

Salt and Hypertension

The case for reducing salt intake

Existing evidence suggests that a high dietary intake of salt may contribute to the rise in blood pressure that occurs with increasing age in Western nations; and can promote the development of hypertension, or aggravate hypertension that is already present.

Hypertension is the most common identifiable chronic disease in Western society with a prevalence that rises steadily with age. In Australia, for those aged 25 years and over, in 1999/2000, approximately 31% of men and 26% of women had high blood pressure. In the same survey, about 70% of those aged 65 – 74 had high blood pressure or were on medication for high blood pressure treatment.¹ Reduction of blood pressure with long-term antihypertensive medications can reduce the risk of complications such as stroke and ischaemic heart disease, but may be accompanied by poorly-tolerated adverse events and represent a considerable cost burden to the individual and the community. Drug dosages can be reduced, and medications sometimes ceased altogether, in individuals who adopt a healthy lifestyle by reducing excess body weight, increasing physical activity, lowering alcohol intake and adopting a low-salt eating pattern. In fact, there are data to suggest that these measures might even prevent development of hypertension in the first place.

Measuring dietary salt intake

Under normal conditions of activity and sweating, approximately 90% of ingested sodium is excreted in the urine. Urinary sodium excretion over 24 hours therefore provides a means by which prevailing daily intake can be measured. This can help guide patients in their endeavour to maintain diets that provide the National Health and Medical Research Council recommended dietary intake (RDI) for sodium. The RDI (40–100 mmol/day for adults) was selected with the intention of preventing hypertension, based on available epidemiological data that demonstrated a relationship between high sodium intakes and an increased prevalence of hypertension.^{2–4} The Intersalt study strongly supported selection of this RDI, with reliable data collected under standardised conditions^{5,6} (see below). It should be noted that, as an individual may demonstrate marked day-to-day variation in sodium intake and excretion rates, a single collection may not reliably reflect habitual intake.

Epidemiological data

If the NH&MRC RDI for sodium is taken as the reference level, then Australians, as a group, consume too much salt. Average sodium intakes among 10 different study populations ranged from 130–200 mmol/day.³ In a National Heart Foundation funded study of 194 individuals living in Hobart, only 36% of females and 6% of males exhibited sodium excretion rates at or below 100 mmol/day.⁷

In the Intersalt study, an international cross-sectional analysis of 52 populations (over 10 000 individuals), 47 centres had median sodium excretion rates above the RDI for sodium, only two had rates within the range of RDI, and three had rates below.^{5,6} The study demonstrated a significant positive correlation between median 24-hour urinary sodium excretion and prevalence of hypertension within each population. Populations with higher rates of sodium excretion showed a steeper rise in blood pressure with increasing age compared with those with lower sodium excretion rates. By contrast, randomly selected individuals among the three populations with rates lower than the RDI demonstrated mean blood pressure levels of 96/61 mm Hg, 100/62 mm Hg and 108/63 mm Hg, a prevalence of hypertension of less than 1% and little if any rise in blood pressure with age. Of the two centres with median sodium excretion rates within the RDI range, one, with a median rate of 51 mmol/day showed very little hypertension (prevalence 5%), while the other, with a rate of 96 mmol/day,

showed a high prevalence of hypertension (26%). This latter population of African Americans, however, demonstrated other characteristics that might have predisposed them to hypertension, including high rates of alcohol consumption, high body mass index and very low potassium excretion.^{4,5} Furthermore, a median sodium excretion rate of 96 mmol/day implies that around one-half of these African-American subjects had sodium excretion rates above the RDI.

Law and coworkers assessed the relationship between mean sodium intake and mean blood pressure across a broad range of populations (47 000 people among 24 different communities) and showed positive correlations among both economically developed and underdeveloped groups of communities.⁸ These investigators also found consistent relationships between blood pressure and 24-hour urinary sodium excretion within 14 cross-sectional studies conducted in American, European and Asian Countries, after adjusting for the large degree of day-to-day variation in urine sodium excretion.⁹ Based on the findings of these population studies Law estimated that in older persons a 100 mmol/24 hr change in sodium intake was associated with a 10/5 mm Hg change in blood pressure, while in hypertensives the estimated pressure difference was 15/7 mm Hg.¹⁰

Salt loading and blood pressure

In a study conducted on 14 normotensive volunteers, experimental diets containing 1200–1600 mmol of sodium per day raised blood pressure in all subjects within three days.¹¹ Randomised, controlled studies on the effects of long-term salt loading on blood pressure have not been carried out in humans primarily because of ethical concerns. Salt supplements given to 13 chimpanzees that were previously living on a salt-free diet produced an average rise in blood pressure of 33 mm Hg systolic and 10 mm Hg diastolic over a 20-month treatment period (blood pressure in the control group did not change).¹² Blood pressure returned to baseline values in all animals during the six month period following cessation of salt supplementation.

Salt reduction and blood pressure

Numerous studies examining the effect of dietary sodium reduction on blood pressure levels have demonstrated a reduction in blood pressure in both normotensives and hypertensives.^{13–20} In a study of 975 subjects (585 obese and 390 nonobese) aged 60–80 years with hypertension controlled on a single medication, the rate of recurrence of hypertension or recommencement of treatment at a median of 29 months after withdrawal of their antihypertensive was reduced by 31% by sodium restriction and, among the obese subjects, by 53% through a combination of sodium restriction and weight loss.¹⁶ Based on Kaplan-Meier estimates, the proportions of randomised participants projected to be free of these endpoints at 30 months were 38% for those assigned to sodium reduction compared with 24% for those not assigned to sodium reduction. For the obese subjects, the proportions were 44% for sodium reduction and weight loss combined, compared with 16% for usual care. A double-blind, randomised, cross-over study of 47 individuals aged 60–78 years, most of whom were normotensive, demonstrated a mean BP fall of 7.2/3.2 mm Hg during one month of dietary sodium reduction.¹⁸ In an Australian double-blind, randomised study of 108 subjects with mild hypertension, modest reduction in dietary sodium intake led to a fall in mean blood pressure that was significant after only two weeks, and was 6.1/3.7 mm Hg by two months.¹³

In a multicentre, randomised trial conducted in the United States, the effect of different levels of dietary sodium, in conjunction with the Dietary Approaches to Stop Hypertension (DASH) diet, which is rich in vegetables, fruits, and low-fat dairy products, was studied in persons with and in others without hypertension. A total of 412 participants were randomly

assigned to eat either a control diet or the DASH diet. Within the assigned diet, participants ate foods with high, intermediate, and low levels of sodium for 30 consecutive days each, in random order. Urinary sodium averaged 142, 107 and 65 mmol per day during the high-intermediate-and low-sodium periods respectively. Reducing the sodium intake from the high to the intermediate level reduced the systolic blood pressure by 2.1 mm Hg during the control diet and by 1.3 mm Hg during the DASH diet. Reducing the sodium intake from the intermediate to the low level caused additional reductions of 4.6 mm Hg during the control diet and 1.7 mm Hg during the DASH diet. The effects of sodium were observed both in participants with and without hypertension, in African Americans and those of other races, and in women and men. The DASH diet was associated with a significantly lower systolic blood pressure at each sodium level and the difference was greater with high sodium levels than with low levels. As compared with the control diet with a high sodium content, the DASH diet with a low sodium content resulted in a mean systolic blood pressure that was 7.1 mm Hg lower in participants without hypertension, and 11.5 mm Hg lower in participants with hypertension.²⁰

Other studies have shown smaller effects, but have evoked criticism because the duration of the trials was too short, or because dietary sodium intake was not lowered sufficiently (to within RDI levels) in the study populations. In a meta-analysis of 56 trials of sodium reduction²¹, the overall fall in blood pressure for any given level of sodium reduction was small, but the median duration of the studies was only two weeks, with the shortest lasting only four days. Another meta-analysis of 32 studies demonstrated an overall fall in blood pressure, per 100 mmol reduction in daily sodium excretion, of 5.8/2.5 and 2.3/1.4 mm Hg in hypertensive and normotensive subjects respectively.²² The majority of trials within that meta-analysis which demonstrated little or no effect were either of short duration or achieved only modest reductions in urinary sodium excretion.

Blood pressure and 'salt sensitivity'

Individuals, both normotensive and hypertensive, vary in their blood pressure responses to changes in dietary salt intake. Most studies have found that, among hypertensive subjects, salt-sensitive individuals far outnumber salt-resistant individuals.²³ Some demographic factors, such as African-American race, obesity and older age, have been shown to be associated with increased salt sensitivity.^{13,23} The genetic bases for certain inherited forms of 'salt-sensitive' hypertension, such as glucocorticoid-remediable aldosteronism and Liddle's syndrome, have recently been described, raising the possibility that more subtle forms of these types of genetic abnormalities may be responsible for salt-sensitive hypertension in a larger population of patients than previously thought.²⁴ Despite these advances in understanding 'salt sensitivity', there remains insufficient information to enable selection of individuals who are more or less likely to respond to dietary salt restriction.

Tolerability and safety of dietary salt reduction

In general, individuals adapt to the taste of low-salt foods within a few weeks, and often develop a strong preference for unsalted foods after several months of full compliance.²⁵ There has been no consistent evidence of any untoward symptoms associated with dietary sodium restriction. In particular, the incidence of muscle cramping does not appear to increase, and most individuals in one trial reported improved well being after commencing dietary sodium reduction.¹⁹

Some widely quoted epidemiological data have been interpreted to suggest possible adverse outcomes associated with dietary salt restriction. In an American study, men treated with antihypertensive medication were reported to demonstrate higher rates of myocardial infarction at lower salt intakes.²⁶ However, salt intake was defined by only a single baseline

24-hour urine sample collected at the end of a five-day period of moderate dietary sodium reduction, with no collections being obtained while subjects were consuming their usual diets for the remaining period of observation. Data from the first US National Health and Nutrition Survey have been used to demonstrate an association of higher salt intakes with paradoxically lower blood pressure, lower prevalence of hypertension and greater survival rates after 20 years.^{27,28} The validity of these results has been challenged because (1) the dietary data were self-reported and (2) improved survival in the individuals with higher salt intakes could have resulted from an increased level of exercise, as they had a higher energy intake but were of similar weight to the comparison group. Overall, the weight of evidence suggests that a dietary salt intake within the RDI is a safe and desirable aim for most individuals.

Caution is recommended in a number of specific circumstances. In individuals with very low dietary sodium intakes (less than 50 mmol/day), administration of diuretics is unlikely to have an additive effect in lowering blood pressure and may cause unacceptable volume depletion and hyponatraemia.²⁹ The therapeutic effect of antihypertensive agents, especially beta-adrenoreceptor blockers³⁰, angiotensin converting enzyme inhibitors³¹ and angiotensin II receptor antagonists, is likely to be potentiated at a lower salt intake; doses of these agents may need to be reduced in order to avoid hypotension. In patients receiving lithium medication, the risk of lithium toxicity may be increased following the introduction of dietary salt restriction, and serum lithium levels should be closely followed under these circumstances.³² Patients with hypertension who reduce dietary salt intake after becoming pregnant may demonstrate a paradoxical rise in blood pressure³³, and theoretical concerns have been raised that dietary salt reduction could worsen manifestations of pre-eclampsia by aggravating the state of volume contraction characteristic of this disorder. This did not appear to be the case, however, for individuals who were already on a low-salt diet before becoming pregnant.³³ Dietary salt restriction is inappropriate in patients with salt-wasting forms of renal and gastrointestinal disease.

Other disorders associated with or aggravated by high-salt intake

The fluid retention that occurs when excessive amounts of salt are consumed can induce or aggravate symptoms of congestive cardiac failure. Excessive salt intake has also been implicated as a causative or aggravating factor in a number of other non-cardiac conditions such as Meniere's syndrome, kidney failure, hepatic failure, kidney stones and pre-menstrual syndrome.

Advising patients with hypertension on how to reduce salt intake

Patients are frequently surprised to learn that the total removal of salt from cooking and at the table will reduce sodium excretion by only 15%. Approximately 10% of the salt in a Western diet is present in the food as a natural ingredient, the remaining 75% comes from sodium compounds, mainly sodium chloride, added during processing.³⁴ Best results in lowering salt intake are therefore achieved by advising patients with hypertension not only to avoid adding salt to food, but even more importantly to buy fresh foods, foods normally processed without salt, and low-salt or no-added-salt groceries, and to learn how to read nutritional information panels on food packages in order to determine sodium content. Low-salt and low-sodium foods are defined by the Australian Food Standards Code as having a sodium content of **120 mg/100 g (52 mmol/kg) or less**. Although patients should not be discouraged from eating bread (which is low in fat and high in carbohydrate content), they should be made aware that bread is a significant contributor to daily sodium intake. Patients should be informed that bread can be purchased as no-added-salt varieties in selected bakeries and that low-salt bread can be made at home using readily available recipes. A less ideal option is for patients to choose standard brands with the lowest available sodium content (usually around 6 mmol/slice), but this will make it more difficult

for them to maintain total daily sodium intakes within the RDI (40–100 mmol/day). Breakfast cereals vary widely in sodium content, but several popular brands are low in salt. Patients with hypertension should be encouraged to eat plenty of fresh fruit and vegetables, low-salt bread and cereals, and advised to avoid those seasonings, processed foods and takeaway foods, which are high in salt.

Substitutes for salt include acids (e.g. vinegar, lemon, lime, plum and other fruit juices), curry spices, garlic, onion and herbs. Potassium chloride is another alternative, but is potentially dangerous in patients with renal dysfunction or those taking potassium-sparing diuretics. Some find the taste too bitter or metallic for continued use, and most prefer to abandon salt-flavoured additives altogether.

Summary of recommendations for patients with hypertension

- Advise patients to adopt a healthy lifestyle by reducing excess body weight, increasing physical activity, lowering alcohol intake and adopting a low-salt eating pattern.
- Encourage patients to eat plenty of fresh fruit and vegetables, low-salt bread and cereals, and advise them to avoid those seasonings, processed foods and takeaway foods, which are high in salt.
- Advise patients not to add salt during cooking or at the dinner table, and to choose low-salt foods (foods with a sodium content of 120 mg/100 g or less).
- Aim for a dietary sodium intake of 40–100 mmol/day, using 24-hour urinary sodium levels as a guide.
- Potassium chloride salt substitutes should be avoided in patients with renal dysfunction or in those who are taking potassium-sparing diuretics.
- Dosages of antihypertensive medications and of lithium carbonate may need to be reduced in patients who adopt a low-salt diet.
- The combination of diuretic treatment and low dietary salt intake may result in unacceptable volume depletion and hyponatraemia.
- Dietary salt restriction is inappropriate in patients with salt-wasting forms of renal and gastrointestinal disease.
- Women who become pregnant should continue their usual level of dietary salt intake.

References

1. Australian Institute of Health and Welfare (AIHW) 2001. Heart, stroke and vascular diseases: Australian Facts 2001. AIHW Cat No. CVD 13. Canberra: AIHW, National Heart Foundation of Australia, National Stroke Foundation of Australia (Cardiovascular Disease Series No. 14).
2. NH&MRC. Dietary guidelines for Australians. Canberra: Australian Government Publishing Service, 1991:7–77.
3. Bullock J. Sodium (Na) J Food & Nutr 1982;39:181–186.
4. Beard TC. Sodium – update. In: Truswell AS, Dreosti IE, English RM, *et al.* (eds). Recommended nutrient intakes. Australian papers. Mosman NSW: Australian Professional Publications, 1990:183–190.
5. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24-hour urinary sodium and potassium excretion. Br Med J 1988;296:319–328.
6. Elliott P, Stamler J, Nichols R, *et al.* Intersalt revisited: further analysis of 24 hour sodium excretion and blood pressure within and across populations. Brit Med J 1996;312:1249–1253.
7. Beard TC, Woodward DR, Ball P, *et al.* The Hobart Salt Study 1995: few meet national sodium intake target. Med J Aust 1997;166:404–407.

8. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? Analysis of observational data among populations. *Br Med J* 1991;302:811–815.
9. Frost CD, Law MR, Wald NJ. By how much does dietary salt reduction lower blood pressure? Analysis of observational data within populations. *Br Med J* 1991;302:815–818.
10. Law MR. Epidemiological evidence on salt and blood pressure. *Am J Hypertens* 1997;10:42S–45S
11. Luft FC, Rankin LI, Bloch R, *et al.* Cardiovascular and humoral responses to extremes of sodium intake in normal black and white men. *Circulation* 1979;60:697–706..
12. Denton D, Weisinger R, Mundy NI, *et al.* The effect of increased salt intake on blood pressure of chimpanzees. *Nat Med* 1995;1:1005–1016.
13. Australian National Health and Medical Research Council Dietary Salt Study Management Committee. Fall in blood pressure with modest reduction in dietary salt intake in mild hypertension. *Lancet* 1989;i:399–402.
14. Report to the Medical Research Council. The rice diet in the treatment of hypertension. *Lancet* 1950;ii:455–458.
15. Law MR, Frost CD, Wald NJ. By how much does dietary salt reduction lower blood pressure? III – Analysis of data from trials of salt reduction. *Brit Med J* 1991;302:819–824.
16. The Trials of Hypertension Prevention Collaborative Research Group. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high–normal blood pressure. *Arch Intern Med* 1997;157:657–667.
17. Whelton PK, Appel LJ, Espeland MA, *et al.* Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). *JAMA* 1998;279:839–846.
18. MacGregor GA, Sagnella GA, Markandu ND, *et al.* Double-blind study of three sodium intakes and long-term effects of sodium restriction in essential hypertension. *Lancet*;ii:1244–1247.
19. Beard TC, Cooke HM, Gray WR, *et al.* Randomized controlled trial of a no-added-salt diet for mild hypertension. *Lancet* 1982;ii:455–458..
20. Sacks FM, Svetkey LP, Vollmer WM, *et al.* Effects on blood pressure of reduced dietary sodium and the dietary approaches to stop hypertension (DASH) diet. *N Engl J Med* 2001;344:310.
21. Midgley JP, Matthew AG, Greenwood CMT, *et al.* Effect of reduced dietary sodium on blood pressure: a meta-analysis of randomized controlled trials. *JAMA* 1996;275:1590–1597.
22. Cutler JA, Follmann D, Allender PS. Randomized trials of sodium reduction: an overview. *Am J Clin Nutr* 1997;65(suppl):653S–651S.
23. Weinberger MH. Salt sensitivity of blood pressure in humans. *Hypertension* 1996;27:481–490.
24. Gordon RD, Stowasser M. Familial forms broaden horizons for primary aldosteronism. *Trends Endocrinol Metab* 1998;9:220–227.
25. Bertino M, Beauchamp GK, Engelman K. Long-term reduction in dietary sodium alters the taste of salt. *Am J Clin Nutr* 1982;36:1134–1144.
26. Alderman MH, Madhavan S, Cohen H, *et al.* Low urinary sodium is associated with greater risk of myocardial infarction among treated hypertensive men. *Hypertension* 1995;25:1144–1152.
27. McCarron DA, Morris CD, Henry HJ, *et al.* Blood pressure and nutrient intake in the United States. *Science* 1984;224:1392–1398.
28. Alderman MH, Cohen H, Madhavan S. Dietary salt intake and mortality: the National Health and Nutrition Examination Survey (NHANES 1). *Lancet* 1998;351:781–785.
29. Van Brummelen P, Schalekap M, de Graeff J. Influence of sodium intake on hydrochlorothiazide-induced changes in blood pressure, serum electrolytes, renin and aldosterone in essential hypertension. *Acta Med Scand* 1978;204:151–157.
30. Owens CJ, Brackett NC. Role of sodium intake in the antihypertensive effect of propranolol. *South Med J* 1978;71:43–46.

31. MacGregor GA, Markandu ND, Singer DRJ, *et al.* Moderate sodium restriction with angiotensin converting enzyme inhibitor in essential hypertension: a double-blind study. *Brit Med J* 1987;294:531–534.
32. Furlong FW. Lithium toxicity occurring with a 'reducing' diet. *Canad Psychiatr Assoc J* 1973;18:75–76.
33. Gallery E. Salt and hypertension in pregnancy. *Med J Aust* 1985;143:519.
34. James WPT, Ralph A, Sanchez-Castillo CP. The dominance of salt in manufactured food in the sodium intake of affluent societies. *Lancet* 1987;i:426–428.

National Blood Pressure Advisory Committee

Professor Lindon Wing (Chair)

Dr Andrew Boyden

Dr Anthony Dart

A/Professor Karen Duggan

Professor Graeme Hankey

Dr Mark Nelson

Professor Ian Puddey

Dr Michael Stowasser

Dr Janet Vial

Ms Jacquie Smith (Executive Officer)

February 2002

Heart Foundation State Division Offices

Australian Capital Territory

Cnr Denison Street &
Geils Court
Deakin ACT 2600
Phone (02) 6282 5744

New South Wales

Sydney

Level 4
407 Elizabeth Street
Surry Hills NSW 2010
Phone (02) 9219 2444

Newcastle

Suite 5, OTP House
Bradford Close
Kotara NSW 2289
Phone (02) 4952 4699

Northern Territory

Third Floor
Darwin Central Building
21 Knuckey Street
Darwin NT 0800
Phone (08) 8981 1966

Queensland

Brisbane

557 Gregory Terrace
Fortitude Valley QLD 4006
Phone (07) 3854 1696

Rockhampton

Unit 6, 160 Bolsover Street
Rockhampton QLD 4700
Phone (07) 4922 2195

Toowoomba

417 Ruthven Street
Toowoomba QLD 4350
Phone (07) 4632 3673

Townsville

36 Gregory Street
Townsville QLD 4810
Phone (07) 4721 4686

South Australia

155-159 Hutt Street
Adelaide SA 5000
Phone (08) 8224 2888

Tasmania

86 Hampden Road
Battery Point TAS 7000
Phone (03) 6224 2722

Victoria

411 King Street
West Melbourne VIC 3003
Phone (03) 9329 8511

Western Australia

334 Rokeby Road
Subiaco WA 6008
Phone (08) 9388 3343

Internet address:

www.heartfoundation.com.au

Heartline

1300 36 27 87

Donation Line

1300 30 11 65



Heart Foundation

ABN 98 008 419 761