Salt and Hypertension (Professional Paper)

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 an increased potassium[i], reduced salt eating pattern.
- Advise patients to reduce their salt intake to less than 4 g of salt a day (approximately 1550 mg of sodium a day), which is approximately one teaspoon of salt.
- Strategies to reduce sodium intake include:
  - choosing foods normally processed without salt and foods labelled 'no added salt' or 'low salt' ('low salt' means no more than 120 mg of sodium per 100 g)
  - choosing 'reduced salt' products if these are the lowest salt options available
  - avoiding high salt processed foods
  - avoiding salty snacks and takeaway foods high in salt
  - avoiding adding salt during cooking and at the table.
- 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.

[i]*Only in people with normal renal function*

Further information on electrolytes and cardiovascular disease (CVD) are available from **www.heartfoundation.com.au** (Professional section):

Summary of evidence statement on the relationships between dietary electrolytes and cardiovascular disease, National Heart Foundation of Australia 2006.

Position statement on the relationships between dietary electrolytes and cardiovascular disease, National Heart Foundation of Australia 2006.

For other information, contact **Heartline**, the Heart Foundation's national telephone information service on email **heartline@heartfoundation.com.au** or **1300 36 27 87** (local call cost).

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Current National Heart Foundation of Australia recommendations on sodium initake[1]:

- For the general population, reduce sodium intake to less than 100 mmol/day (2300 mg sodium/day).
- For those patients with or at risk of CVD, reduce sodium intake to less than 70 mmol/day (1550 mg sodium/day).

These recommendations are in line with the National Health and Medical Research Council (NHMRC) recommendations of an upper limit (UL) of sodium of 100 mmol/day and a suggested dietary target (SDT) of 70 mmol/day to reduce chronic disease risk.[2]

These values were selected with the intention of preventing hypertension, based on available epidemiological data that demonstrated a relationship between dietary sodium intake and blood pressure. The Dietary Approaches to Stop Hypertension (DASH) study[3], intervention trials[4,5], several meta-analyses and systematic reviews[6-12], and the Intersalt study[13] strongly supported selection of the NHMRC UL and SDT.

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 already present. Raised blood pressure is one of the most common and preventable risk factors for CVD.[14] The 1999–2000 Australian Diabetes, Obesity and Lifestyle Study measured blood pressure and the results indicated that 30% or 3.7 million Australians aged 25 years or over had high systolic or diastolic blood pressure or were on medication for high blood pressure—32% of males and 27% of females. The proportion of males and females with high blood pressure increased with age.[15] 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 increased physical activity, reduced salt intake, weight loss, moderate alcohol intake, increased potassium intake and an overall healthy eating pattern. In fact, there are data to suggest that these measures might even prevent development of hypertension in the first place.[16]

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 daily intake of sodium can be measured. This measure can help guide Australians in their endeavour to reduce their sodium intake to recommended level. 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 NHMRC UL for sodium is taken as the reference level, then, Australians as a group consume too much salt. Average sodium intakes among ten different study populations ranged from 130–200 mmol/day.[17] In a 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.[18]

In the Intersalt study, an international cross-sectional analysis of 52 populations (over 10,000 individuals), four (isolated) centres had median sodium excretion rates below 52 mmol/day, one centre was below 100 mmol/day, while 47 centres had rates above the UL for sodium.[13,19] 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

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excretion showed a steeper rise in blood pressure with increasing age compared with those with lower sodium excretion rates. By contrast, in the four isolated centres with very low sodium excretion, median systolic blood pressure was also low (95–110 mm Hg) and slopes of blood pressure with age were negative or small and positive. It was thought that the relationship between sodium and blood pressure was weak in the mid-range of sodium intake but stronger at both the lower and upper extremes of sodium reported in various populations. Of the two centres with median sodium excretion rates between 50 mmol/day and 100 mmol/day, 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.[13,20] Furthermore, a median sodium excretion rate of 96 mmol/day implies that around half of these African-American subjects had sodium excretion rates above the UL.

Law and co-workers[21] 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. 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.[22] Based on the findings of these population studies, Law estimated that in older persons a 100 mmol/day change in sodium intake was associated with a 10 mm Hg change in systolic blood pressure, while in hypertensives the estimated pressure difference was 15 mm Hg.[23]

Salt loading and blood pressure

In a study conducted on 14 normotensive volunteers, experimental diets containing 1200–1600 mmol/day of sodium raised blood pressure in all subjects within three days.[24] 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 systolic blood pressure of 33 mm Hg over a 20-month treatment period (blood pressure in the control group did not change).[25] Blood pressure returned to baseline values in all animals during the six month period following cessation of salt supplementation.

Salt reduction and blood pressure

Many systematic reviews and meta-analyses have been conducted to assess the efficacy of salt reduction on hypertension[6-12], and whether advice on dietary salt restriction is effective in lowering blood pressure. [26,27]

He and MacGregor[28] conducted a meta-analysis of randomized controlled trials testing interventions that reduced salt intake for four or more weeks. Individuals classified as hypertensive achieved a median reduction in sodium excretion of 78 mmol/day and a fall in systolic blood pressure of 5 mm Hg. Individuals classified as normotensive achieved a median reduction in sodium excretion of 74 mmol/day and a fall in systolic blood pressure of 2 mm Hg. The authors concluded that lower salt intake was associated with achievement of lower blood pressure recordings (within the daily intake range of 3-12 g salt/day). Similarly, a meta-analysis by Law *et al*[29] estimated that sodium excretion reduction of 50 mmol/day can lower systolic blood pressure by an average of 7 mm Hg in hypertensive individuals and by 5 mm Hg in normotensive individuals. Jurgens and Graudal[30] found that for Caucasians with elevated blood pressure, short-term (4–52 weeks) sodium reduction decreases systolic blood pressure by about 4 mm Hg. Midgley *et al*[11] conducted a meta-analysis of 28 trials of hypertensive individuals, showing that a decrease in sodium excretion of 100 mmol/day was associated with a fall in systolic blood pressure of 3.7 mm Hg.

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Hooper *et al*[26] assessed the effects of using health professional advice to reduce dietary sodium and blood pressure. The analysis found that systolic blood pressure was reduced by 8 mm Hg at six to 12 months in those provided with low sodium advice compared to controls. Dickinson *et al*[27] also found advice to restrict dietary sodium reduced systolic blood pressure by 5 mm Hg in hypertensive subjects participating in short- to long-term interventions.

In a multicentre, randomised trial conducted in the United States, the effect of different levels of dietary sodium, in conjunction with the DASH diet, which is rich in vegetables, fruits, and low-fat dairy products, was studied in persons with and in others without hypertension.[3] 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.

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.[31] Salt sensitivity is commonly reported in certain population groups, especially those with renal disease, diabetes, obesity, hypertension, older aged and in African-American populations.[31,32] A study following-up subjects for almost 30 years found that salt sensitivity of blood pressure was a novel risk factor for mortality.[33] The genetic bases for certain inherited forms of 'salt-sensitive' hypertension, such as glucocorticoid-remediable aldosteronism and Liddle's syndrome, have 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.[34] 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.[35] It has been shown that the sodium content of processed foods can be reduced without loss of product acceptability.[36] 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 wellbeing after commencing dietary sodium reduction.[37]

Some widely quoted epidemiological data have been interpreted to suggest possible adverse outcomes associated with dietary salt restriction. For example, in an American study, men treated with antihypertensive medication reported higher rates of myocardial infarction at lower salt intakes.[38] This study has been criticised because 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.[39] 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.[40,41] The validity of these results have been challenged for several reasons: 1) the dietary data were self-reported; 2) salt intake was defined by a single baseline 24-hour urine sample with no measure of table or cooking salt that may have been added; 3) 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 NHMRC recommendations 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.[42] The therapeutic effect of antihypertensive agents, especially beta-adrenoreceptor blockers[43] angiotensin converting enzyme inhibitors[44] 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.[45] Patients with hypertension who reduce dietary salt intake after becoming pregnant may demonstrate a paradoxical rise in blood pressure[45], 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.[45] 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%. In the Western diet, approximately 80% of sodium compounds we consume, mainly sodium chloride, are added to foods during processing.[46,47] Patients with hypertension should be advised to lower salt intake by a number of strategies such as:

- avoiding adding salt to food at the table and when cooking
- buying mainly plant-based foods, foods normally processed without salt, and 'low salt' or 'no added salt' groceries
- learning how to read nutritional information panels (NIPs) on food packages in order to determine sodium content. NIPs display sodium content of foods—not the salt content. (Refer to the Heart Foundation's "Reading labels for healthier eating" on **www.heartfoundation.com.au**.)

Low salt and low sodium foods are defined by the Australian Food Standards Code as having a sodium content of **120 mg/100 g 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. 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 snacks, seasonings, processed foods and take-away foods, which are high in salt.

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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.

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