Alderman does not address the recent DASH-sodium trial that measured the effects of diet and salt on blood pressure. This trial randomized subjects to two diets—a control diet like a typical American diet, and a modified ‘Mediterranean’ diet. Subjects were rotated through three levels of salt.

The protocol for this trial—published two years after the trial was underway—states that one of the two primary research questions was the effect of the DASH diet at various levels of salt intake. Additivity and linearity of effects of diet and salt were protocol-specific secondary questions. Such questions are given short shrift in the final report, which stresses the impact of reducing salt intake. The accompanying news release is even more one-sided.

Published data summaries, sketchy as they are, indicate that adopting the DASH diet will lead to a marked reduction in systolic blood pressure, at ordinary levels of salt intake; but there are striking non-linear interactions between salt and diet. Indeed, one way to read the data is this: with a good diet, salt has almost no impact on systolic pressure. Practically no attention is given to the effect of salt on diastolic pressure, which is independently associated with cardiovascular mortality, and is the benchmark for estimating cardiovascular mortality, and is the benchmark for estimating public health benefits from blood-pressure reduction.

Moreover, the study population substantially over-represents salt-sensitive demographic groups; extrapolating to the general population is therefore unwarranted, despite sweeping claims. The response of the DASH investigators to these points is unconvincing. The INTERSALT investigators refuse to make the data public. So do the DASH investigators, who even decline requests for data on diastolic pressure. The take-home message from the DASH trials, despite the NHLBI spin-meisters, is that diet matters more than salt. The effect of salt has been confirmed by a clinical trial on secondary prevention, with endpoints defined in terms of mortality and morbidity.

Funding agencies and medical journals have taken a stronger position favouring the salt hypothesis than is warranted, raising questions about the interaction between the policy process and science. Dietary advice on salt is presented to the public with rhetorical force that is not in any reasonable balance with the evidence. How did this come about?

As we see it, public policy programmes, once in place, rapidly develop a life of their own. The possibility of benefits becomes probability, and probability becomes certainty. Ambiguity must be suppressed—just as data must be hidden—because the public is too easily confused. Only professionals can be trusted to weigh the evidence, and not all professionals at that. Besides, where is the harm in salt reduction?

The harm is to rational discourse. The appearance of scientific unanimity is a powerful political tool, especially when the evidence is weak. Dissent becomes a threat, which must be marginalized. There soon comes about the pretence of public policy based on science, without the substance. Salt is only one example of this phenomenon.

---

**References**


---

© International Epidemiological Association 2002 Printed in Great Britain

International Journal of Epidemiology 2002;31:320–327

---

**Commentary: Salt, blood pressure and health**

G MacGregora and HE de Wardenerb

The Yanomamo Indians still lead a life very similar to the last million or so years of our evolution, and like primitive man eat a diet that is very low in salt and saturated fat and high in fruit, vegetables and roots. The Yanomamo Indians are not

---

a Blood Pressure Unit, St George’s Hospital Medical School, Cranmer Terrace, London SW17 0RE, UK. E-mail: gmacgregor@sghms.ac.uk

b Department of Clinical Chemistry, Imperial College of Technology and Medicine, Charing Cross Campus, St Dunstan’s Road, London W6 8RP, UK. E-mail: h.dewardener@ic.ac.uk
overweight, do not smoke and are very fit. Their blood pressure does not rise with age although they spend much of their time fighting and are under great stress.\(^3\) This tribe does not develop vascular disease, although many die of infection. However, when they migrate to a Venezuelan or Brazilian town and adopt a western lifestyle, they, like native Americans, become overweight and develop diabetes and premature vascular disease. They appear therefore, to be a group which, though predisposed to vascular disease, is protected by the way they live. There are other similar examples\(^4\) which clearly indicate that cardiovascular disease (strokes, heart attacks and heart failure) could be entirely prevented if we changed our diet and lifestyle.

Approximately half of the population in the UK dies of cardiovascular disease\(^5\) yet very little is done to prevent this by changing lifestyle or diet. Clearly, much greater efforts are needed to try to understand what simple changes could have beneficial effects. To relate dietary variables such as salt, saturated fat, fruit and vegetable consumption to outcome is not easy because at present the intake of salt, fat, fruit and vegetables varies considerably from day to day, as does the blood pressure. Unfortunately it is not possible to set up dietary outcome experiments in which babies are randomized at birth, or ideally at conception, into one group that eats less salt or less fat and more fruit and vegetables, while another group continues on a ‘normal’ diet for life. Such experiments are never going to be done, we therefore have to rely on evidence from epidemiology, migration, intervention, treatment, animal and genetic studies. In spite of the difficulties of quantifying salt intake in an individual because of the variability of western diets and the variability of the blood pressure, the evidence that dietary salt is related to blood pressure, both in the rise that occurs with age and the number of people whose blood pressure is raised, is stronger than all other dietary variables.

Alderman has been one of the main proponents of the dangers of reducing salt intake. He encouraged the National Heart, Lung and Blood Institute (NHLBI) in the USA to set up a large workshop on sodium and blood pressure in January 1999 in order to critically review all of the current scientific evidence on salt. For reasons that are unclear Alderman did not attend the meeting which concluded that ‘a high sodium intake is associated with higher blood pressure levels, and other cardiovascular and non-vascular conditions continue to increase’, and that ‘a population wide strategy of reducing salt in the food supply is an important public health strategy that can lower blood pressure among populations’.\(^7\) Alderman’s current overview of the subject is highly selective. He ignores over 40 careful studies in unacculturated human populations who at one time consumed, or continue to consume, less than 3 grams of salt per day.\(^4\) In these populations blood pressure did not and does not rise with age. Furthermore, there are other unacculturated populations which demonstrate that it is the low salt intake which is responsible for the lack of rise in blood pressure, not some other aspect of unacculturation.\(^6,7\) Alderman points out that Kuna Indians appear to be an exception\(^8\) to the general experience that a high salt intake is associated with a rise in blood pressure with age but he does not mention that in this study 24-h urinary sodium excretion was not measured. The estimation of salt intake relied in part on the recollection of how many teaspoons of salt each person had added to their food. One of the reasons the large InterSalt study\(^9\) was set up with careful attention to methodology, including 24-h urine collection, was precisely because of the difficulty of estimating salt intake from dietary history. Alderman also refers to an observational study in Italian nuns where the blood pressure did not rise with age\(^10\) but he does not quote the investigators’ own conclusions. It appeared to them in order to avoid the customary rise in blood pressure with age it is necessary to live in a stress free environment, characterized by total silence, continuous meditation and isolation from society. It is noticeable that although the first account of these nuns appeared 10 years ago the findings have never been confirmed elsewhere.\(^11\)

The InterSalt study\(^9\) was set up to study a wide range of salt intakes across the world and measure blood pressure and other variables under well controlled conditions. By the time the study was completed there were only four communities who ate less than 3 grams of salt a day. The others were in a very narrow range of salt intake, approximately 7–12 grams of salt a day. The majority of these communities were eating a western diet in which salt intake varies so much from day to day that individual variations in sodium excretion may be considerable, e.g. more than five-fold.\(^12\) It follows that in many of these communities salt intake in any one individual might vary from day to day by the same amount as the variability between individuals. It is not surprising therefore that, in the InterSalt study, if one excludes the few communities who eat less than 3 grams of salt, and only considers those communities which consume between 7 and 12 grams of salt a day, there is no clear relationship with blood pressure. Alderman fails to point out that if all the communities are considered, there is a clear relationship between salt intake and blood pressure and the rise in blood pressure with age (Figure 1).

The only intervention study that has successfully managed to reduce salt intake remains the Portuguese study where two villages were studied.\(^13\) One village was given information on how to reduce salt intake, particularly in relation to processed foods, and it was given processed foods with less salt. Salt intake was reduced by approximately 50%, as judged by 24-h urinary sodium. Over two years this resulted in substantial differences
in blood pressure, compared to the control village where no reduction in salt intake was made (Figure 2).

Perhaps it is understandable that Alderman all but ignores all the animal work that relates salt intake to blood pressure. He reduces it to one dismissive sentence. There are now numerous studies in the rat, dog, chicken, rabbit, baboon and chimpanzee, all of which have shown that when there is a prolonged increase in salt intake there is an increase in blood pressure. Furthermore in all forms of experimental hypertension, whatever the animal model, a high salt intake is essential for the blood pressure to rise. A recent study was carried out in chimpanzees, the nearest relative to humans (98.8% genetic-homology). The normal salt intake of a chimpanzee, which weighs up to 50 kg, is less than 0.5 gram a day. When it was increased to 15 grams a day the blood pressure rose slowly and the rise became significant after one year when it was still rising. Blood pressure returned to normal when the salt intake was reduced (Figure 3). These results show that if the animal species most closely related to humans, which normally consumes a diet as low in salt as the one it (and the human race) is genetically programmed to eat, increases its salt intake into the same range as that of present day humans, they, like humans, develop hypertension.

Alderman now concedes that when salt intake is reduced there is a fall in blood pressure in both normotensive and hypertensive humans. He even concedes that a fall of ‘a few millimetres of mercury if sustained, assuming the method of its achievement induces no harm, could produce more reductions in morbidity and mortality than is currently achieved by treating high blood pressure’. His ensuing comment that this ‘possibility energises advocates of sodium restriction’ presumably indicates that nevertheless Alderman wishes to distance himself from these findings. It could be put forward that this is a reasonable uncritical reaction to meta-analyses which have included studies of extremely large changes in salt intake over periods of less than one week. Such manoeuvres have been known to stimulate the sympathetic nervous system. The inclusion of such short term studies, particularly in normotensives, is inappropriate when the recommendations for public health are for a modest reduction in salt intake from 10 grams to 5 grams a day over a lifetime, not a few days. A more recent meta-analysis that only included studies of modest and longer term reductions in salt intake, showed that the fall in blood pressure in normotensives was greater than in the previous meta-analyses. In the most rigorous trial (DASH Sodium Study) in which there were several hundred participants, and the daily sodium intake was well controlled, the fall in pressure was even greater (Figure 4). There is evidence that the full effect of salt restriction may not be seen within a month so that with longer term reductions in salt intake there may well be greater falls in blood pressure. In other words, there is every indication that the recommendations of a modest reduction of salt intake from 10 grams to 5 grams a day over a prolonged period of time has a pronounced effect on blood pressure, not only in the hypertensives but also in the normotensive population.

When discussing the other effects of ‘sodium restriction’, Alderman forgets to mention that in addition to, and independent of, raising blood pressure, a high salt intake increases the mass of the left ventricular wall, stiffens conduit arteries and thickens and narrows resistance arteries, including the coronary and renal arteries. A high salt intake is also directly related to the number of strokes, severity of cardiac failure, adhesiveness of platelets, carcinoma of the stomach and, to bone demineralization. Alderman misunderstands the relationship of sodium intake to vascular compliance; he conveniently reverses it—what has been found is that a reduction in sodium intake increases vascular compliance. He rightly but ominously points out that salt intake is well documented to relate closely to the renin angiotensin system. It is true that the renin angiotensin aldosterone system is one of the major compensatory mechanisms that maintains blood pressure and reduces the excretion of sodium, and thus when extracellular volume is reduced with diarrhoea or diuretics there is a rise in plasma renin. This is an entirely normal physiological response, and to try to intimate that it is abnormal, and harmful, for plasma renin to rise in response to a reduction in extracellular volume, is extraordinary. The Yanomamo Indians, who still live an evolutionary form of life and have a very low salt intake
(<0.5 g per day), have the highest renin and aldosterone levels measured. But they do not develop vascular disease. According to Alderman's hypothesis, such high levels of angiotensin and aldosterone should precipitate an acceleration of vascular disease at an early age. Animal experiments, however, have shown that it is the appropriateness of the level of renin and aldosterone to the blood volume or extracellular volume that may cause damage, not the absolute level of plasma renin activity and aldosterone which, according to Alderman, should be harmful. A modest reduction in salt intake lowers blood pressure in an identical way to diuretics and increases both renin and aldosterone to the same levels. Alderman needs to explain why he considers diuretics to be so beneficial, and yet according to him salt restriction is not.

Whilst it is true that non-acculturated societies have shorter life spans, this is not due to an increase in the incidence of cardiovascular disease, which they almost totally avoid, but to the greater risk of infection, particularly when exposed to western populations. It is true that the Japanese are an example of a society that has a longer life expectancy than many of those in the west. As the Japanese have a high salt intake, Alderman suggests that this illustrates that salt intake is not related to life expectancy. But the Japanese did not, and to a large extent still do not, eat much fat and they have a low plasma cholesterol which protects them from the development of vascular disease and atherosclerosis. Alderman fails to point out that the major causes of death in Japan are cerebral haemorrhage and cancer of the stomach, both of which are due to the high salt intake. Indeed, he appears to be unaware that in the 1960s there was a government campaign in Japan to reduce salt intake. It was successful in lowering blood pressure and causing large reductions in stroke mortality. It is very likely that if the Japanese were to reduce their salt intake further, whilst continuing to eat a minimum amount of saturated fat, they would live even longer than they do now.

Alderman fails to mention the North Karelia project in northern Finland where the incidence of cardiovascular disease was very high. In co-ordination with the food industry, a government-backed campaign has been successful in reducing salt and fat intake, increasing fruit and vegetable consumption and cutting cigarette smoking. This has resulted in a reduction in blood pressure and fat intake and significant falls in stroke and coronary heart disease mortality. Three-quarters of the fall in coronary heart disease and two-thirds of the fall in stroke mortality were due to the change in risk factors. This study clearly indicates that if we were prepared to change our lifestyle, particularly our diet, major reductions in cardiovascular mortality could ensue.

In a final attempt to obfuscate, Alderman once more revives the myth that a moderate reduction in salt intake is harmful. He again quotes completely inappropriate experiments in which the sodium intake of rats was so low that it stunted growth. In other words, the deleterious effect of aldosterone on cardiac fibrosis, etc, appears to be due to the associated retention of an excess amount of salt and water.

In view of Alderman’s belief in the dangers of an increase in renin and aldosterone, it is surprising that he supports the use of diuretics for the treatment of hypertension. Diuretics have been shown to be very effective in reducing cardiovascular disease, i.e. strokes, heart failure and coronary artery disease, particularly in the elderly, but they cause increases in plasma renin activity and aldosterone which, according to Alderman, should be harmful. A modest reduction in salt intake lowers blood pressure in an identical way to diuretics and increases both renin and aldosterone to the same levels. Alderman needs to explain why he considers diuretics to be so beneficial, and yet according to him salt restriction is not.

Figure 4 Changes in blood pressure and 24-hour urinary sodium excretion with the reduction in salt intake in all participants (hypertensives: n = 169; normotensives: n = 243) on the normal American diet (i.e. control diet) and on DASH diet. (Redrawn from Ref. 19.)
Alderman's second paper, a 24-h dietary recall on nutrient intake was used to gauge the habitual salt intake of sodium and calories in more than 10,000 subjects between 1971 and 1975. The subjects were then re-examined 20 years later. The calculation of sodium intake, which was based on dietary recall, took no account of discretionary salt intake, i.e. salt added by an individual at the table or in his own cooking which, around 1980, would have accounted for approximately half of salt intake. The measurement of salt intake in this study is therefore inaccurate. A simple inspection of the overall results of the paper also reveals that these are insecure. For instance, Engleman pointed out that in the lowest quartile of daily salt intake in both men and women (approximately 1 and 2 grams respectively) calorie intake was 50% lower than the national recommended daily dietary allowance. Karppanen and Mervaala commented that they thought it remarkable that on such a near starvation diet there were so many survivors. They thought it should have surprised, even Alderman, that women on this extraordinary calorie intake in the second paper invalidate any claim that a daily dietary allowance. Karppanen and Mervaala commented that the weight of the product can be raised by 10 to 20% at no cost to the producer. Total salt intake is an important stimulus to thirst and therefore fluid consumption. Any reduction in the population's salt intake will have a large effect on soft drinks, mineral water and beer consumption.

It is not surprising, therefore, that commercial interests which represent the salt manufacturers and extractors, e.g. the Salt Institute in the US and the soft drinks industry, together with many sections of the food processing industry, have co-operated in perpetuating the idea that salt is not involved in hypertension. They have also suggested that dietary salt only effects a small number of people and that therefore it is not worthwhile for the normotensive population to reduce its salt intake. They also perpetuate the myth that reducing salt intake can be dangerous. Alderman has acted as a member of the Medical Advisory Board for the Salt Institute, which represents the salt manufacturers and extractors not only in the US but worldwide. The Institute kindly distributed his paper in which the claim was made that salt intake was related to myocardial infarction as a ‘professional courtesy’. At the same time the Institute put out a news release which claimed that this study unequivocally ‘showed that hypertensives consuming low

---

**Increased risk of death related to a 6 g/day increase in salt intake (N=2436)**

<table>
<thead>
<tr>
<th></th>
<th>CHD Death</th>
<th>CVD Death</th>
<th>All Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hazard Ratio</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>0.50</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.75</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.95</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.35</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.55</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.75</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Figure 5** The hazards ratios for coronary heart disease (CHD), cardiovascular disease (CVD), and all-cause mortality associated with a 6 g/day increase in salt intake as judged by 24-hour urinary sodium excretion. (Adapted from Ref. 40.)
Sodium diets had dramatically increased rates of having heart attacks. This so-called evidence formed the main plank of the Salt Institute’s aborted petition to the FDA to try to change the regulations concerning commercial claims for low salt foods. Alderman was one of the co-signatories of this petition.

When all of the evidence is considered from epidemiological, migration, intervention, treatment trials, genetic studies in humans and animal studies that relates salt intake to blood pressure and other harmful effects, the evidence is very strong. It is stronger than evidence for other dietary variables that are also important in cardiovascular disease, e.g. saturated fat intake and fruit and vegetable consumption. Nearly all government appointed bodies and nutrition experts who have considered the evidence have recommended a reduction in salt intake from around an average consumption of 10–12 grams to 5–6 grams per day. This recommendation was made in the US in 1970 and in the UK in 1994. Nevertheless salt intake is increasing due to the much greater consumption of processed and ready prepared foods which are very high in salt, often equivalent to that of seawater. In the UK there is a strategy to make small, i.e. 10 to 25%, reductions in the salt concentration of all processed foods. These reductions will not be detected by the consumer and as the salt taste receptors get used to the lower concentration, further reductions can be made in four or five years time. In this way salt intake could be reduced throughout the population without the consumer having to reduce their salt intake consciously. However, the reduction would be greater if consumers themselves added less salt to the food both in cooking and at the table. In this way it should be possible to reduce salt intake in the Western world by half over the next decade. Of all the dietary strategies to reduce the intake of salt, this is probably the easiest to achieve, provided the food industry is prepared to co-operate. In the UK, by manoeuvres which resemble those employed by the tobacco industry, such steps are being vigorously opposed by the Salt Manufacturers’ Association and the Food and Drink Federation, an umbrella organization representing the soft drinks, the snack producers (many of which are owned by the soft drinks producers) and other food processors.

References


Hence if too much salt is used for food, the pulse hardens ...'

Huang Ti Nei Ching Su Wen, 2698–2598 BC (the Yellow Emperor’s classic on internal medicine).

Although ancient Chinese medical literature—the Yellow Emperor’s classic on internal medicine—reported that a high intake of dietary salt (sodium chloride) might produce a hardened pulse nearly 5000 years ago, the first meaningful scientific evidence for a positive association between salt consumption and level of blood pressure was published by Dahl in 1960.1 Dahl described a remarkable linear relationship between average sodium intake and prevalence of hypertension across five population groups.1 Since then, abundant evidence of a causal association between dietary sodium intake and high blood pressure has emerged from animal experiments and from observational epidemiological studies and randomized controlled clinical trials.2,3

Animal Experiments

Studies in a variety of laboratory animals have demonstrated that a high dietary intake of salt results in hypertension.4 Recently, Denton et al. reported the findings of a 3-year experiment conducted in 26 chimpanzees with a baseline diet which was very low in sodium and high in potassium content.5 Following a year of baseline observations, salt was added to the diet of 13 animals in increasing amounts (5 g/day for 19 weeks, 10 g/day for 3 weeks, and then 15 g/day for 67 weeks) during an 89-week period of active intervention. Dietary intake of salt remained unaltered in the other 13 control animals. The average level of blood pressure did not change significantly during the intervention phase in the control group. However, in the 13 animals assigned to the active intervention mean systolic blood pressure (SBP) increased by 12 mmHg compared to the corresponding baseline level ($P_{0.05}$) after the first 19 weeks of supplementary salt intake (5 g/day). Following the 39 weeks of supplementation with 10 g/day salt (3 weeks) and 15 g/day salt (36 weeks), mean SBP was increased by 26 mmHg ($P_{0.001}$) and mean diastolic blood pressure (DBP) was increased by 5 mmHg ($P_{0.05}$). Following a further 26 weeks of supplementation with 15 g of salt/day (a total of 84 weeks of supplementation with dietary salt), mean SBP was increased by 33 mmHg ($P_{0.001}$) and DBP was increased by 10 mmHg ($P_{0.01}$). Twenty weeks after the end of the period of salt addition, the animals’ average level of blood pressure returned its baseline level. This experiment, conducted in the species that is phylogenetically closest to humans, provides direct evidence in favour of a causal relationship between high salt intake and hypertension.5

Observational Epidemiological Studies

Studies in low blood pressure populations and in migrants from these societies to more westernized environments provide strong evidence for a causal relationship between high salt intake and hypertension. More than 30 populations with an average blood...