

The Dietary Guideline with Great Therapeutic Potential

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Prescribing “salt restriction” for patients with salt-related health problems—using diets measuring sodium content and portion sizes—has been notoriously unpopular and unreliable, and the only therapeutic alternative has been to prescribe diuretics. This article reports a new observation that total salt intake is low enough (sodium < 50 mmol/day) to be more effective and less troublesome than diuretics in people who follow the Australian dietary guideline to choose foods low in salt while using the definition of low salt foods in the Australia New Zealand Food Standards Code (Na ≤120 mg/100g). Low salt foods—eaten exclusively during human evolution—can usually abolish the severe vertigo of Meniere’s disorder as the sole treatment, and reverse the universal rise of blood pressure with age, preventing hypertension. This simple prescription for better food (a healthier salt intake) enables health professionals to obtain measurable and permanent clinical improvement in motivated patients. The full public health potential of better food will require a long period of gradual reform in food processing and the evolution of a new cuisine. Shoppers must know what they are buying, and Australia needs Britain’s traffic light labels that identify low salt foods at a glance with green lights for salt.

Key words: Salt, Sodium, Dietary guidelines, Prevention, Treatment

This article reports an accidental finding that simple advice intended for the whole population has a surprisingly powerful therapeutic effect when it is accurately defined. Total salt intake is low enough (sodium < 50 mmol/day) to be more effective and less troublesome than diuretics in treating salt-related health problems. Patients only need to follow the Australian dietary guideline to choose foods low in salt and comply at the same time with the definition of low salt foods in the Australia New Zealand Food Standards Code (Na ≤120 mg/100g).

Few could have predicted this discovery at any stage in the development of the dietary guideline on salt. The first edition of the guideline advised people to “use less salt” (Commonwealth Department of Health, 1982), but advice on discretionary salt was clearly inadequate after the demonstration later of the five-fold dietary predominance of the salt added to processed foods (James, Ralph, & Sanchez-Castillo, 1987).

The second edition advised people to “choose low-salt foods and use salt sparingly” (National Health and Medical Research Council, 1991). However, the position of table salt on the surface of the food has a disproportionately large effect on the taste (Beauchamp, Bertino, & Engelman, 1987) and on the adaptation of the palate both upwards (Bertino, Beauchamp, & Engelman, 1986) and downwards (Bertino, Beauchamp, & Engelman,

1982). It is relatively easy to give up discretionary salt without missing it, and for the present (third) edition my co-author and I recommended the simpler guideline to “choose foods low in salt” (Riley & Beard, 2003, pp. 133-150). By implication, no added salt is recommended either in cooking or at the table.

A note on units of measurement

One of the obstacles to clear thinking about salt is the present use of multiple units of measurement for salt and sodium. It is largely the result of administrative negligence, which needs to be remedied (Appendix 1).

The empirical observation

The Menzies Research Institute has a research interest in better self-management of chronic disease, and patients referred for salt-related health problems were offered this simple guideline and told that the Australia New Zealand Food Standards Code (FSC) happens to have an accurate definition of low salt foods—the sodium content must not exceed 120 mg/100g. No patients left without demonstrating their competence in finding the Nutrition Information Panel (NIP) on food labels and reading it accurately for sodium content.

The clinical routine included 24-hour urine collections to measure sodium, potassium and creatinine excretion at baseline and at follow-up. It

rapidly became clear that patients who applied the FSC definition to the salt guideline exclusively at all meals had 24-hour sodium excretion well below 50 mmol (Beard, 2007). Full dietary compliance typically produces male sodium excretion rates between 20 and 40 mmol (female 15 to 30 mmol). Women excrete from 25% to 30% less sodium than men (Beard, Blizzard, O'Brien, & Dwyer, 1997); the difference being attributable to smaller meals, due to smaller body size, muscle mass and energy requirement (Woodward et al., 1997).

The 24-hour sodium excretion rate is regarded as the most reliable index of dietary sodium intake, as about 90% of the intake is excreted in the urine under normal conditions of activity and sweating, so for men the excretion rate of 20-40 mmol/day is equivalent to an intake of about 22 to 44 mmol/day (about 500 to 1000 mg), which has previously been a very difficult and demanding diet to follow.

Much of the difficulty and poor compliance in the past can probably be attributed to the perception—by both prescribers and recipients—that salt “restriction” would be an artificial and unpleasant departure from a “normal” diet. The perception of artificiality was reinforced by the fact that patients had to add up the sodium content of measured portions in every meal throughout the day. Artificiality implied further that restriction could even be harmful if carried too far, as everybody knew that salt was essential for life, and assumed this meant added salt.

In the past it was also rare for any health professionals to follow their own advice—“normal” prescribers ate “normal” food—but, today, perhaps a million or more health professionals have prehypertension and hypertension. There is unequivocal evidence that they too could expect measurable benefit—like their clientele—from paying much closer attention to three of the dietary guidelines—a lot more fruit and vegetables, low fat and low salt (Appel et al., 1997; Sacks et al., 2001).

Prescribing low salt foods in primary care

Prescribing better food is so radically different from traditional “salt restriction” that it may be useful to outline one method that works well clinically. It is fundamentally easy—the message could hardly be shorter—just choose low salt foods. Moreover, fresh foods in their natural state are all low in salt, with rare exceptions (Appendix 2).

Fresh foods

Fruit: Every fruit on the market and in the home garden is low in salt.

Vegetables: With rare exceptions (Appendix 2) every fresh vegetable on the market and in the home garden is low in salt as long as no salt is added in the kitchen or at the table.

Meat: All the fresh meat on the market (except kidneys) is low in salt.

Fish: All fresh fish except marine invertebrates are low in salt.

Poultry: All fresh poultry is low in salt in its natural state, but “self-basting” poultry, although it may be uncooked, has been injected with brine like ham. Eggs are only marginally over the limit (sodium 133 mg/100g), and prescribers can tell patients one egg will not take a low salt meal above the limit.

Processed foods

Nearly all processed foods have too much added salt, with rare exceptions—a problem discussed in several Australian websites (Appendix 3) and in the author’s book (Beard, 2007).

Patients need reassurance on several common misconceptions:

- That *added* salt is essential for life
- That *added* salt is essential to good cuisine
- That their existing diet is already low in added salt.

Patients who thought added salt was essential for life will admit they would never consider giving salt to a breastfed baby. They are astonished to hear that breast milk has a sodium content of only 14 mg/100g, whereas low salt foods—safe to eat—can by definition have up to 120 mg/100g, which is almost 10 times more. Common foods like fresh carrots with no added salt have about three or more times more sodium than breast milk.

The practitioner or practice nurse can next teach patients to read the NIP in a few minutes, using a few empty boxes and cans carrying standard Australian and New Zealand food labels. Patients who start with an empty box of Kellogg Corn Flakes (sodium 720 mg/100g in 2008) will never forget the next revelation. They can discover for themselves with an empty box of another Kellogg breakfast cereal (Just Right Original) that the NIP shows a sodium content of only 30 mg/100g. Neither of these best-selling breakfast cereals taste

salty, yet in 2008 corn flakes had 24 times more salt than their highly palatable competitor Kellogg Just Right Original and six times more salt than a low salt food.

The misconception that added salt is essential to good cuisine is universal but understandable. Chefs and caterers have a very good reason to add salt—they are serving a public with heavily salt-adapted palates. None of their clientele was allowed in childhood to keep the discriminating palates they were born with. Patients can be told that the best salt substitute is an adapted palate, and that the palate adapts to changes in habitual salt intake so rapidly that adaptation is well advanced within four weeks, and occurs over a wide range in either direction—both upwards (Bertino et al., 1986) and downwards (Bertino et al., 1982). The palate recovers rapidly even after years of suppression by salt, and patients who loved olives and bacon wonder in a few weeks how they had ever tolerated anything so salty.

The misconception that “normal food” is already low in salt is also surprisingly common. It rapidly changes when patients start reading the NIPs on the food labels they find in the cupboard at home. Some consultant otolaryngologists refute this misconception by ordering a baseline 24-hour urine collection. They then tell patients they must get below 50 mmol to control vertigo. They prescribe the book (Beard, 2007) and leave further management to the referring practitioner.

Baseline results show remarkably wide random variation. This figure, for example, plots the 24-hour sodium and potassium excretion from a Hobart survey ($n=194$) selected systematically from the Hobart Electoral Roll (Beard et al., 1997). Except for one woman who followed the Pritikin diet they all said they were eating normal food.

The 12-fold range from 26 to 334 mmol/day arose because no one selected foods for sodium content. Random food selection makes sodium excretion in industrial societies vary within wide limits from day to day (Liu et al., 1979), and many or all of the six people with results below 50 mmol on the day of the survey would probably have changed places next day with perhaps six others who were above the 50 mmol line in this plot.

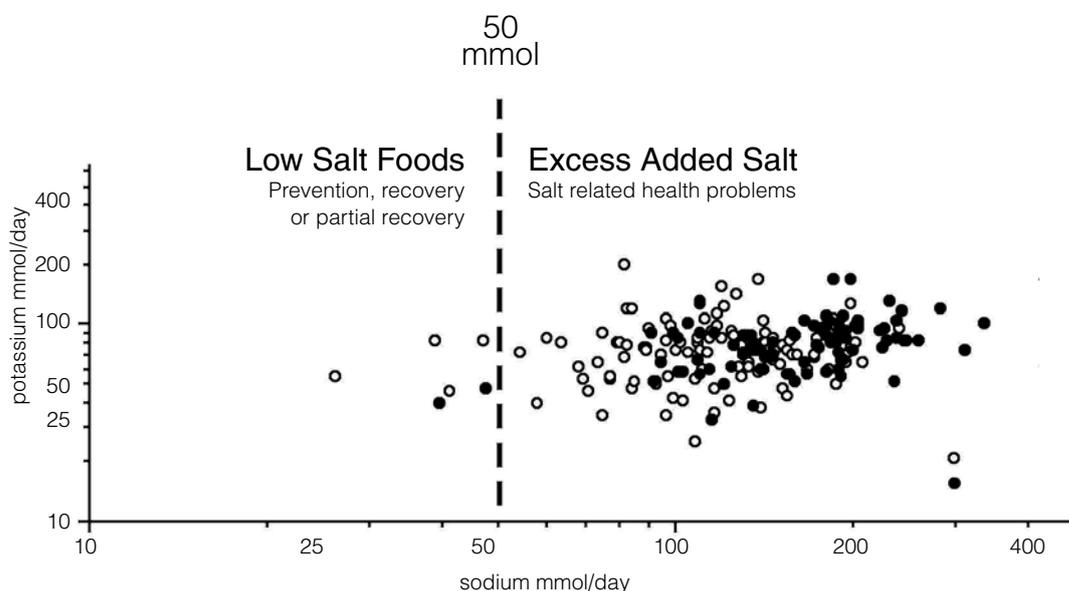
Consistent compliance with the salt guideline, however, takes motivated patients below 50 mmol/day very reliably, regardless of their baseline result.

Patients are shopping in a food market that has never catered for them, and a handout on low salt shopping is essential unless the book (Beard, 2007) is prescribed. Patients need detailed advice from the outset, especially on bread, and the bare minimum is the four-page Shopping Guide described in Appendix 3.

Clinically, the 50 mmol boundary varies with lean body mass

The 50 mmol threshold is an estimate of the population mean for clinically significant fluid

Figure 1:



retention, but clinical practice has to take account of wide individual variation in lean body mass. Small sedentary women reporting good dietary compliance, for example, have much lower sodium excretion rates than large muscular men with labouring occupations. For good control of vertigo in Meniere's disorder there can be no question that 40 mmol may be a safer limit for small sedentary women, whereas large muscular men with high energy requirements might tolerate sodium excretion rates as high as 65 mmol. Note that 50 mmol is approximately the logarithmic mean of the range 40-65 mmol.

The mean should be logarithmic because the progression in dosage needs to be logarithmic. In a stepwise progression of increasing daily intake from 20 to 200 mmol in equal arithmetic steps of 20 mmol, the first step would double the dose but the last would add only about 10%. Thus, a steady progression of equal arithmetic steps in rising dosage becomes increasingly trivial, and it is inconceivable that the last step would have as much biological effect as the first.

Equal steps in biological effect would be expected from an equal percentage increase in dose; in other words, from a logarithmic progression. This is dictated by mathematics (not biology). It is both expected and observed that drug dosage and stepwise taste perception of increasing concentrations of salt (Meiselman, 1968) require logarithmic progression. This is the reason for the log scale for sodium and potassium excretion in the figure.

The paradox of vertigo and evidence-based medicine

Clinical experience in Hobart has confirmed an old observation that sodium excretion below 50 mmol controls the vertigo of Meniere's disorder (Furstenberg, Lashmet, & Lathrop, 1934) and two Sydney hospitals have used it for this purpose for a decade, prescribing the Australian salt guideline with the Hobart educational materials and monitoring compliance with 24-hour urine collections. The Royal Prince Alfred Hospital finds a urinary sodium less than 50 mmol/day "more effective and less troublesome than diuretics" (Halmagyi & Cremer, 2000), and the number of patients requiring surgery for vertigo at St Vincent's Hospital has "decreased dramatically" with this approach (Fagan & Beard, 2007).

The paradox in evidence-based medicine is that the relief from vertigo is dramatic, more effective and less troublesome than diuretics, and yet has been reported with the caveat that "there has been no proper clinical trial" (Halmagyi & Cremer, 2000). The ethical and practical barrier to a randomised controlled trial is finding enough volunteers who will give informed consent to be randomly allocated to a control group at risk without warning of sudden attacks of severe vertigo, vomiting and prostration. In the 1960s the ethics committee intervened and stopped a London trial that had somehow recruited enough volunteers (Gibson, 1999, personal communication).

Cochrane himself—the leading pioneer of randomised controlled trials—readily accepted the best alternative evidence when there were practical or ethical obstacles to randomised trials (Cochrane, 1972). Another example of an important treatment that can never have a "proper clinical trial" is the dramatic cure of vertigo by vestibular nerve section, since major head surgery with sham operations would always be unethical in human control groups.

Evidence-based medicine must come to terms with this and accept the validity of the 50 mmol boundary for vertigo as readily as it accepts the validity of vestibular nerve section. Does any other food additive create severe ethical problems that make it impossible to test intakes at two levels—above and below a certain boundary?

An early review by Freis may help to explain the dramatic relief of vertigo in Meniere's disorder. The first measurable effect of adding salt to a natural (unsalted) diet is expansion of the extracellular fluid (ECF) volume, which is well-established at 1-2 litres by the time sodium excretion reaches or exceeds 50 mmol/day (Freis, 1976). Diuretics will contract the ECF volume when it is expanded, but there is osmotic equilibrium at salt intakes low enough to prevent expansion, and vertigo would not be expected (Harrison & Naftalin, 1968, pp. 85-101).

The interaction with drugs and a few rare diseases that affect sodium metabolism

It is important for many reasons to withhold diuretics. Sodium excretion below 50 mmol reverses the benefit/risk ratio of diuretics (van Brummelen, Schalekamp, & de Graeff, 1978)—making them inappropriate in primary care.

At sodium excretion rates below 70 mmol/day diuretics have little if any therapeutic effect that is not already provided by the diet (Morgan & Myers, 1981), while below 50 mmol/day the risk of prescribing diuretics is considered unacceptable (Brown & Whitworth, 1993; Mathew, Boyd, & Rohan, 1990; van Brummelen et al., 1978) except in life-threatening illnesses such as severe heart failure, where patients receive closer than usual supervision, with closer than usual attention to serum electrolytes. For ethical and medicolegal reasons, therefore, the Menzies Research Institute has introduced patients to the salt guideline with the FSC definition only when their doctors have agreed to withhold diuretics.

Excess salt and diuretics both have metabolic consequences and long-term side effects. The 50 mmol boundary has the outstanding advantage of having no excess salt; with none entering there is none to remove. This provides measurable clinical benefit without side effects (the popular belief that cramp is a side effect of avoiding salt needs to be revised).

Less mild cramp and less severe cramp occurred in the diet group of a randomised controlled trial at 37 mmol/day than they had reported at entry at 150 mmol/day (Beard, Cooke, Gray, & Barge, 1982)—even after being warned at entry to expect cramp—while the control group reported as much cramp as the diet group at entry at 175 mmol/day, with no change when finishing at 161 mmol/day, raising the question how much more salt they would need to improve their cramp.

There is evidence that cramp does accompany rapid change in sodium transport, regardless of the direction of change—it is a recognised adverse effect of diuretics, and is common during renal dialysis—while some patients who live below the 50 mmol boundary have reported cramp within eight hours after a salty meal (a sudden upward change (Beard, 2007, pp. 181-183).

Diuretics have significant adverse effects, which may occur at any level of salt intake (Mathew et al., 1990). When the ALLHAT study recommended thiazide diuretics as the first line of treatment for hypertension, it paid too little attention to adverse effects (Scott & Stowasser, 2003), but in Australia the Heart Foundation guide to management of hypertension 2008 prescribes a lower salt intake for all patients with hypertension, with a ceiling of 65 mmol/day, and withholding diuretics in order

to avoid their adverse effect on glucose tolerance (National Heart Foundation of Australia [National Blood Pressure and Vascular Disease Advisory Committee], 2008).

Interaction with other drugs

The 50 mmol boundary potentiates most antihypertensive drugs except calcium channel blockers. It usually potentiates ACE inhibitors and angiotensin antagonists well enough to permit prescribers to reduce the dose and side effects, and in some cases to discontinue these drugs.

A dose of lithium carbonate that is therapeutic at a normal salt intake usually becomes an overdose below the 50 mmol boundary. The prescriber (usually a psychiatrist) needs to be made fully aware beforehand of any proposal for a change in salt intake and invited to supervise the adjustment of dose.

Interaction with medical conditions

Pregnancy was normal throughout human evolution without added salt, but this is a time of turbulent change in the hormonal control of sodium metabolism. After the onset of pregnancy many obstetricians are cautious about using diuretics or making an abrupt reduction in salt intake, as paradoxical deterioration in toxemia has been reported (Gallery, Hunyor, & Gyori, 1979). Trouble is unusual in normal pregnancy; for example, 42 women in a Dutch study dropped to 20 mmol/day from the fourteenth week of pregnancy until after delivery without incident (Stegers et al., 1991).

A few rare conditions affect salt metabolism, including Addison's disease, salt-losing nephritis, Bartter's syndrome, cystic fibrosis, gastro-intestinal fistula and ileostomy. The salt intake of patients with these conditions must be high enough to replace abnormal losses.

The past and present of the 50 mmol boundary

There is evidence that primates evolved mainly on a diet of fruit and vegetables, and even the heavy use of meat could not have taken human hunter-gatherers above 30 mmol/day (Eaton & Konner, 1985).

Modern humans (*Homo sapiens sapiens*) date from about 40,000 years ago, having evolved like other terrestrial mammals on food with no added salt. There is no evidence that they used salt as a

food additive for about another 30,000 years until lakeside salt was harvested in 6000 BC in China. Agriculture and language had developed earlier, including some ancient written languages that have no word for salt.

For millennia, salt was expensive and poverty widespread, and it is difficult to estimate what small proportion of the population would have exceeded the 50 mmol boundary.

The mean 24-hour sodium excretion rate in modern industrial societies exceeds 50 mmol by a big margin, being about 130 mmol for women and 170 mmol for men (Beard, Blizzard et al., 1997; Beard, Woodward et al., 1997). This great excess is recent and unprecedented, and would not have been feasible before the industrial revolution. For example, giving the whole American population of 300 million an average daily supplement of 90 mmol needs an annual production of over 500,000 tonnes (90 mmol is contained in one level metric teaspoonful of salt weighing 5.4 g).

The lapse of barely more than 10 generations since the industrial revolution has allowed no time for genetic adaptation, and over half of the adult population of an industrial society has at least one of over 20 diagnosable health problems caused or aggravated by salt (www.saltmatters.org).

The two most common salt-related health problems are hypertension, affecting 3.7 million Australian adults (Australian Institute of Health and Welfare, 2001, p. 80) and prehypertension, affecting about 3 million (National Heart Foundation of Australia, 1990, p. 44); these two conditions alone affect over half of the 13 million who voted in the 2007 election. There are over 20 other miscellaneous salt-related health problems including an estimated quarter of a million women with severe premenstrual syndrome (5% of the female population aged 15 to 45) and an estimated 40,000 with the vertigo of Meniere's disorder. We lack reliable prevalence data on the rest of the salt-related health problems.

The high prevalence, severe sequelae and higher mortality of both prehypertension and hypertension (Havas, Dickinson, & Wilson, 2007) make them by far the most serious of the salt-related health problems. When prehypertension (blood pressure between 120/80 mm/Hg and 140/90 mm/Hg) was called high normal blood pressure it seldom received any treatment (Joint National Committee, 2003), but the landmark DASH-Sodium

study—a randomised controlled trial that provided all the food—showed convincingly that a sodium intake of 65 mmol/day reverses prehypertension and can therefore literally prevent hypertension (Sacks et al., 2001).

The aim of DASH-Sodium was to test a notionally feasible (not necessarily ideal) salt intake for treating and preventing hypertension, and for that purpose the likelihood of some fluid retention at 65 mmol/day (Freis, 1976) was ignored.

The DASH-Sodium diet used low salt bread with a sodium content of 36 mg/100g (Obarzanek, 2003, personal communication), but was nevertheless so well-accepted that a US Government website recommended the entire population adopt a habitual sodium intake below 65 mmol (www.nhlbi.nih.gov/new/press/may17-00.htm). Although it has been on the internet since 2001, it is not clear that very many Americans are adopting it.

DASH-Sodium looks like other artificial diets and is never described as better food. We can hardly expect 300 million Americans to follow a prescribed diet by measuring serving sizes and counting milligrams of sodium at every meal. America has not yet found a practical way to ask normal people to replace their regular meals with a diet that would give them a sodium intake of 65 mmol/day.

However, Australia has discovered by accident a simple way for motivated patients to get below 50 mmol/day and stay there. And a dietary guideline by definition gives people better food.

Potential future of the 50 mmol boundary

Public health needs to accommodate the 50 mmol boundary because populations that live above it are at risk of all salt-related health problems associated with fluid retention. Above 50 mmol some degree of mild subclinical oedema is universal (Freis, 1976), and the only available option for preventing or partly reversing it is to put the whole population on a diuretic or alternatively recommend a return to the low salt foods that were eaten exclusively during human evolution. A thiazide diuretic has been seriously suggested as one component of a “polypill” to counteract the worst features of the industrial diet (Wald & Law, 2003) as an alternative to improving it. Better food would make mass medication unnecessary, and the polypill was designed expressly for treating presymptomatic cardiovascular disease. It would do little to prevent

the growing nutritional epidemics of obesity, metabolic syndrome and Type 2 diabetes.

We have seen that a marginally higher boundary of 65 mmol will reverse hypertension and prehypertension, but we have no dietary guideline for healthy people to hold their sodium excretion reliably inside a boundary of 65 mmol/day.

The DASH studies highlighted the vagueness of national and international dietary guidelines. They showed that the typical American diet can prevent or partly reverse hypertension and prehypertension if it has far more fruit and vegetables than usual and is low in fat as well as very reduced in salt (65 mmol/day [Appel et al., 1997; Sacks et al., 2001]), but these prescriptions were precise only for sodium and vague for fruit, vegetables and fat. Australian guidelines are equally vague for fruit, vegetables and fat, and were vague for salt until the guideline was linked with the definition of low salt foods in the Food Standards Code.

The UK Food Standards Agency (FSA) has broken new ground with its traffic light labels. To correspond with the pre-existing low salt boundary, they have replaced vagueness with precision for fat, saturated fat and sugar. The wording of the dietary guidelines could share this new precision if linked with traffic light food labels.

Low salt foods carry a green light (low) for salt if the sodium content is low, and the amber light distinguishes a new level (“moderate” or medium”) between low (green light) and “high” (red light). The colours are based on the concentration of each nutrient in grams per 100 g or per 100 mL, and the boundaries are set and kept under evidence-based review by an expert panel.

Critics may object that sharp boundaries are illusory in biology, but front-of-pack food labels have to be simple. All the degrees of difference are still shown at the back of the pack in the Nutrition Information Panel (NIP), where health professionals and the shoppers who know what sodium is can discover perhaps that a sodium content of only 133 mg/100g required the amber light for salt (“only slightly amber”).

There is widespread agreement that success in the battle against childhood obesity will need food labels that tell the whole population clearly what they are buying. Green, amber and red lights give an unambiguous message to everybody without reading glasses—and regardless of age, income or education—about the nutrient density of the

four major nutrients causing the massive modern epidemics of preventable disease. Even chubby children in the gathering epidemic of childhood obesity can see why their mothers refuse to buy foods with red lights for fat.

The Australia 2020 Summit and the Australian Medical Association strongly support traffic light labels, and there is evidence that they would have bipartisan support (May, 2007).

In primary care the boon of traffic lights is not limited to salt. Patients with a cholesterol problem can select processed foods with a green light for saturated fat, and countless other examples would transform dietary prescribing in primary care.

The food industry is promoting an alternative (the DI Counter) without these advantages, and it would be naïve to forget that public health is fragile in democracies with free markets, and politically weak compared with the enormous power of vested interests and commercial profits (Nestlé, 2002). Even one red light on a breakfast cereal advertised as “healthy” is anathema to the food industry, which has massive economic power and a permanent and well-funded political lobby and will go to any length to prevent red lights appearing on any of its leading brands.

We must acknowledge at the same time that food companies are by no means devoid of social responsibility. To its great credit Kellogg (Australia), for example, has worked for over a decade on reducing the sodium content of its breakfast cereals. In 1997, Kellogg’s Corn Flakes had a sodium content of 1100 mg/100g, which was reduced progressively to 1020, 900, and 820 mg/100g, and has currently reached 720 mg/100g. The most spectacular achievement was to reduce the sodium content of Just Right from 284 mg/100g to 49 mg/100g (an 83% reduction) in one step in 1998, a time when Kellogg (UK) were selling Just Right with 600 mg/100g. As noted previously this cereal is now called Just Right Original and its Australian sodium content is only 30 mg/100g.

But the prime responsibility of every company is to its shareholders, and reformulation of processed foods is time-consuming, expensive and never free from commercial risk in a competitive market. Kellogg (Australia) foresees significant financial loss if traffic lights became mandatory when every box of Corn Flakes still had a sodium content high enough to give it a red light for salt, and if Just Right Original still had 30% sugar, requiring a red

light for sugar. Already a rival breakfast cereal, Sanitarium Weet-Bix, would have three green lights and only one amber light (sodium 290 mg/100g), while Lite-Bix and Weet-Bix Kids are already low in salt and would both have four green lights.

Kellogg and the Australian Food and Grocery Council can represent traffic light labels politically as an economic disaster severe enough to drive some companies into manufacturing off-shore. Conversely, the DI Counter, consisting of a row of percentages without warning shoppers what they mean in nutrient density, would be a disaster to public health.

Motivated patients can use the NIP to identify low salt foods, but the general public needs the simple, obvious and unmistakable message that some foods are much too salty, some marginal and a few with green lights for salt are already meeting the international definition of low salt foods, and low enough in salt to be safe to eat without measurement.

Conclusion

Humans evolved on low salt foods, and their total daily sodium excretion could not have exceeded 30 mmol (Eaton & Konner, 1985). For barely more than 10 generations the industrial revolution has given industrial societies up to a 12-fold increase in habitual sodium excretion, with no time for genetic adaptation. The prevalence of diagnosable health

problems caused or aggravated by salt is greater than half the adult population.

The treatment of salt-related health problems has been limited to two options—salt “restriction” and diuretics. Restriction is unpopular, unreliable and often neglected, and many doctors prescribe diuretics without even mentioning salt.

This article reports a simple dietary guideline with a powerful and more reliable therapeutic effect in patients who have the motivation to decipher the NIP. It can be summarised in eight words—choose low salt foods (sodium up to 120 mg/100g)—and a 294-page book (Beard, 2007) and several websites (Appendix 3) answer most of the questions. In motivated patients this simple dietary guideline is more effective and less troublesome than diuretics.

The benefit to public health is potentially enormous. The UK has front-of-pack labels that identify the salt content of foods instantly with green, amber and red traffic lights. If Australia can introduce them in spite of the strong opposition of the food industry, with adequate publicity, this guideline can provide the underpinning for a long period of gradual reform in food processing—and the evolution of a new cuisine—that will have the potential to limit and perhaps ultimately prevent the almost universal rise of blood pressure with age.

Appendix 1: Causes of confusion

Two main causes of confusion are the distinction between salt and sodium and the use of multiple units of measurement for weight and volume (grams, milligrams and teaspoons) and molar SI units (millimoles).

1. Salt and sodium

An ingredient list needs to speak of salt, and can use additive numbers without naming other sodium compounds.

The front-of-pack label is a new idea to make it clear to all shoppers what they are buying, including all the people who have never learnt chemistry at school. Front-of-pack labels must confine themselves to language people already understand without explanation. The word sodium is inappropriate at the front of the pack because it is not universally understood.

The Australian Nutrition Information Panel (NIP) at the back of the pack is informative for health professionals and others who remember enough of their school chemistry to understand what sodium means. The Australian NIP is a world leader with its mandatory disclosure of sodium in mg/100g, which allows two foods to be directly compared for sodium content.

Text intended for health professionals can discuss both *salt* and *sodium* according to the context. The UK decision, however, to include notional salt content in the Nutrition Information Panel (NIP) can be criticised as artificial (done without a chloride analysis), unnecessary and confusing. It required the UK definition of low salt food to be altered from the international definition ($\text{Na} \leq 120 \text{ mg}/100\text{g}$) to a local definition ($\text{NaCl} \leq 0.3 \text{ g}/100\text{g}$), which is $\text{Na} \leq 118 \text{ mg}/100\text{g}$.

The difference between salt and sodium in units of mass leads to many mistakes. As explained below, the difference disappears in molar SI units, because 100 mmol of salt contains 100 mmol of sodium and 100 mmol of chloride (Beard, 2007, pp. 241-246).

The policy while waiting for front-of-pack labels. Food regulations define low salt foods by their sodium content, and it is easy to teach patients to identify them using the NIP. Experience at the Menzies Institute is that motivated patients can readily learn how to read the NIP for sodium well enough to select low salt foods very reliably.

2. Molar SI units

The SI (an abbreviation of its French title) is the International System of Weights and Measures. All countries that legislated for metric conversion—including the United States—adopted the SI. The SI establishes only one appropriate unit for each physical quantity, replacing the duplications and ambiguities of the traditional metric system with a self-consistent and universal language of simplified terminology. In theory the SI eliminates confusion, but in practice every country that legislated to introduce the SI has failed to finish implementing it.

All Australian medical laboratories adopted the SI in 1974 and for over three decades have reported clinical results to referring practitioners in mmol/litre for all substances of known molecular or atomic weight. But as late as 2008:

- UK reports sodium in food in grams/100g
- USA reports sodium in food in mg/serve
- Australia and New Zealand report sodium in food in both mg/serve and mg/100g.

This is inexcusable and in breach of the legislation in all three countries. This article has had to report sodium in food in mg/100g and sodium in urine in mmol/24 hours, otherwise it would have been unintelligible to readers in Australia and New Zealand. Confusion is due entirely to administrative failure, which ought to be remedied (Beard, 2007, pp. 241–246).

Appendix 2: Natural foods that may be high in salt

Invertebrate seafood—shellfish, squid, shrimps, prawns and crayfish—are less able to defend themselves against the high salt content of seawater (1068 mg/100g), and their sodium content may exceed 300 mg/100g even when freshly caught, rising further if stored in refrigerated seawater. Some Australian oysters have over 900 mg/100g.

The flesh (muscle) of marine and terrestrial vertebrates is low in salt, except when tuna or sardines are stored in refrigerated seawater in the hold of a ship that remains at sea for several weeks. Two other fresh animal foods with sodium above 120 mg/100g are hens' eggs (133 mg/100g) and kidneys (160-200 mg/100g).

Plants belonging to the beet family have salt-tolerant relatives that can grow in sand dunes. Silver beet and the closely related Swiss chard and beetroot (red beet) are low in salt like other vegetables when grown in home gardens and normal market gardens, but they may be too salty from passive absorption when grown commercially in places where the soil and/or irrigation water are too salty to grow anything else.

This Appendix has relevance mainly for patients who are taking great care to prevent the vertigo of Meniere's disorder. Probably none of these exceptions would normally be very likely to take a whole meal above the low salt limit unless the patient happened to be very fond of invertebrate seafood and unaware of the problem.

Appendix 3: Resources available for helping patients

The bare minimum is a four-page shopping guide that can be downloaded and printed (preferably in colour) from the Internet by clicking Shopping Guide in www.saltmatters.org. This is also available in hard copy as a useful handout from the publisher, the Meniere's Support Group of Victoria Inc., 4/18-28 Skye Road, Frankston, Vic 3199, phone (03) 9783 9233, FAX (03) 9783 9208, email info@menieres.org.au

The Meniere's Support Group of Victoria (MSGV) also sells *Salt Matters: a consumer guide* (first printing 2004) and *Salt Matters: the killer condiment* (second printing 2007), The Dizzy Chef, a low salt cookbook published by MSGV, and a combined CD (for computers) and SVCD (for DVD players) of resource materials called Low Salt – Better Health, which was put together by a working party of multi-skilled volunteers in SMANZ (Salt Matters – Australia New Zealand), described in the next paragraph.

SMANZ is the title adopted by a completely informal email discussion group on choosing low salt foods, which is open to anyone to join. For a description of SMANZ and instructions on joining click Email Discussion Group in www.saltmatters.org. SMANZ offers a 30-page shopping guide that can be downloaded and printed from www.smanz.info

Two very informative amateur websites on how and where to find low salt foods—along with colour pictures of food labels—are at www.findlowsaltfood.info and <http://home.exetel.com.au/sharksaus>

References

- Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., et al. (1997). A clinical trial of the effects of dietary patterns on blood pressure. *New England Journal of Medicine*, 336(16), 1117–1124.
- Australian Institute of Health and Welfare. (2001:80). *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.
- Beard, T. C. (2007). *Salt matters: The killer condiment*. Sydney: Hachette Livre.
- Beard, T. C., Blizzard, L., O'Brien, D. J., & Dwyer, T. (1997). Association between blood pressure and dietary factors in the Dietary and Nutritional Survey of British Adults. *Archives of Internal Medicine*, 157(2), 234–238.
- Beard, T. C., Cooke, H. M., Gray, W. R., & Barge, R. (1982). Randomised controlled trial of a no-added-sodium diet for mild hypertension. *Lancet*, 2(8296), 455–458.
- Beard, T. C., Woodward, D. R., Ball, P. J., Hornsby, H., von Witt, R. J., & Dwyer, T. (1997). The Hobart Salt Study 1995: Few meet national sodium intake target. *Medical Journal of Australia*, 166, 404–407.
- Beauchamp, G. K., Bertino, M., & Engelman, K. (1987). Failure to compensate decreased dietary sodium with increased table salt usage. *Journal of the American Medical Association*, 258(22), 3275–3278.
- Bertino, M., Beauchamp, G. K., & Engelman, K. (1982). Long-term reduction in dietary sodium alters the taste of salt. *American Journal of Clinical Nutrition*, 36, 1134–1144.
- Bertino, M., Beauchamp, G. K., & Engelman, K. (1986). Increasing dietary salt alters salt taste preference. *Physiology & Behavior*, 38(2), 203–213.
- Brown, M. A., & Whitworth, J. A. (1993). Think again about combination diuretics. *Australian Prescriber*, 16(1), 4–5.
- Cochrane, A. L. (1972). *Effectiveness and efficiency: Random reflections on health services*. London: The Nuffield Provincial Hospitals Trust.
- Commonwealth Department of Health. (1982). *Dietary guidelines for Australians*. Canberra: Australian Government Publishing Service.
- Eaton, S. B., & Konner, M. J. (1985). Paleolithic nutrition: A consideration of its nature and current implications. *New England Journal of Medicine*, 312(5), 283–289.
- Fagan, P. A., & Beard, T. C. (2007). Effective control of vertigo in Meniere's disorder. *The Balancer* (49), 8–11.
- Freis, E. D. (1976). Salt, volume and the prevention of hypertension. *Circulation*, 53, 561–563.
- Furstenberg, A. C., Lashmet, F. H., & Lathrop, F. (1934). Ménière's symptom complex: Medical treatment. *Annals of Otology, Rhinology and Laryngology*, 43, 1035–1046.
- Gallery, E. D. M., Hunyor, S. N., & Gyori, A. Z. (1979). Plasma volume contraction: A significant factor in both pregnancy-associated hypertension (pre-eclampsia) and chronic hypertension in pregnancy. *Quarterly Journal of Medicine*, 192, 593–602.
- Halmagyi, G. M., & Cremer, P. D. (2000). Assessment of dizziness. *Journal of Neurology, Neurosurgery and Psychiatry*, 68, 129–134.
- Harrison, M. S., & Naftalin, L. (1968). *Meniere's disease: Mechanism and management* (pp. 85–101). Springfield, Illinois: Charles C Thomas.
- Havas, S., Dickinson, B. D., & Wilson, M. (2007). The urgent need to reduce sodium consumption. *Journal of the American Medical Association*, 298(12), 1439–1441.
- James, W. P. T., Ralph, A., & Sanchez-Castillo, C. P. (1987). The dominance of salt in manufactured food in the sodium intake of affluent societies. *Lancet*, 1, 426–429.
- Joint National Committee. (2003). The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Journal of the American Medical Association*, 298, 2560–2572.
- Liu, K., Cooper, R., McKeever, J., McKeever, P., Byington, R., Soltero, I., et al. (1979). Assessment of the association between habitual salt intake and high blood pressure: Methodological problems. *American Journal of Epidemiology*, 110(2), 219–226.
- Mathew, T., Boyd, I., & Rohan, A. (1990). Hyponatraemia due to the combination of hydrochlorothiazide and amiloride (Moduretic): Australian spontaneous reports 1977–1988. *Medical Journal of Australia*, 152(6), 308–309.
- May, M. (2007). Private member's bill. *Hansard, Monday 17 September*, 12–13.
- Meiselman, H. L. (1968). Adaptation and cross-adaptation of the four gustatory qualities. *Perception & Psychophysics*, 4, 368–372.
- Morgan, T., & Myers, J. (1981). Diuretics. *Current Therapeutics*, 22, 93–97.
- National Health & Medical Research Council. (1991). *Dietary guidelines for Australians*. Canberra: Australian Government Publishing Service.
- National Heart Foundation of Australia. (1990:44). *Risk factor prevalence study: Survey no. 3–1989*. Canberra: Author.
- National Heart Foundation of Australia (National Blood Pressure and Vascular Disease Advisory Committee). (2008). *Guide to management of hypertension 2008*. Quick reference guide for health professionals.
- Nestlé, M. (2002). *Food politics: How the food industry influences nutrition and health*. Berkeley, California: University of California Press.
- Riley, M. R., & Beard, T. C. (2003). Choose foods low in salt. In: *Food for health: Dietary guidelines for Australian adults* (pp. 133–150). Canberra: National Health and Medical Research Council.
- Sacks, F. M., Svetkey, L. P., Vollmer, W. M., Appel, L. J., Bray, G. A., Harsha, D., et al. (2001). Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *New England Journal of Medicine*, 344(1), 3–10.
- Scott, I., & Stowasser, M. (2003). Are thiazide diuretics preferred as first-line therapy for hypertension? An appraisal of the Antihypertensive and Lipid-lowering Treatment to Prevent Heart Attack Trial (ALLHAT). *International Medical Journal*, 33(7), 327–330.
- Steegers, E. A. P., Van Lakwijk, H. P. J. M., Jongsma, H. W., Fast, J. H., De Boo, T., & Eskes, T. K. A. B., et al. (1991). (Patho)physiological implications of chronic dietary sodium restriction during pregnancy: A longitudinal prospective randomized study. *British Journal of Obstetrics and Gynaecology*, 98(10), 980–987.

- van Brummelen, P., Schalekamp, M., & de Graeff, J. (1978). Influence of sodium intake on hydrochlorothiazide-induced changes in blood pressure, serum electrolytes, renin and aldosterone in essential hypertension. *Acta Medica Scandinavica*, 204, 151-157.
- Wald, N. J., & Law, M. R. (2003). A strategy to reduce cardiovascular disease by more than 80%. *British Medical Journal*, 326, 1419-1423.
- Woodward, D. R., Beard, T. C., Ball, P. J., Hornsby, H., von Witt, R. J., & Dwyer, T. (1997, 14-17 May). *Should the male and female RDI for sodium be the same?* [abstract]. Paper presented at the 16th Dietitians' Association of Australia National Conference, Hobart, Tasmania.
- www.nhlbi.nih.gov/new/press/may17-00.htm. (Retrieved September 14, 2008).
- www.saltmatters.org. Follow links to *Why Salt Matters* and *Salt Related Health Problems* (Retrieved September 14, 2008).

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