United Kingdom, and elsewhere) are based solely on Intersalt findings. The findings of Intersalt agree with a whole body of concordant evidence.

Availability of data

Our final point concerns availability of data from Intersalt. We are aware of few studies that have published so much detailed, peer reviewed data as has Intersalt. The original article in the *BMJ* in 1988 included three tables in an appendix giving detailed information on 27 variables (descriptive and analytical).[1] One year later, in a special issue of the *Journal of Human Hypertension,* detailed age and sex specific Intersalt data were given in 38 appendix tables, with 20 columns of data each.[6]

This widespread dissemination of data was based on an early decision by the steering and editorial committee to share data with fellow scientists and public health agencies. As is customary in scientific investigation, raw data on individuals remain the confidential property of local investigators, in this case the 52 investigators in 32 countries. The most recent Intersalt paper[2] further described reasons for our declining the request to supply the study's raw data to the Salt Institute, the trade organisation of salt producers.

The first request from the Salt Institute came through its attorneys, six years after the original data were published. The Intersalt group's reply stated our willingness to perform additional reasonable scientific analyses. These requests were received, the analyses were performed, and the data were sent to the Salt Institute. They have not been cited anywhere by the institute.

Conclusions

Repeated recommendations to the population for salt reduction,[7-15 18] such as those made by the US Departments of Agriculture and of Health and Human Services in 1995[12,18] and by the report of the Cardiovascular Review Group of Britain's Committee on Medical Aspects of Food Policy in 1994,[11] rest on a strong research base. They need to be implemented. Since 75% of salt consumed in the United States, the United Kingdom, and other countries comes from salt added in food production,[2 8] the food industry—by reducing salt added in food processing—has an important role in implementing these public health recommendations.

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An extended version of this paper is on the BMJ world wide web page (http://www.bmj.com/bmj/).

Salt—overwhelming evidence but still no action: can a consensus be reached with the food industry?

Graham A MacGregor, Peter S Sever, conveners of CASH (Consensus Action on Salt and Hypertension)

This paper comes from two members of a group called Consensus Action on Salt and Hypertension (CASH). The group was set up after the disbanding of Britain's Committee on Medical Aspects of Food. It believes that the British government has dissociated itself from taking action on salt despite strong evidence on its harmful effects. The aims of the group are to work with the food industry to reach a consensus on salt and blood pressure and then to devise means to reduce the amount of salt in food

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Evidence that our current high salt intake plays an important role in blood pressure regulation comes from many sources, chiefly epidemiological studies, intervention studies, migration studies, studies of salt restriction, and evidence in mammals.

Epidemiological studies illustrate that salt intake may be a major factor in determining differences in blood pressure between and within communities as well as being closely associated with the rise in blood pressure with age in communities with a high salt intake. But these studies can only suggest relationships. Attempts to derive from epidemiological evidence simple relationships between a rather crude index of sodium intake and blood pressure will inevitably be confounded by the variable expression of other environmental and genetic factors, which may modulate the blood pressure response to sodium.[1] Further analyses of results from large epidemiological studies such as Intersalt are unlikely to shed further light on this complex issue. It is therefore necessary to look at different types of evidence that more clearly show the effect of salt on blood pressure.

Stronger evidence

Stronger evidence comes from intervention studies such as a carefully controlled six month double blind study in newborn babies.[2] Those receiving a modestly restricted salt intake had a significantly lower blood pressure (2·1 mm Hg) than those with a normal sodium intake. As the sodium restriction was discontinued at six months it is not known whether this difference would have increased, but a reduction of even 2·0 mm Hg in population blood pressure would have immense benefits in reducing cardiovascular morbidity and mortality. In another study, a village in Portugal

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reduced salt intake by reducing salt in cooking and also in processed food, including bread. At the end of the observation period blood pressure was significantly lower than in a control village.[3]

More evidence comes from migration studies. Subsistence farmers in rural Kenya who consumed a low salt diet were followed when they migrated to a urban community, where salt intake increased to levels seen in Western countries. Blood pressure was higher in migrants than in a control group who did not migrate.[4] Short term studies of salt restriction also provide data. Many well controlled studies show that in patients with high blood pressure very modest salt restriction causes a fall in blood pressure[5] similar in magnitude to that seen with a diuretic.[6] In normotensive subjects this fall in blood pressure is less great. This finding is often cited as evidence that salt is not important in blood pressure regulation, or important only in the 10% or so of people who are hypertensive. However, those who use this evidence in this way misunderstand the difference between decades or a lifetime of high salt intake and a few weeks of modest salt restriction, and they conveniently forget that in Western populations at least 30% of people over the age of 60 have raised blood pressure, which responds particularly well to salt restriction.

There is an immense amount of evidence in other mammals than man that salt plays a critical role in regulating blood pressure. In virtually all mammalian models, high blood pressure is caused or aggravated by a high salt intake.[7] For those who require further evidence, two recent studies provide it. Firstly, in Nigeria in two rural villages inhabited by the same tribe of subsistence farmers, unexposed to Westernised foods or lifestyle and consuming a similar diet—except that one village had access to salt from a nearby salt lake—blood pressure and sodium intake were higher in the village with access to salt.[8] The differences in blood pressure were difficult to explain other than by the salt intake. Secondly, increasing salt intake from the amount in chimpanzees' natural diet to that which chimpanzees and humans are given in Western societies caused an increase of about 30 mm Hg in systolic blood pressure over a 12 month period, in the absence of any other environmental change.[9] When the salt intake was reduced in the chimpanzees' diet, blood pressure fell to previous levels. It is difficult to produce reasons why humans should differ from their closest animal relative.

It is ironic that some years ago a double blind study of short term salt supplementation was proposed in rural Kenyans to increase salt intake to Western levels. The study was refused permission on the grounds that it would be unethical to expose these people to additional salt.

Public relations

In spite of all this evidence there has been little concerted attempt to reduce sodium intake in Western countries. Indeed the salt and processed food industry has fought a careful, expensive, and largely successful public relations campaign over the past decade in the United States, United Kingdom, and elsewhere to convince the rest of the food industry, food suppliers, politicians, nutritionists, and doctors that the evidence for salt is not substantial or at least that it is not sufficient for any action and that more studies are needed.

In the United Kingdom this campaign involved the Salt Manufacturers' Association, which includes Rank Hovis McDougall, maker of many of the table salts marketed in the United Kingdom, which in 1985 set up with Kingsway Publications a public relations company, the so called Salt Data Centre. Shortly afterwards a press conference convened for the Salt Data Centre claimed that salt intake bore little or no relation to blood pressure, and this claim received widespread publicity. The Rowland Company, which superseded Kingsway Publications, has continued this campaign —producing, for instance, a booklet in the style of the Health Education Council extolling the benefits of salt and making the remarkable assertion that if a person has a family history of high blood pressure, restricting salt intake does not prevent high blood pressure in later life. There was no indication in the booklet that it had been written and paid for by the salt industry.[10]

In spite of this media campaign the British government recently sought the opinion of those who had worked on salt and blood pressure. All views were canvassed and it was concluded that salt intake should be reduced from the current daily average of 9 grams (150 mmol) to 6 grams (100 mmol).[11] The report said, "A reduction (or even an elimination) in the amount of salt added to foods by individuals in cooking or at table would not be sufficient to reach this target and there needs to be a gradual reduction in the amount of sodium, from salt, added to processed foods. We recommend that food manufacturers, caterers and individuals explore and grasp the opportunities for reducing the sodium content of foods and meals."

These recommendations, along with others on diet and cardiovascular disease, were published in full by the government, but at a subsequent press conference the government disowned the specific recommendations about salt, with the cryptic comment that there was continuing controversy about salt and blood pressure. It subsequently emerged that the food industry had exerted immense pressure on the government concerning the specific recommendations on salt.

Salt and taste

This raises the question of why is the food industry so defensive of the high salt content of processed food. Much of the food industry relies on high volume and a low profit margin, and small changes in sales are critical. Any change in the composition of processed food is therefore a risk, and rivals will try to exploit such an opportunity. According to the Salt Data Centre, a major justification for the use of salt in processed food is that "salt makes unpalatable food edible." The sensitivity of the salt taste receptors, however, depends on the individual's habitual salt intake. Once salt intake has been reduced for a month or more, highly salted food becomes distasteful. Many salt addicts may not realise that many of the processed foods that they currently consume contain a salt concentration approaching or equal to that of sea water.

It would be relatively easy to gradually reduce the salt content of processed food, particularly bread, which is the single largest source of salt in the British diet. Some years ago the Health Education Council persuaded the main bread manufacturers to make a small reduction in the salt content of bread, and there were no subsequent problems. Also, Heinz has successfully made small reductions in the salt content of some of its foods.

Whose responsibility?

The British government, at least in relation to salt, seems to have dissociated itself from its social responsibility for the welfare of its citizens. Where does the responsibility now lie? Many feel that the food suppliers and food industry also have a responsibility to those who purchase their food. However, the industry seems to be reluctant to act, indicating that it is up to consumers to lead the demand for change —a somewhat unrealistic prospect, particularly as most consumers are unaware of the large amount of

salt added to processed food such as bread. Therefore we feel that the best way of tackling these issues, given the lack of government interest, is to enter into direct dialogue with the food industry and food suppliers. With this in mind we have set up a group for Consensus Action on Salt and Hypertension (CASH) with the aim of involving those who have researched salt and blood pressure, the food suppliers, and the food processors.

The first objective of this action group is to reach a consensus about salt and blood pressure. Many in the food industry have been misled by their own public relations campaigns or propaganda and are impressed when all the evidence is discussed. The second objective of the group is to agree about how we can gradually reduce the salt content of processed food.

The suggested reduction in salt intake from an average of 150 mmol to 100 mmol a day is likely to be of immense benefit. This benefit can be predicted from results of epidemiological, intervention, migration, and treatment studies to reduce strokes by 22% and heart attacks by 16% in Britain[12]—which, at a population level, would confer a greater benefit than all current treatment for high blood pressure. Furthermore, there is increasing and disturbing evidence that a high salt intake may have other serious adverse effects on human health, including a direct effect on strokes and left ventricular hypertrophy independent of and additional to salt's effect on blood pressure.[13] A high salt intake also seems to be an important aggravating factor in bone demineralisation (osteoporosis),[13] so the eventual benefits of this modest reduction in salt intake may be even larger than those predicted.

Members of the consensus group are P S Sever, London; N Wald, London; D G Beevers, Birmingham; H de Wardener, London; P Sleight, Oxford; M Marmot, London; P Elliott, London; C Warlow, Edinburgh; W P T James, Aberdeen; N Poulter, London; P Dodson, Birmingham; A G Shaper, London; M Law, London.

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Lesson of the Week

Hyperkalaemic cardiac arrest successfully treated with peritoneal dialysis

M A Jackson, R Lodwick, S G Hutchinson

Peritoneal dialysis may be lifesaving in hyperkalaemic cardiac arrest

Cardiac arrest caused by asystole, as a result of hyperkalaemia, is usually fatal. Resuscitation is often curtailed early because a successful outcome is unlikely. If the hyperkalaemia cannot be corrected electrical transmission is blocked to distal areas of the heart and asystole will persist.[1,2]

We report on a patient subsequently shown to have muscular dystrophy who was successfully treated with peritoneal dialysis after conventional pharmacological methods had failed.

Case report

A 16 year old Asian man was admitted for a routine nasal septoplasty. He admitted that for several years he had had myalgia after exercise. He was not taking any medication and he had no history of allergies. There was no family history of any adverse reaction to anaesthesia. Examination at the time of admission showed nothing abnormal.

Temazepam and metoclopramide were given as premedication. Propofol mixed with lignocaine and suxamethonium were used to facilitate intubation. Anaesthesia was maintained using isoflurane and a mixture of nitrous oxide and air. An electrocardiogram, oxygen saturation, and end tidal carbon dioxide were normal throughout the operation, which proceeded uneventfully and lasted about 30 minutes.

On arrival in the recovery area his breathing became increasingly laboured, culminating in respiratory arrest. Cardiopulmonary resuscitation was started promptly and the patient was reintubated. An electrocardiogram showed that his heart was in asystole.

Atropine (3 mg) and 1 mg of adrenaline were given, which resulted in broad complex tachycardia. The patient's heart reverted rapidly back into asystole and again atropine and adrenaline were given. This produced broad complex tachycardia with a good cardiac output. Lignocaine (100 mg) was given. Polymorphic broad complex tachycardia persisted.

Episodes of broad complex tachycardia and periods of asystole occurred after the administration of 50 mg of disopyramide. During the periods of asystole additional infusions of 1 mg of adrenaline and 10 ml of 10% calcium gluconate were given. The patient again developed a broad complex tachycardia. Amiodarone (150 mg) was given through an internal jugular venous line followed by 50 ml of 8·4% bicarbonate. After 30 minutes the patient's heart went into asystole.

When the patient's serum potassium concentrations were available 100 mg of 50% dextrose was given with 20 units of soluble insulin. Initial transthoracic and then intraventricular cardiac pacing using the internal jugular route was unsuccessful despite a good wire position on screening.

Serum electrolytes measured 30 minutes after the start of the cardiac arrest were within the normal range except for a potassium concentration of 9·8 mmol/l in an unhaemolysed sample. Arterial gases indicated a profound metabolic acidosis (table 1). Despite calcium gluconate, sodium bicarbonate, isoprenaline, glucose, and insulin infusion, there was no sustainable output. A repeat serum potassium sample showed a value of 9·4 mmol/l.

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