SODIUM

Evidence Paper

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SUMMARY

RECOMMENDATIONS

General population	▪ Reduce your sodium intake to 2300mg of sodium a day (approximately 6g salt) or less. This includes sodium from processed food.
	▪ Base your eating on the Heart Foundation's *Nine steps for heart healthy eating* with emphasis on minimally processed (fresh or plain) foods.
	▪ If you choose to eat some processed foods:
	  ▪ Read Nutrition Information Panels (NIP) and choose the lowest sodium options available.
	  ▪ Preferably choose foods without added salt in the ingredient list or "no added salt" on the front of pack.
	  ▪ Alternatively choose foods with "low salt" on the front of pack. "Low salt" means no more than 120mg sodium per 100g.
	▪ Avoid adding salt during cooking and at the table.
	▪ Limit salty snacks and takeaway foods high in salt.

Health professionals	▪ When advising people with high blood pressure, heart failure or those with CVD, include nutrition recommendations above with the following amendments:
	  ▪ Reduce salt intake to 1600mg of sodium a day (approximately 4g a day of salt), including salt in processed foods.
	  ▪ No addition of salt during cooking or at the table.

Food Industry	▪ All sectors of the food industry are encouraged to be engaged in a salt-reduction programme to decrease the sodium content of foods within their product range.

KEY POINTS

IMPACT OF SODIUM ON BLOOD PRESSURE

Sodium intake (over 1265mg/day) is directly associated with blood pressure.

The Intersalt observational study reported:

- that at 1265mg sodium/day or less there was no evidence of high blood pressure or increasing blood pressure with age (Intersalt Cooperative Research Group, 1988); and
- that for within-population analyses, individual intakes that were higher by 2300mg sodium/day were associated with systolic blood pressure higher, on average, by 3-6mm Hg (Elliot et al, 1996).

It is estimated from the Intersalt study that a 2300mg/day higher sodium intake over a 30-year period from 25 years of age, translates into an approximate 10-11mm Hg higher systolic blood pressure at 55 years of age (Elliot et al, 1996).

Population-based interventions and meta-analyses of randomised controlled trials have shown that it is possible to achieve significant reductions in blood pressure with reduced salt intake in both hypertensive and normotensive individuals.

A meta-analysis of sodium reduction trials (at least 4 weeks) reported that a reduction of:

- 1800mg sodium/day, reduced systolic blood pressure by 5mm Hg (hypertensive[*])
- 1700mg sodium/day, reduced systolic blood pressure by 2mm Hg (normotensive[†]) (He & McGregor, 2009).

TOPH II trial reported that at 36 months a modest sodium intake reduction (920mg/day) was associated with an 18% lower incidence of hypertension (Kumanyika, 2005).

Impact of Sodium + Diet on Blood Pressure

The combination of reduced sodium and DASH[‡] diet is particularly effective in reducing blood pressure.

Sodium intake reduced by 2070mg/day for 30 days using the "reduced-sodium +DASH diet" lowered the average systolic blood pressure by 9mm Hg (Sacks et al, 2001).

Impact of Sodium on Cardiovascular Disease

Sodium intake is associated with incidence of strokes and total cardiovascular disease events.

A Finnish prospective study reported that a third reduction in salt intake was accompanied by a more than 10mm Hg lowering of the population average of both systolic and diastolic blood pressure and a 75% to 80% decrease in both stroke and coronary heart disease in the population younger than 65 years (Karppanen & Mervaala, 2006).

Meta analysis of prospective studies reported that a difference of 2000mg sodium/day in habitual salt intake is associated with a 23% difference in the rate of stroke and 17% difference in the rate of total cardiovascular disease (Strazzollo et al, 2009).

Long-term observational follow up of randomised controlled trials reported reduction of between 760 and 1012mg sodium/day resulted in a 25% lower risk of cardiovascular disease events (Cook et al, 2007).

When all of the findings from various types of research studies are considered, the evidence for the impact of sodium on blood pressure and thereby cardiovascular disease is strong (He & MacGregor, 2009, Titze Ritz 2009, Ness 2009).

[*] hypertensive - systolic blood pressure >140mm Hg

[†] normotensive - systolic blood pressure < 140mm Hg

[‡] DASH diet is high in fruit, vegetables, legumes, fish, nuts and low-fat dairy and contains more potassium, calcium, magnesium, fibre and protein than the typical Western diet

EVIDENCE

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in New Zealand, responsible for 40% of all deaths (Hay, 2004). High blood pressure (BP), a major risk factor for CVD is impacted by sodium intake.

AIMS AND OBJECTIVES

The purpose of this document is to summarise the latest evidence and provide New Zealand context on the impact of sodium intake on CVD. The National Heart Foundation of New Zealand (NHF) acknowledges the National Heart Foundation of Australia document *Summary of evidence statement on the relationships between dietary electrolytes and cardiovascular disease* (National Heart Foundation of Australia, 2006) which should be read in conjunction with this update document on sodium. These updated findings are consistent with those in the National Heart Foundation of Australia *Summary of evidence statement*.

LITERATURE OVERVIEW

High BP is the leading global risk factor for mortality, outranking tobacco use, high blood glucose, physical inactivity, and overweight/obesity. In high-income countries, it is responsible for 17% of all deaths (World Health Organisation, 2009).

A meta-analysis of observational studies reported that throughout middle and old age, usual BP is strongly and directly related to CVD mortality, without any evidence of a threshold down to at least 115/75mm Hg (Lewington et al, 2002). The authors reported that while trials (typically of only a few years) have demonstrated that BP lowering can produce rapid reductions in CVD risk, this meta-analysis of observational studies provided complementary evidence of even greater differences in risk. These greater differences were likely to be produced by the more prolonged differences in BP; for example, a 10mm Hg lower usual systolic BP was associated with a 40% lower risk of stroke death and about 30% lower risk of death from ischaemic heart disease or other vascular causes. Even a 2mm Hg lower usual systolic BP would involve about 10% lower stroke mortality and about 7% lower mortality from other vascular disease in middle age. The researchers also reported that if just one single measurement of BP is to be used, systolic BP is slightly more informative in predicting risk than diastolic BP.

Sodium is an essential nutrient, but the main impact of too much sodium on health is to raise BP. Sodium intake has long been known to influence BP among hypertensive patients (systolic BP > 140mmHg or diastolic BP > 90mmHg, unless otherwise specified), but its effect among those without overt hypertension, as well as its effects on CVD, has been disputed (Cook, 2008).

The variability of the impact of sodium intake on BP that has been reported between many study results has in part been attributed to flaws in study design, analysis or interpretation (Ministry of Health, 2003; Cook 2008; Titze & Ritx, 2009). In addition, there is large within-individual variation in sodium intake, and individual response to sodium intake has been shown to be inconsistent over time (Chang et al, 2006, Cook 2008).

This update relies primarily on evidence as compiled in key trials, meta-analyses and systematic reviews.

Salt (40% sodium and 60% chloride) is the main source of sodium in the diet. See Table 1 for conversion between salt and sodium.

Table 1: Conversion between salt and sodium

1 g salt = 0.4g sodium 1 g salt = 17.1 mmol sodium 1 mmol sodium = 23mg sodium

RELATIONSHIP BETWEEN SODIUM INTAKE AND BLOOD PRESSURE

OBSERVATIONAL STUDIES

A large body of observational studies supports the strong positive relationship between sodium and BP within and between populations (Cook, 2008). [See *Summary of evidence statement on the relationships between dietary electrolytes and cardiovascular disease* (National Heart Foundation of Australia, 2006) for more information.]

Despite some criticism, the Intersalt study (Intersalt, 1988; Elliott et al, 1996; Elliott, 2002) is acknowledged by many researchers as a significant study (Cook, 2008; He and Macgregor, 2008; Dickinson & Havas, 2007). The study (n=10,074 individuals in 52 population samples from 32 countries) demonstrated that salt intake is significantly related to BP and that BP rises with age worldwide. At intake levels of 1265mg sodium/day or less, there is no evidence of high BP or increasing BP with age, indicating that 1265mg/day is a level at which harmful effects of sodium intake on CVD begin (Intersalt, 1988). Individual 24-hour urinary sodium excretion higher than 100mmol (equivalent to 2300mg sodium intake/day) was associated with systolic BP higher on average by 3 to 6mm Hg. Associations were larger in the 40-59 year age group compared with younger adults. The authors estimated that 100mmol/day greater sodium excretion over a 30 year period (age 25 to 55 years) resulted in 10-11mm Hg higher systolic BP (Elliott et al, 1996).

INTERVENTION TRIALS

Population-based intervention studies and randomised controlled clinical trials of sodium reduction in people with and without hypertension have supported these observational findings (Strazzullo, 2009, Hollenberg, 2006, Cook 2008). The effectiveness of dietary salt reduction on BP was highest in controlled clinical trials where the salt dose was administered by provision of prepared food items, compared with less rigorously controlled studies in the general population.

Short term sodium reduction trials

The Dietary Approaches to Stop Hypertension (DASH-sodium) intervention study looked at the effect of well-controlled sodium reduction along with other dietary changes (Sacks et al, 2001) and offers strong evidence of short term effects (thirty days) on BP in a dose-response fashion. Importantly, sodium reduction significantly lowered BP in non-hypertensive individuals on both the intervention diet (high in fruit, vegetables, legumes, fish, nuts and low-fat dairy and which contained more potassium, calcium, magnesium, fibre and protein than the typical western diet) and the control diet (typical Western foods). However, the combined reduced-sodium/DASH diet proved particularly effective for BP reduction - when sodium was reduced by 2070mg (90mmol) from approximately 3450 to 1380mg/day (150 to 60mmol/day), the average systolic BP was lowered by 9mm Hg (Sacks et al, 2001).

He & McGregor conducted an updated Cochrane meta-analysis and review (2004, updated 2006). It was restricted to randomised trials lasting longer than four weeks with a modest reduction in sodium intake. Median sodium intake reductions (as measured by urinary excretion) and their impact on BP were virtually unchanged from the original estimates (He & McGregor, 2009). It demonstrated that a reduction of 1800mg sodium/day reduced systolic BP by 5mmHg in people with high BP, and a median reduction of 1700mg sodium/day reduced BP by 2mmHg in people with normal BP. There was a continuous relationship between sodium and BP, so that within a salt intake range of 1200 to 4800mg sodium/day, a lower sodium intake resulted in lower BP.

In a recent Swedish double-blind placebo controlled cross-over trial (4 weeks/treatment, n=39), BP was reduced by approximately 6mm Hg when sodium intake was lowered by 2300mg (from 3450 to 1150mg sodium/day (150mmol to 50mmol/day)) (Melander et al, 2007).

Longer term sodium reduction trials

Five large randomised trials of at least one year have confirmed a modest effect of sodium reduction on BP in those with high normal BP ("pre-hypertension") (Cook et al, 2007). Modest reduction in average sodium intake (between 713 to 1012mg sodium) (31 to 44mmol/day) decreased the percentage of pre-hypertensive subjects who developed hypertension (Dickinson & Havas, 2007). For example, the Trials of Hypertension Prevention, Phase II reported that at 36 months, a 40mmol reduction in 24-hour urinary sodium excretion was associated with an 18% lower incidence of hypertension (Kumanyika et al, 2005).

IMPACT OF SODIUM ON CARDIOVASCULAR DISEASE

Until recently, data on the effect of dietary sodium intake on subsequent morbidity and mortality have been limited and inconclusive (Cook et al, 2007).

OBSERVATIONAL STUDIES

Despite imperfect measures of sodium intake, giving mixed results, prospective studies have generally suggested a direct association between sodium intake and CVD (Cook et al, 2007).

A prospective study in Finland (n= 2436, age 25-64 years) reported that 100mmol increase in 24-hour urinary sodium excretion was associated with increased coronary heart disease (51%), CVD (45%) and all-cause mortality (26%) (Tuomilehto et al, 2001). Sodium excretion appeared to be a greater risk factor for mortality in men who were overweight than for normal weight men. Over 30 years of follow-up, one third lowering of salt intake was accompanied by more than a 10mm Hg lowering of the population average of both systolic and diastolic BP and a 75% to 80% decrease in both stroke and coronary heart disease mortality in the population younger than 65 years (Karppanen & Mervaala, 2006).

Recently the evidence for an association between habitual sodium intake and CVD was assessed in a meta-analysis of prospective studies published from 1996 to 2008 (Strazzullo et al, 2009). The analysis pooled data from over 177,000 participants in 13 separate studies. Studies were conducted in 6 countries, with follow up from 3.5 years to 19 years, and with over 10,500 strokes or cardiovascular events experienced. The meta-analysis calculated that consuming an extra 2000mg sodium/day was associated with a 23% increased rate of stroke and a 17% increased rate of total cardiovascular disease. The associations observed were greater the larger the difference in sodium intake and the longer the follow-up. Because of imprecision in measurement of salt intake, the authors reported that these effect sizes are likely to be underestimated.

INTERVENTION TRIALS

Only limited data are available from randomised controlled trials of sodium reduction on cardiovascular outcomes. Because of logistical and ethical considerations, long term clinical trials evaluating the efficacy of sodium reduction on clinical events have not been conducted (Cook et al, 2007).

A long-term observational follow up (10 to 15 years) of the two American life-style intervention trials of hypertension prevention, TOPH I and TOPH II, was conducted with over 3000 participants (adults aged 30-54 years with prehypertension) (Cook et al, 2007). Reduction of 760 and 1012mg sodium [33mmol and 44mmol], in the original intervention groups respectively, resulted in a 25% lower risk of cardiovascular disease events. There were no differences in effects when examined by age, gender, race or body mass index. Self-reported sodium intake and no direct measurement of risk factors during follow up were reported by the researchers as some of the study limitations.

OTHER ISSUES FOR CONSIDERATION

SUBGROUPS

It has been indicated that some population subgroups (elderly, African Americans and people with hypertension or Type 2 diabetes) are generally more sensitive to the short-term BP-elevating effects of sodium (Dickinson and Havas, 2007; Sacks et al, 2001). It has been suggested that the between-individual variation in BP response to a reduction in salt intake may be partly due to different levels of plasma renin activity (He & MacGregor, 2009).

In subgroup (age, hypertension status, gender, and ethnicity) analyses of the DASH-Sodium trial, generally the lower the sodium intake, the greater the reduction in BP (Bray et al, 2004). Age had a strong and graded influence on the impact of sodium on BP reduction with its effect being especially strong in the non-hypertensive subgroup. The authors commented that reduced sodium intake and the DASH diet should be promoted for the prevention and treatment of high BP especially as the benefits to BP increase as subjects enter middle age, when the rate of cardiovascular disease increases sharply.

However further analysis was recently conducted of the Trial of Non-pharmalogic Interventions in the Elderly (TONE), a sodium reduction trial (n=681 hypertensive patients aged 60 to 80 years, mean follow up about 28 months) (Appel et al, 2010). The impact of sodium reduction on BP and CVD endpoints in elderly African-Americans and other subgroups (based on age, weight, gender) were not significantly different to overall findings.

The effect of sodium on BP extends to children and adolescents. A meta-analysis of ten controlled trials (n=966) in children and adolescents found that salt restriction (average 42% reduction) led to immediate improvements in BP. Reductions in systolic BP (2.5mmHg) were most pronounced in infants (He & MacGregor, 2006).

INTERACTION OF SODIUM AND POTASSIUM

Sodium and potassium may act jointly in development of hypertension and CVD (Chang et al, 2006; Cook, 2008) [see National Heart Foundation of Australia (2006)]. Meta-analyses of trials have reported that high potassium intake is associated with reduced BP (Whelton et al, 1997; Geleijnse et al, 2003). Fruit and vegetables through an increase in potassium intake have the opposite effect of sodium and may, in certain circumstances, partially offset the effects of a high salt intake (Cook, 2009).

However recent animal experiments with primates have been able to provide even better controlled conditions to quantify sodium effects in a high potassium and high calcium environment. The study (total 3 years) showed that even in mineral-replete states, sodium intake directly impacted on BP (Elliot et al, 2007).

As noted above, the combined reduced-sodium/DASH diet (which not only contains more potassium, but also more calcium, magnesium, fibre and protein than the typical western diet) proved particularly effective for BP reduction (Sacks et al, 2001).

SODIUM IMPACT ON CARDIOVASCULAR DISEASE, INDEPENDENT OF BLOOD PRESSURE

While much of the evidence in relation to sodium is on its impact on BP, there is also a strong body of evidence showing an association directly with cardiovascular disease. This means the effect on cardiovascular disease can be either through raised BP or an independent and additive effect (He & McGregor, 2009).

Table 2: Key findings on the impact of sodium on BP and CVD

Impact of Sodium on BP

Sodium intake (over 1265mg/day) is directly associated with BP

The Intersalt observational study reported:

- that at **1265mg** sodium/day or less, there was no evidence of high BP or increasing BP with age (Intersalt Cooperative Research Group, 1988),
- that for within-population analyses, individual intakes that were higher by **2300mg** sodium/day were associated with systolic BP that was higher, on average, by **3 - 6mm Hg** (Elliot et al, 1996).

It was estimated from the Intersalt study that a **2300mg**/day higher sodium intake over a **30 year period** from 25 years of age, translates into an approximate **10 - 11mm Hg** higher systolic BP at 55 years of age (Elliot et al, 1996).

Population-based interventions and meta-analyses of randomised controlled trials have shown that it is possible to achieve significant reductions in BP with reduced salt intake in both hypertensive and normotensive individuals:

A meta-analysis of sodium reduction trials (at least **4 weeks**) reported that a reduction of:

- **1800mg** sodium/day reduced systolic BP by **5mm Hg (hypertensive*),**
- **1700mg** sodium/day reduced systolic BP by **2mm Hg (normotensive**)** (He & McGregor, 2009).

TOPH II trial reported that at **36 months** observational follow up, a modest sodium intake reduction (**920mg**/day) was associated with an 18% lower incidence of hypertension (Kumanyika, 2005).

Impact of Sodium + Diet on BP

The combination of reduced sodium and DASH* diet is particularly effective in reducing BP.**

Sodium intake reduced by **2070mg**/day for **30 days** using the "reduced-sodium + DASH diet" lowered the average systolic BP by **9mm Hg** (Sacks et al, 2001).

Impact of Sodium intake on CVD

Sodium intake is associated with incidence of strokes and total cardiovascular events.

A Finnish prospective study reported that a third reduction in salt intake was accompanied by a more than 10mm Hg lowering of the population average of both systolic and diastolic BP and a **75% to 80%** decrease in both stroke and coronary heart disease in the population **younger than 65 years** (Karppanen & Mervaala, 2006).

Meta-analysis of prospective studies reported that a difference of **2000mg** sodium/day in habitual salt intake is associated with a **23%** difference in the rate of stroke and **17%** difference in the rate of total CVD (Strazzollo et al, 2009).

Long-term observational follow up of randomised controlled trials reported reduction of between **760 and 1012mg** sodium/day resulted in a **25%** lower risk of CVD events (Cook et al, 2007).

When all of the findings from various types of research studies are considered, the evidence for the impact of sodium on BP and thereby CVD is strong (He & McGregor, 2009).

*hypertensive - systolic BP >140mm Hg

**normotensive - systolic BP < 140mm Hg

***DASH diet is high in fruit, vegetables, legumes, fish, nuts and low-fat dairy and contains more potassium, calcium, magnesium, fibre and protein than the typical western diet.

IMPLICATIONS OF EVIDENCE

When considering the impact on health, it is important to bear in mind that sodium reduction benefits most of the population, not only those who are classified as having high BP. Around 80% of the adult population in most countries around the world are at risk of cardiovascular disease due to their high BP (He & McGregor, 2007). This is due to the continuous graded relationship between BP and CVD, starting at a BP of 115/75mmHg, well within the "normal" range. As the majority of the population have BP within the "normal" range, the greatest number of CVD cases attributed to BP actually occur in the upper part of the "normal" range, despite individual risk being higher with increasing BP (Project HeartSAFE, 2010).

CONCLUSION

The evidence reviewed suggests that reducing sodium intake in hypertensive and normotensive people is associated with decreasing BP.

There is evidence that high sodium diets are associated with increased incidence of CVD and that reducing dietary sodium is associated with decreased incidence of CVD.

NEW ZEALAND CONTEXT

Prevalence of high BP

The 2006/2007 New Zealand Health Survey found that one in seven adults (13%) or 425,000 people reported taking medication for high BP (Ministry of Health, 2008). This underestimates the number of people with high BP, as not all those with high BP will be diagnosed or taking medication (Project HeartSAFE, 2010). In New Zealand in 1997, 11% of all deaths (3699) were attributed to high BP (Stefanogiannis et al, 2005; Ministry of Health and the University of Auckland, 2003).

Sodium intake

Urinary sodium analysis is an accurate indicator of steady state sodium intakes, and the latest estimate (1993-98) using this method gives an average daily intake of 3464mg sodium (Thomson & Colls, 1998).

Major sources of sodium

The leading sources of sodium in the New Zealand diet identified In the 1997 National Nutrition Survey, were bread (25.7%), processed meats (10.3%), sauces (6.7%), potatoes and kumara (6.7%) breakfast cereals (5.8%) (Russell et al, 1999). It is estimated that three-quarters of sodium consumed is obtained from salt added during food manufacture, with the remainder either added at the table or in cooking (discretionary salt), or naturally present in food (James, Ralph & Saucz-Castillo, 1987, Mattes & Donnelly, 1991). Sodium content in these processed foods will need to be reduced to enable population goals for sodium intake to be achieved.

Iodised Salt

Iodised salt has been the main vehicle for iodine in the New Zealand diet. However this should not be used to argue that salt shouldn't be reduced, as iodine intake can be addressed in other ways. Furthermore, it is now mandatory for iodised salt to be used in manufactured bread. It is yet to be determined if this is a sufficient measure on its own to achieve adequate iodine intake.

Predicted impact of BP and sodium reduction

It has been projected that even a 0.5mm Hg average reduction in population systolic BP over the decade to 2011, would avoid 282 deaths from ischaemic heart disease and stroke in New Zealand a year (Ministry of Health and the University of Auckland, 2003).

Food Standards Australia New Zealand (FSANZ) has modelled the impact of an across-the-board reduction in sodium intake from manufactured foods (Goodall, 2008). They predicted that with a 15% sodium reduction, there would be approximately a 20% reduction in risk of a CVD event. They estimated that by 2018 this would result in 930 fewer deaths/year from CVD within 10 years.

RECOMMENDATIONS

The current New Zealand goal for population level sodium intake, or the recommended upper level of intake, is 2300mg/day (see Table 3) (National Health and Medical Research Council, 2006). This has been set as a realistic target for sodium reduction.

Additional health benefits accrue if sodium intakes are further reduced to 1600mg/day, which is the suggested dietary target (National Health and Medical Research Council, 2006). Ideally, this would be a long-term goal for sodium reduction.

Table 3: Nutrient Reference Values for Sodium in New Zealand

Age	Adequate intake (mg/day)	Upper daily level (mg/day)	Suggested dietary target	Actual intake (mg/day)	% of upper daily level
Adults	460-920 mg/day	2300mg/day	1600mg/day	3464mg/day	150%
Adolescents 14-18 years	460-920mg/day	2300mg/day			
Children 9-13 years	400-800mg/day	2000mg/day			
Children 4-8 years	300-600mg/day	1400mg/day			
Children 1-3 years	200-400mg/day	1000mg/day			

Source: National Health and Medical Research Council, 2006

These recommendations are consistent with those from other organisations (National Heart Foundation of Australia, (2006); American Heart Association, (Appel et al, 2006); Institute of Medicine, (2004) and are supported by the National Heart Foundation of New Zealand. To achieve the population goal of 2300mg sodium, New Zealanders need to reduce the intake of sodium from manufactured food and discretionary salt by one-third (1164mg sodium/day).

Recommendations

The following recommendations with respect to dietary sodium intake are made to improve the health of New Zealanders and reduce the current level of cardiovascular disease. The main focus for sodium reduction is salt added during food manufacture and discretionary salt.

All New Zealanders

1. Reduce your sodium intake to 2300mg of sodium a day (approximately 6g salt) or less. This includes sodium from processed food.

2. Base your eating on the Heart Foundation's *Nine steps for heart healthy eating* with emphasis on minimally processed (fresh or plain) foods.

3. If you choose to eat some processed foods
   - Read Nutrition Information Panels (NIP) and choose the lowest sodium options available
   - Preferably choose foods without added salt in the ingredient list or "no added salt" on the front of pack
   - Alternatively choose foods with "low salt" on the front of pack
   - "Low salt" means no more than 120mg sodium per 100g.

4. Avoid adding salt during cooking and at the table.

5. Limit salty snacks and takeaway foods high in salt.

6. Discuss healthy eating and concerns about nutrition with a registered dietitian, registered nutritionist, your doctor or practice nurse.

Health Professionals

When advising people with high BP, heart failure or those with CVD, include nutrition recommendations above with the following amendments:

1. Reduce salt intake to 1600mg of sodium a day (approximately 4g salt a day), including salt in processed foods.

2. No addition of salt during cooking or at the table.

Food Industry

All sectors of the food industry – retailers, manufacturers, trade associations, caterers, public procurement and suppliers to the catering industry – are encouraged to be engaged in a salt-reduction programme to decrease the sodium content of foods within their product range.

Recommendations and strategies for reducing sodium in processed foods are addressed in *Reducing our sodium footprint: Project HeartSAFE Situation Analysis* (Project HeartSAFE, 2010).

RESOURCES

Visit www.heartfoundation.org.nz or ring 09 571 9191 for further healthy eating information

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APPENDICES

APPENDIX 1: ABBREVIATIONS

BP	Blood pressure
CVD	Cardiovascular disease
DASH	Dietary approaches to stop hypertension
IHD	Ischaemic heart disease
NZGG	New Zealand Guidelines Group
RCT	Randomised controlled trial

APPENDIX 2: KEY EVIDENCE TABLES

Level of evidence: 2++	Strazzullo P, D'Elia L, Kandala N-B and Cappuccio F, 2009. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies
Study type	Meta-analysis of prospective cohort studies on habitual salt intake and risk of stroke or CVD with follow-up for at least three years.
Population	Adults. Eleven studies recruited both male and female participants, two included only men.
Intervention	Observational studies - salt intake assessed at baseline.
Comparator	Relative risk of higher versus lower salt intake; event rates were compared between categories which were as close as possible to a 6g salt/day difference.
Outcomes	Outcomes: stroke or CVD. Studies: 19 independent cohort samples from 13 studies with 177,025 adults from six countries included in the meta-analysis. Follow-up of 3.5-19 years. Over 11,000 CVD events and/or mortality reported. Higher salt intake was associated with greater risk of stroke (pooled relative risk 1.23, 95% confidence interval 1.06 to 1.43; P=0.007) and cardiovascular diseases (1.14, 0.99 to 1.23; P=0.07). Dose response was demonstrated. Consuming an extra 2000mg sodium/day was associated with a 23% increased rate of stroke and a 17% increased rate of CVD.
Limitations	Estimation of baseline population salt intake in each study was based on a single measurement (whether through 24-hour urine collection, dietary assessment, or food frequency questionnaire). This also meant that the authors were unable to correct for regression dilution bias, although this is likely to have led to an underestimation of effect. There were differences between studies in how salt intake was categorised. Study quality was assessed by Browns & Black score, with an average of 15.5 out of 19 (range 12-18). Funnel plots showed no evidence of publication bias.

Level of evidence: 1+	He F and Macgregor G, 2004. Effect of longer-term modest salt reduction on blood pressure (review)
Study type	Meta-analysis of randomised trials on the effect of salt reduction on blood pressure.
Population	Adults with normal or elevated blood pressure but not on concomitant interventions such as medication. Children and pregnant women excluded.
Intervention	Modest reduction in salt intake for four weeks or more (average salt reduction across the studies was 2g/day), measured by 24-hour urinary sodium excretion.
Comparator	Usual salt intake.
Outcomes	Main outcome: net changes in systolic and diastolic blood pressure. Twenty trials in individuals with elevated blood pressure (n=802) and 11 trials in individuals with normal blood pressure (n=2220) included in the meta-analysis. In individuals with elevated blood pressure, the median reduction in urinary sodium was 78 mmol/24h (4.6g salt/day), mean reduction in blood pressure was -5.06mm Hg for systolic and -2.70mm Hg for diastolic. In individuals with normal blood pressure, median reduction in urinary sodium was 74mmol/24h (4.4g salt /day), mean reduction in blood pressure was 2.03mm Hg for systolic and 0.99mm Hg for diastolic.
Limitations	Risk of bias: adequate allocation concealment reported in 23 out of 31 trials; 7 out of 31 trials used intention to treat analysis; small losses to follow up (average 6.5%); one non-blinded study. Funnel plots suggestive of publication bias, but resolved with exclusion of two trials with small salt reductions.