).

The few participants with very late follow up (seven years) had non-significant reductions in systolic (by 3.8 mm Hg, 95% CI 7.9 to -0.3) and diastolic (by 2.2 mm Hg, 95% CI 4.8 to -0.4) blood pressure. It should be noted that this late follow up of the TOHP phase I study was technically after the end of the trial. TOHP phase I ran for 18 months with a consistent intervention to help the low sodium group stick to a low sodium diet. The 7-year results are described as ‘posttrial’ results, and as 7 years follow up, and the trialists implied that they were assessing the long term effect of their 18-month intervention. We (as reviewers) felt that if the trial just stopped intervening, without altering the diets of either the intervention or control groups then we could include data from the later follow up (in many studies the intervention only happens once or twice at the beginning, but the effect is measured months later). The paper states that ‘after 18-months, there was no further contact with the trial participants to enhance the intervention effect’. We could not contact the reviewers to confirm that there were no suggested alterations to the diets of the participants after the eighteen month intervention, so the data are included here but with this note of caution.

Statistical heterogeneity was present for systolic blood pressure at intermediate follow up and diastolic blood pressure at late follow up, but was resolved when sensitivity analyses removed trials with inadequate or unclear allocation concealment, or with imputed standard deviations, or when trials were sub-grouped into normotensive or hypertensive at baseline.

Sensitivity analysis, excluding trials with inadequate allocation concealment, resulted in all trials on untreated hypertensives being removed. As these trials were small, the effect on pooled estimates of blood pressure change was minor. Adding in data for the weight reduction arms of factorial trials strongly reduced the effect of low sodium advice on blood pressure, and slightly reduced the effect on sodium excretion (Meta-analysis, subgrouping and sensitivity analysis results).

Meta-regression of blood pressure change up to 12 months using all trials with relevant data (or trials with adequate allocation concealment, effectively trials on normotensives) showed no relationship with change in urinary sodium excretion, baseline systolic blood pressure or age (Meta-regression results, effects on SBP at 6 to 12 months). However, the meta-analyses subgrouping by ‘normotensive’ or ‘hypertensive’ participants at baseline did suggest a consistently greater effects of salt restriction on blood pressure in hypertensives. Insufficient data were available of effects on specific races or genders to enable statistical exploration of these factors.

Quality of Life.

Information on quality of life was patchy, with no common outcome measures. HPT asked participants whether they were having problems with their diets. 69% of those in the low sodium group reported problems at some time during the 3 years of the trial, and problems were reported at 42% of clinic visits. Problems related to the diet being inconvenient, conflicting with schedules, lack of time for planning, and difficulty in adherence while eating out.

TOHP phase I reported psychological well-being scores. These
improved significantly in participants in the low sodium groups at 18 months compared with the non-intervention control group (p<0.01). It was stated that the improvement was generally consistent across race and sex subgroups but no further information was provided.

Thaler (Thaler men 1982; Thaler women 1982) reported that stopping adding salt at table was not difficult for participants, but many found cutting down on salt in cooking harder. The majority found their low salt bread (salt cut from 2.1% to 1.0% dry weight) and salt-free butter acceptable. Only 13% of participants reported their salt restricted diet as unpleasant or worse.

TONE found that the most common non-cardiovascular event recorded was headache: the low sodium group had a significant reduction in headaches as compared to the control group.

Thaler (Thaler men 1982; Thaler women 1982) asked about presence or absence of muscle cramps in control and low sodium participants. At eight months 13% of control subjects reported getting cramps a lot or sometimes (as opposed to occasionally or never) whilst this outcome was reported in 30% of the low sodium group.

Overall drop out rates were very similar (relative risk 1.04, 95% CI 0.86 to 1.25) in low sodium compared with control groups.

Weight.

The suggestion from food diaries in HPT was that men on a low sodium diet take in roughly 240 kcal less per day than their control counterparts. Women on low sodium diets take in 120 fewer kcal per day. This did not result in a large difference in weight; at 3 years those in the control group had gained about 1 kg on average more than those in the low sodium group.

TOHP phase I observed significantly greater weight loss in the low sodium group compared with control at six (1.2 kg) and twelve (0.8 kg) months, but the difference at 18 months (0.4 kg) was no longer significant. Similarly, in TOHP phase II those on a low sodium diet lost more weight initially (1.2 kg difference at 6 months, p<0.001), but the difference had disappeared by 36 months.

Arroll 1995 found a weight loss of 1.4 kg in the low sodium group relative to the controls at six months. However, Morgan 1987, Thaler (Thaler men 1982; Thaler women 1982), and Silman 1983, found no change in weight in either control or low sodium groups.

In TONE eight participants not assigned to a weight loss intervention experienced excessive weight loss, but it is not clear how many of these were in the control or low sodium groups.

Overall, in the larger studies, where one is more likely to see any real effect, there appeared to be initial weight reductions accompanying the low sodium diet, but the effect was lost over several years.

Urinary sodium excretion.

Meta-analysis demonstrated a reduction in urinary 24 hour sodium excretion at intermediate (48.9 mmol/24 hours, 95% CI 65.4 to 32.5), and late follow up (35.5 mmol/24 hours, 95% CI 47.2 to 23.9) in those advised to follow a low sodium diet compared with control. Significant heterogeneity was seen in results at intermediate and late assessment, and was not resolved by sensitivity analysis leaving out trials with unclear or inadequate allocation concealment. The one trial to assess very late outcomes (TOHP phase I, in normotensives) found that at seven years sodium excretion in a small subset of their original sample was similar in intervention and control groups.

Other nutrients.

The relationship between low sodium dietary advice and other dietary components has not been fully explored in these studies. Potassium is the most reported component, usually measured as urinary excretion alongside sodium. Other nutrients were measured as dietary intakes using food record and recall systems.

Minerals

Potassium. In HPT potassium excretion was consistently greater in low sodium than control groups (about 6 mmol/24 hours at 3 years) but whether this difference was statistically significant is not clear. In TONE potassium intake was also greater in the low sodium group than in control (by 160 mg/24 hours, 95% CI 25 to 295). The rest of the trials found no significant differences in reported intakes or excretion of potassium including: TOHP phase I, TOHP phase II, Thaler men 1982, Thaler women 1982, Morgan 1978, Morgan 1987 and Silman 1983.

Magnesium. TONE found a higher intake of magnesium in low salt as compared with control groups (by 24 mg/24 hour, 95% CI 8 to 39), whereas TOHP phase I reported no significant difference between groups.

Calcium. TONE found a significant fall in calcium intake in the low salt as compared with the control group (of 71 mg/24 hours, 95% CI 119 to 23). HPT found a reduction in salt from dairy foods (suggested in all groups, but only significant in normal weight men), while TOHP phase I reported no significant net differences in reported intake of calcium.

Iron. TOHP phase I reported lower intakes of iron (3.6 mg/day at 18 months) in the low sodium group. The differences in iron were due to differences in men (women were similar between low sodium and control groups) and the reported iron intakes (15 mg/day at 18 months in men) in the low sodium group were still well over the RDA (10 mg/day for men). TONE also found lower iron intakes in the low sodium group (lower by 2.8 mg/24 hours, 95% CI 3.8 to 1.8).

Phosphorus and zinc were not significantly different in low sodium and control groups in TONE.
Vitamins.

TOHP phase I reported no significant net differences in reported intakes of vitamin A, vitamin C, thiamine, riboflavin or niacin. TONE found lower intakes of thiamine (0.12 mg/24 hours, 95% CI 0.22 to 0.02) and riboflavin (0.2 mg/24 hours, 95% CI 0.3 to 0.1) in low sodium groups, but no significant differences in vitamins A, B, C, D, E, folate or niacin (excluding supplements).

Macronutrients.

Energy. TOHP phase I reported significantly lower daily intakes of total energy (207 kcal) in the low sodium group, as did TONE (by 119 kcal/24 hours, 95% CI 197 to 41).

Fats. Lower intakes of total fat (by 5.8 g/24 hours, 95% CI 10.1 to 1.5), saturated fat (by 2.4 g/24 hours, 95% CI 4.0 to 0.8) and mono-unsaturated fat (by 2.2 g/24 hours, 95% CI 4.0 to 0.4) were seen in the low sodium group of TONE. No significant differences were seen in polyunsaturated fat intake. TOHP phase I reported significantly lower daily intakes of total fat (11.4 g) in the low sodium group, but no significant net differences in saturated fat.

Alcohol. TOHP phase II reported that there were no differences between the low sodium and usual care groups in alcohol intake, while Arroll 1995 reported an increased intake of alcohol in the control group (2.4 g/day), though it was not clear whether this was statistically significant. TOHP phase I reported no significant net differences in reported intake of alcohol.

Protein and carbohydrates were not significantly different in the low sodium and control groups in TONE.

Overall, there is a trend towards increases in potassium and magnesium, and a fall in calcium, iron, some B vitamins, total energy, total and saturated fats in low sodium groups.

Anti-hypertensive medications used.

Two trials in patients with elevated blood pressure considered the ability of low salt diets to maintain blood pressure control after stopping anti-hypertensive medication. In the smaller trial (Morgan 1987) anti-hypertensive therapy was stopped two months after randomisation to usual or low sodium diet, but restarted if diastolic blood pressure rose. After six months, four of ten men on low sodium diet were taking anti-hypertensive medication, compared to nine of ten on usual diet (relative risk 0.44, 95% CI 0.20 to 0.98).

In the larger study (TON, 975 participants, including those on weight reduction interventions) withdrawal of medication was attempted 3 months after randomisation to low sodium diet (with behavioural therapy) or usual care. The primary combined endpoint (a combination of high blood pressure at any visit, restarting of anti-hypertensive medication or any clinical cardiovascular event) was less common in the low sodium group, relative risk 0.83 (95% CI 0.75 to 0.92), ARR 14%, NNT 7.

**DISCUSSION**

Eleven long term randomised controlled trials of dietary salt reduction (including 3514 participants) provided few data on mortality (17 deaths in total), cardiovascular events or quality of life, but did demonstrate a significant decrease in systolic blood pressure (1.1 mm Hg, 95% CI 1.8 to 0.4) and urinary sodium excretion (35.5 mmol/24 hours, 95% CI 47.2 to 23.9) at 13 to 60 months after initial advice. The decrease in diastolic blood pressure was smaller (0.6 mm Hg, 95% CI 1.5 to -0.3). The data suggest that a low salt diet may help people on anti-hypertensives to stop their medication without losing blood pressure control. The data from TONE suggest that for every 7 patients assigned a goal of achieving a sodium intake of less than 80 mmol/day, one would remain off antihypertensive medication with a BP less than 150/90 mm Hg and with no adverse cardiovascular events.

Effects of low salt dietary advice on mortality and cardiovascular morbidity

Health promotion interventions involve several stages before any health outcome is seen. First, the advice must result in changed behaviour (cutting down on salt in foods) and secondly that behaviour must result in an improved health outcome (reduced cardiovascular illness, increased life expectancy). A major weakness of this review is that we were not able to assess the overall effect of advice to reduce dietary sodium on mortality or morbidity (as not enough events have been accumulated to see any definitive answer). Instead we have tried to follow the process by assessing several intermediate outcomes including urinary sodium excretion and blood pressure; however there may be effects on other risk factors.

It is not clear what effects a low sodium diet has on cardiovascular events and mortality. It has been suggested that lowering sodium intake may have adverse effects on the vascular endothelium through stimulation of the renin-angiotensin system (Alderman 1997), and adverse effects on serum total and LDL cholesterol levels (Graudal 1998) have been suggested. In cohort studies, lower salt intake in hypertensives has been associated with higher levels of cardiovascular disease (Alderman 1995) and in general populations (Alderman 1998; Tunstall-Pedoe 1997) with greater all-cause mortality. However, among obese people lower salt intake may be associated with reduced risk of cardiovascular events (He 1999; Tuomilehto 2001). These apparently contradictory findings emphasizes the fact that we do not know whether long-term salt restriction is beneficial or harmful.

Effects of low salt dietary advice on sodium excretion

The review suggests that sodium reduction of about a quarter of usual sodium intake in US and UK populations (MAFF 1999) can be achieved long term. This may be exaggerated. For example, HPT found that 48% of participants ate differently on the day of their food record, eating less food, and substituting simpler foods.
Several people in the low sodium group also reported eating less salt on days salt intake was recorded. Whether food adjustment also occurred when urine samples were collected (and whether these were complete) is not known. Male participants in Thaler's trial (Thaler men 1982) were believed to have relaxed their salt restriction between urine samples (O. Simpson, personal communication, 2001).

Is it realistic to ask people to alter their salt intake long term? Advice to reduce dietary salt is common in primary care if the British Hypertension Society's Guidelines (Ramsay 1999; Ramsay 1999a) are being followed. These guidelines advise that 'reduced use of salt when preparing food and elimination of excessively salty foods from the diet' 'be offered to all hypertensive people and those with a strong family history of hypertension'. It does appear that the degree of salt restriction attained attenuates over time (Meta-analysis, subgrouping and sensitivity analysis results) and this occurs despite a great deal of ongoing encouragement and support (comprehensive interactive programmes of dietary and behavioural education involving specialized and highly trained staff; vast input of skills, time and materials) in all of the four large high quality trials. The resulting falls of 1.1 mm Hg systolic and 0.6 mm Hg in diastolic blood pressure may be useful at a population level; however the intensity of intervention applied to individuals required to achieve this is not realistic for community control of high blood pressure, which would need to be through changes in food production and catering practices.

Effects of low salt dietary advice on blood pressure

While both urinary sodium excretion and blood pressure fell, the salt reduction may not have caused the fall in blood pressure. Alterations in diet aimed at reducing salt intake may perhaps systematically affect other dietary components (such as alcohol, potassium, calcium, fat or energy intake) that may themselves alter blood pressure (Cappuccio 1991; Allender 1996; Whelton 1997; Ebrahim 1999; Brand 1999; Griffith 1999). The only available data suggest that potassium is not consistently affected by a low sodium diet, and that weight may be reduced in the medium term, but is unlikely to be exerting much effect on blood pressure by three years. Very little information is available on alcohol (suggesting no major effect), calcium (TOHP phase I reported no significant changes in intake of calcium, but HPT reported a reduction in salt from dairy foods) or fat (suggesting that significant reductions may be occurring in low sodium groups, reported in only one large trial). The significant reduction in weight of people given low sodium dietary advice in the medium, but not the longer term, may explain why the effect of a low sodium diet on blood pressure ‘drops off’ so much between intermediate and late follow up in this review. It may also explain why, in this review, no relationship is seen between the degree of reduction in sodium excretion and change in blood pressure. However the number of trials is small and relating a mean change in blood pressure to a mean change in urinary sodium is statistically weak. In previous meta-analyses (Characteristics of systematic reviews on salt and blood pressure) a relationship has been seen in some cases but not in others. Individual participant data are required to take this issue further.

We expected that short duration trials would achieve larger falls in blood pressure that would attenuate over time, in line with attenuation of salt restriction. Trials in normotensives in the Graudal review (Graudal 1998) (Characteristics of systematic reviews on salt and blood pressure) had a median length of 8 days, a reduction of 160 mmol/24 hours in urinary sodium excretion and a fall of 1.2 mm Hg in systolic blood pressure, while in this review (median trial length 36 months, 34 mmol/24 hours difference in sodium excretion) systolic blood pressure fell by 1.1 mm Hg. In hypertensives our results are less easy to interpret due to the low quality of included studies, but there is no clear suggestion that blood pressure effects diminish with longer duration trials or with smaller reductions in sodium excretion. This suggests that homeostatic mechanisms (Navar 1997) do not operate over the longer term to re-set usual blood pressure levels as might be expected. It has been suggested that ‘usual’ blood pressure may be set in utero or early childhood (Barker 1998) so it is possible that dietary salt intake in early childhood has a greater role in determining adult blood pressure than salt intake in adulthood; however the evidence is mixed (Lucas 1988; Hofman 1983; Singhal 2001), and open to varying interpretations (He 2001). A systematic review in this area would be helpful.

Part of the blood pressure lowering effect at longer follow up may be due to lower sodium diets preventing blood pressure rise with age. The Intersalt observational study (Elliott 1996) suggested that a population excreting 100 mmol/day less sodium would experience a 10 and 6 mm Hg lower rise in systolic and diastolic blood pressure over 30 years. This review suggests that voluntary reduction of only 35 mmol Na/24 hours is realistic for periods of over one year. This would prevent 3-4 mm Hg systolic (2 mm Hg diastolic) blood pressure rise over thirty years. However, the sodium reduction achieved may decline over time so this additional protective effect of low salt advice may be limited.

The sodium reduction arms of the DASH (Sacks 2001) study are not included in this review as their intervention periods were only 30 days; however the strength of the study was in providing food for the participants and so tightly regulating sodium (as well as potassium and calorie) intake. Participants in the ‘control intermediate sodium’ arm reduced their sodium excretion by 35 mmol/day compared with the ‘control normal sodium’ arm, reducing systolic (2.1 mm Hg, 95% CI 3.4 to 0.8 mm Hg) and diastolic (1.1 mm Hg, 95% CI 1.9 to 0.2 mm Hg) blood pressure by amounts similar to those seen in 13-60 month follow up in this review. With greater reductions in sodium, systolic blood pressure decreased by a greater amount (6.7 mm Hg, 95% CI 5.4 to 8.0).

Effects of low salt dietary advice on other outcomes

There is evidence that a low sodium diet improves the chance
of maintaining controlled blood pressure following withdrawal of antihypertensives.

There are several reasons for assessing levels of other nutrients in a low sodium diet. Altering any one component of a complex diet will in turn alter the intake of many other micro and macro-nutrients. It is important to ensure that a low sodium diet is nutritionally adequate. It is also necessary to be aware that changes in many nutrients have their own long term effects on blood pressure and other aspects of cardiovascular health. The available data are scant but suggest increases in potassium and magnesium intake, and reductions in energy and total fat intakes, all of which might be expected to help reduce blood pressure in their right as well as protecting against cardiovascular disease in other ways. This is good news for health, but raises further questions about the extent of the effect of salt reduction itself on blood pressure. It may be that the small changes in blood pressure seen in these long term trials are due to increases in potassium and decreases in fat intake. On the other hand, the reductions in calcium and iron seen in some trials might endanger dietary adequacy for a few people, increasing the risk of osteoporosis and anaemia. It may be that the effect on blood pressure, and more generally on health, of a low sodium diet depends on the types of messages used, the specific dietary measures taken. These may differ considerably from trial to trial, or even from participant to participant.

We have included only a small number of the many randomised controlled trials on the effect of salt manipulation, and none of the intra- or inter-population surveys, cohorts or animal trials that are commonly referred to when the effect of salt reduction on health is discussed. Most of the randomised controlled trials that have been performed have been of short duration and do not assess whether dietary advice has any long term effect on health outcomes or blood pressure. Despite an extensive search, only eleven trials fulfilled our inclusion criteria (determined by our question). Where randomised controlled trials in humans are available to answer a question on health, it would be inappropriate to include animal studies, surveys or cohort studies, which have contradictory results and interpretations (Taubes 1998).

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**TONE (published data only)**


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References to other published versions of this review

Hooper 2002  

* Indicates the major publication for the study

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