Sodium as salt

Introduction

Salt is nutritionally equivalent to sodium chloride (NaCl) and is used as a food ingredient or condiment. Sodium is also found in unprocessed foods but usually in very low concentrations. 1 g salt corresponds to about 0.4 g sodium and 1 g sodium is equivalent to 2.5 g salt. 1 mmol sodium corresponds to 23 mg and is equivalent to about 58 mg sodium chloride.

Dietary sources and intake

The main sources of sodium in the diet are processed foods e.g. bread, cheese, spreads, meat and fish products (88). The contribution of sodium from added salt in cooking and at the table varies but in average it constitutes approximately 10 to 20 % of the total salt intake (88, 89). Data on the total dietary intake of sodium in Nordic populations are scarce. According to national food balance sheets the availability of salt in the Nordic countries is estimated to be 10-12 g per capita and day. Estimations of the sodium intake from national dietary surveys among adults generally show somewhat lower values. Average dietary sodium contents calculated from national dietary surveys among adults were in: Denmark 3.9 g/day (9.8 g salt) in men and 2.9 g/day in women (7.3 g salt), Finland 3.7 g in men (9.3 g salt) and 2.7 g in women (6.8 g salt), Iceland 3.7 g (9.3 g salt) and in Sweden 3.4 g (8.5 g salt) (88-93). The contribution from discretionary salt intake, e.g. from extra salt added to meals etc., is generally not included. Data from Finnish population studies suggest that sodium intake assessed from dietary records, 24-hour and 48-hour dietary recalls are valid and give similar estimates of the mean level of sodium intake as determinations of 24-hour urinary sodium (88)

Physiology and metabolism

The sodium ion is essential for a number of metabolic processes in the cell and is involved in the regulation of the acid-base balance, the osmotic pressure in ECV, blood volume, nerve function and the transport mechanisms for glucose and certain amino acids (1).

The body pool of sodium in an adult is approximately 100 g. About half is found in the extracellular fluid (ECV) and 10 % in the cells. The rest is mainly bound in the skeleton, of which half is exchangeable and thereby functions as a store for the body fluids.

The absorption of sodium is effective and generally amounts to more than 90 % of the dietary intake. The excretion of sodium mainly occurs through the kidneys, where it is effectively regulated depending on sodium and fluid intake. Losses through the skin in our climate are generally not more than 1 mmol/d (2). Small amounts of sodium (0.1-8 mmol) are also lost daily in the faeces (3). During profound sweating, in massive diarrhoea or vomiting, the extrarenal loss may be clinically significant. Healthy kidneys can retain almost all sodium in the body, since the tubuli cells reabsorb sodium up to 99.5 %. Healthy kidneys can also excrete large amounts of sodium. This requires a satisfactory water supply, since the urine cannot be concentrated more than to a limited degree. The daily excretion through kidneys and skin is normally 100-200 mmol.

Requirement

Dietary sodium deficiency does normally not occur in the Nordic countries. Acute deficiency can develop in connection with heavy sweating in combination with large fluid intakes devoid of sodium, or in connection with prolonged vomiting and diarrhoea without salt supply. Clinical symptoms include muscle seizures, loss of appetite and circulation disturbances. Severe deficiency can result in coma and death.

Among adults, sodium balance can be maintained at intakes as low as 10 mmol (230 mg) per day corresponding to about 0.6 g of salt. An intake of 25 mmol (575 mg) per day, corresponding to about 1.5 g salt, is set as the estimated lower limit of intake, accounting for variation in physical activity and climate (1).

Salt and blood pressure

From a public health perspective the role of sodium as dietary salt in the regulation of blood pressure has received most interest. The relationship between salt and blood pressure has been studied for a long time. Kempner made classical observations during the 1930s and 1940s (4). He treated e.g. diabetics and hypertensive subjects with a salt-restricted rice and fruit diet containing less than 2 grams of salt per day and found that blood pressure was drastically reduced among most of the patients.

Cross-sectional population studies

Population studies have shown that hypertension is rare in populations with a very low salt intake (< 2 g/d) and that blood pressure does not rise with age (5, 6). In areas with very high salt intakes (30-35 g/d), severe hypertension has been reported among 30-35% of the population (5). In the large, multi-centre Intersalt study (6) the relationship between 24-h sodium and potassium excretion and blood pressure was investigated. The study included 10 000 men and women aged 20-59 years from 52 centres around the world. The median

sodium excretion varied from 0.2 mmol/d to 242 mmol/d between centres. In four centres with very low sodium excretion, blood pressure was low and no age-related increase was observed. In the other 48 centres, sodium excretion was related to the increase in blood pressure with age but not to median blood pressure or prevalence of high blood pressure. Potassium excretion was negatively related to blood pressure on an individual basis, while the sodium: potassium ratio showed a pattern similar to that of sodium. Body mass index and heavy alcohol intake were strongly related to blood pressure.

Law et al. (7) analysed published data on blood pressure and sodium intake for 24 different communities (47,000 subjects) throughout the world, including the Intersalt study. Allowance was made for differences in blood pressure between economically developed and underdeveloped communities to minimise overestimation of the association through confounding with other determinants of blood pressure. The authors found that blood pressure was higher on average in the developed communities, but the association with sodium intake was similar in both types of community. A difference in sodium intake of 100 mmol/24 h was associated with an average difference in systolic blood pressure that ranged from 5 mm Hg at age 15-19 years to 10 mm Hg at age 60-69. The differences in diastolic blood pressure were about half as great. The authors concluded that the association of blood pressure with sodium intake is substantially larger than is generally appreciated and increases with age and initial blood pressure. Data from within population studies also generally support an association (8, 9).

In the EPIC-Norfolk (the European Prospective Investigation into Cancer in Norfolk) study with 23,104 community-living adults aged 45 to 79 years, mean systolic and diastolic blood pressure increased as the ratio of urinary sodium to creatinine increased, with differences of 7.2 /3.0 mmHg for systolic/diastolic blood pressure between the top and bottom quintiles (10). This trend was independent of age, body mass index, smoking and ratio of urinary potassium to creatinine, and was consistent by sex and history of hypertension.

Clinical trials

Several meta-analyses of clinical trials of dietary salt reduction have been published (10-14). These differ in scope and inclusion criteria. Law et al. (11) analysed 68 cross-over and 10 randomised controlled trials of salt reduction among normotensives and hypertensives, which included studies published up to 1989. They found that the blood pressure lowering effect of salt restriction was related to the duration of the study, with less effect in trials lasting less than 4 weeks. They concluded that in people aged 50-59 years, a reduction in daily sodium intake of 50 mmol (approximately 3 g of salt) would, after a few weeks, lower systolic blood pressure by an average of 5 mm Hg, and by 7 mm Hg in those with high blood pressure (170 mm Hg). The diastolic blood pressure would be lowered by about half as much.

Midgley et al. (12) analysed 56 trials, published between 1966 and 1994, that had randomised allocation of subjects to control and dietary sodium intervention groups, monitored by sodium excretion, with outcome measures of both systolic and diastolic blood pressure, selected by blinded review of the methods section. Several of these studies, including some published before 1990, were not included in the analysis by Law et al. (11). The mean reduction in daily urinary sodium excretion was 95 mmol/d (71-119 mmol/d) in 28 trials with 1,131 hypertensive subjects and 125 mmol/d (95-156 mmol/d) in 28 trials with

2,374 normotensive subjects. In hypertensive subjects, a reduced urinary sodium excretion of 95 mmol/d reduced systolic blood pressure by 5.9 mm Hg (95 % CI 4.1 to 7.8 mm Hg) and diastolic blood pressure by 3.8 mm Hg (95 % CI 2.9 to 4.8 mm Hg). In normotensive subjects, the corresponding changes for a reduced urinary sodium excretion of 125 mmol/d were a reduction of 1.6 mm Hg (95 % CI 0.9 to 2.4 mm Hg) for systolic and of 0.5 mm Hg (non-significant; 95 % CI -0.1 to 1.2 mm Hg) for diastolic blood pressure. A weakness of the analysis of trials on normotensives was the short duration of the trials (on average 14 days), although the authors state that there was a tendency for a greater blood pressure reduction in trials with a shorter duration (< 2 weeks). This is in contrast with the findings of Law et al (10) and could be due to problems of compliance in some of the more long term-studies. Trials on normotensive subjects involved mainly young subjects, while the trials on hypertensives mainly involved middle-aged or older subjects. The decreases in blood pressure were larger in trials on older hypertensive individuals than on younger, whereas no data are given for the normotensives.

Graudal et al. (13) published another meta-analysis including 58 randomised trials on dietary sodium restriction among hypertensives and 56 trials among normotensives published between 1966 and 1997. In 58 trials of hypertensive persons (exact criteria not stated), a reduced urinary mean sodium excretion of 118 mmol/24 h gave a significant reduction in systolic blood pressure of 3.9 mm Hg and diastolic blood pressure of 1.2 mm Hg. In 56 trials of normotensive persons, a reduced mean sodium excretion of 160 mmol/24 h was associated with a significant average reduction in the systolic blood pressure of 1.2 mm Hg, while a non-significant reduction in the diastolic blood pressure of 0.26 mm Hg was observed. In this study too, trials on normotensives had a short duration, mean of only 8 days, and included younger subjects (mean age 27 years) with a mean systolic blood pressure of 120 mm Hg. This limits the relevance of the results for public health action. The mean duration of trials of hypertensives was 28 days and the mean age of the subjects was 49 years, which is comparable to the analysis by Midgley et al (12).

The meta-analysis by Cutler et al. (14) included 23 trials published up to mid-1994. The lower number of trials included was due to stricter inclusion criteria. The combined weighted data showed that a decrease in sodium excretion of 100 mmol Na/24 h (5.9 g salt) was associated with a reduction in systolic blood pressure of 4.8 mm Hg in hypertensives and 2.3 mm Hg in normotensives. The corresponding figures for diastolic blood pressure were 2.5 and 1.4 mm Hg, respectively.

In the meta-analysis by Geleijnse et al. (15), only randomised controlled trials with duration greater than 2 weeks were included. Forty trials published between 1966 and 1991 were included. A median reduction in sodium excretion of 77 mmol/24 h (4.5 g salt) was associated with a 2.5 mm Hg reduction in systolic blood pressure and 2.0 mm Hg in diastolic blood pressure. Reductions were more pronounced in hypertensives and the same tendency was seen in older subjects. A subsequent meta-analysis including trials with a duration of 4 weeks or more with a similar reduction in sodium excretion (74-78 mmol/24h) found a 5.0 mm Hg reduction in systolic blood pressure and 2.7 mm Hg in diastolic blood pressure among hypertensives. Corresponding figures for subjects with normal blood pressure were 2.0 mm Hg and 1.0 mm Hg, respectively (16). A dose-response relationship was observed with a SBP/DBP reduction of 7.2/3.8 mmHg among hypertensive and of 3.6/1.7 mmHg among normotensive individuals per 100 mmol/24h (6g salt) reduction in sodium excretion.

Only a few studies have examined the long-term effects on blood pressure of sodium restriction. Jula et al. (17) studied the effects on blood pressure and serum lipids of a non-pharmacological treatment based mainly on sodium restriction in a 12-month controlled randomized study with 91 middle-aged untreated mildly hypertensive men and women. The estimated daily sodium intakes, calculated from 24-h urines, decreased in men from 227 mmol to a mean level of 105 mmol, and in women from 129 mmol to 63 mmol. After 12 months of non-pharmacological treatment, the mean weight in men was 1.9 kg lower and in women 0.3 kg higher compared to the baseline. In the treatment group, energy derived from fats decreased in men by 4 % and in women by 3 % reflecting decreased intake of saturated and monounsaturated fats. The net blood pressure decrease (difference in changes between treatment and control group) during the 12 months in men was 8.2 mm Hg for systolic and 5.8 mm Hg for diastolic blood pressure, and in women 9.5 mm Hg for systolic and 5.6 mm Hg for diastolic blood pressure. All changes were significant. In the treatment group LDL-cholesterol also decreased, by 6.8 % in men and by 12.1 % in women.

In the DASH trials (Dietary Intervention to Stop Hypertension) the effects of various controlled diets on the blood pressure of adult Americans with normal or moderately elevated blood pressure were studied (18,19). In the study by Sacks et al. (19) the influence of sodium intake on blood pressure was assessed in 412 subjects who were randomly assigned to eat either a control diet typical of intake in the United States or the DASH diet. In both groups, a second randomization was done, and the subjects ate their assigned diets at three sodium levels for 30 days in random order in a crossover design The subjects were selected among adults 22 years or older, who were not taking antihypertensive medication, and with a systolic blood pressure exceeding 120 but below 160 mm Hg and a diastolic ranging from 80 to 95 mm Hg. The control diet had a fat composition corresponding to the usual American diet (36 E% total fat, 14 E% saturated fat), but a low content of fruit vegetables and milk products. The DASH diet was rich in fruit, vegetables and low-fat dairy products, but low in edible fats, snacks and sweets, with a low content of total fat (25 E%) and saturated fat (7 E%) and cholesterol. The content of calcium, potassium and magnesium in the control diet was lower than in the average American diet, whereas the level in the DASH-diet was higher. The intake of dietary fibre was similar in both groups. Within the assigned diets, sodium levels were adjusted to provide a daily intake of 150 mmol (high, about 9 g salt), 100 mmol (intermediate, about 6 g salt), and 50 mmol (low, about 3 g salt) for 30 consecutive days each, in random order. The estimated sodium intakes, calculated from 24-h urines, indicated a lower intake during the high (141-144 mmol/l, about 8 g salt) and higher intake during the low (64-67 mmol, about 4 g salt) and intermediate (106-107 mmol, about 6 g salt) sodium phases.

Reducing the sodium intake from the high to the intermediate level significantly reduced the systolic blood pressure by 2.1 mm Hg during the control diet and by 1.3 mm Hg during the DASH diet. A further reduction from the intermediate to the low level caused additional reductions of 4.6 mm Hg on the control diet and 1.7 mm Hg on the DASH diet. A regression analysis of these data shows that a reduction in the sodium intake of 100 mmol per day would lead to a reduction in the systolic blood pressure of about 3 mm Hg in the DASH group and of about 7 mm Hg in the control group. Corresponding values for diastolic blood pressure are 1.5-2 and about 3 mm Hg, respectively. The effects of sodium were observed in normotensive and hypertensive subjects, whites, blacks and other races, women and men, and were not dependent on weight (19, 20).

An aspect that was only partly addressed the meta-analyses is the relationship between the sodium intake and the age-related change in blood pressure. Data from the Intersalt study strongly indicate a relationship between the median daily urinary sodium excretion and the difference in blood pressure with age (21). In within population analyses, individual 24 h urinary sodium excretion higher by 100 mmol was associated with a 3-6 mm Hg higher systolic and 0-3 mm Hg diastolic blood pressure. Associations were larger at ages 40-59 than at younger ages. In cross-population analyses, median 24-hour sodium excretion higher by 100 mmol was associated with 5-7 mm Hg higher median systolic and 2-4 mm Hg higher median diastolic pressure. At age 55 the estimated mean difference in systolic and diastolic blood pressure was 10-11 and 6 mm Hg greater, respectively, compared to at age 25, indicating a strong age-related effect of high sodium intakes on blood pressure. In the DASH-trial, the blood pressure reduction was higher in older (> 45 yr) than in younger subjects, e.g. a 100 mmol reduction in sodium excretion was associated with a 6 mm Hg lower systolic blood pressure among non-black older subjects (20).

The DASH-trials clearly showed an effect of sodium restriction, ranging from 2-5 g/d, on blood pressure, which is independent of other dietary and lifestyle factors. An important finding is that the blood pressure reduction was larger in the control group than in the DASH group. This implies that the benefits of sodium restriction are more pronounced among persons consuming a diet which is less optimal, e.g. with respect to fat, fruit and vegetables etc. (and similar to the current dietary patterns in the Nordic countries), than among those already consuming a diet in line with the general nutrition recommendations. A limitation of the study is the relatively short duration (30 days) and the fact that the study excluded subjects with low (SBP < 120 mm Hg) and high (SBP > 160 mm Hg) blood pressure. However, the blood pressure lowering effect of dietary salt reduction on hypertensives is well documented, while the proportion of the adult population with systolic blood pressure below 120 mm Hg is small, especially among the middle-aged and older.

Observational population studies and population-based intervention studies

In Japan, the population salt intake was very high (average 13.5-18 g/d) in the late 1950´s. A national campaign over the following decade resulted in reduced salt intake (to an average 12.1-14 g/day) which was associated with a population BP decrease and a large reduction in stroke mortality (Sasaki 1979). In Finland, salt intake decreased by 40 % from 1970s to 2002 together with decreases in the intake of saturated fats and increases in the intakes of fruits and vegetables (22, 23). The dietary changes were associated with a 10-20/6-10 mmHg decrease in population systolic/diastolic BP and with a 70% decrease in stroke and CHD mortality (24,25,26) In a Portuguese population-based intervention study, sodium intake was reduced by dietary advice (27). The mean dietary intake of salt decreased by approximately 40 % (from approximately 20 to 11.5 g/d), estimated by food consumption data, while estimations based on urinary sodium to creatinine ratios indicated a lower reduction, approximately 25 % (5 g salt) after one year and 9 % (2 g salt) after 2 years. After 2 years of intervention, the systolic and diastolic blood pressure had both decreased by approximately 5 mm Hg. The systolic blood pressure rose in the control community, and after 2 years there was a 13/6 mmHg difference in SBP/DBP between the intervention and control communities.

Salt intake and blood pressure among children

The blood pressure of children living in industrialized countries rises with age, more rapidly in children of hypertensive parents than in children of normotensive parents (28, 29, 30, 31). In the STRIP study, systolic blood pressure of children living in South-Western Finland increased with age along with sodium intake and exceeded the adult systolic blood pressure level of low-sodium cultures already at the age of 10 years. The mean daily sodium intake was 1500 mg at the age of 13 months and 3000 mg at the age of 15 years. Similar levels of salt intake of children have been reported from other countries (32, 33). Childhood blood pressure tracks with adult blood pressure (34, 35) and predicts early atherosclerosis in adolescence (36) and adulthood (36, 37).

In a randomised trial among 476 Dutch newborn infants, the effect of a low (on average 120 mg/d) or normal (on average 330 mg/d) sodium diet on blood pressure during the first 6 months of life was studied (38). The sodium intake in the low sodium group was approximately similar to the intake of breast-fed infants, whereas the intake in the normal group was similar to the sodium intake of infants fed commercial infant formula. At the end of the trial, systolic blood pressure in the low sodium group was 2.1 mm Hg lower than in the control group. The authors also measured blood pressure in 167 children from the original cohort (35 %) after 15 years of follow-up. The adjusted systolic blood pressure at follow-up was 3.6 mm Hg lower and the diastolic pressure was 2.2 mm Hg lower in adolescents who as infants had been assigned to the low sodium group compared with those assigned to the control group.

One meta-analysis of controlled trials has been carried out to assess the effect of reducing salt intake on blood pressure in children and adolescents (39). Ten trials with 966 participants were included. Among adolescents (mean ages for individual trials from 8 to 16 years) salt intake reduced by 42 %. Systolic blood pressure reduced by 1.2 mm Hg (95 % CI 0.6 to 1.8 mm Hg) and diastolic blood pressure by 1.3 mm Hg (95 % CI 0.7 to 1.9 mm Hg) after a median duration of 4 weeks. In the three trials with infants, sodium excretion reduced by 54 %. Systolic blood pressure decreased by 2.5 mm Hg (95 % CI 0.9 to 4.0 mm Hg) after a median duration of 20 weeks.

Findings from epidemiological studies suggest that an early prevention of elevated blood pressure is important (6, 7, 21). Data from clinical trials ascertain that reduction in sodium intake in early life can slow or prevent blood pressure rise with age (38, 39).

Other dietary factors and blood pressure

A number of dietary factors and physical activity have been associated with blood pressure. These include e.g. alcohol, potassium, calcium, magnesium, and fatty acid composition (see respective chapter).

Salt and morbidity and mortality

There are only few studies that have investigated the relationship between sodium intake and morbidity and mortality. The multi-centre CARDIAC study (WHO Cardiovascular Diseases and Alimentary Comparison Study) (57) investigated the relationship between biological markers of dietary factors with blood pressure and age-adjusted mortality rates of stroke and ischaemic heart disease from 55 centres in 24 countries. From each population, 100 men and

100 women aged 48 to 56 years were randomly selected for BP measurement, 24-hour urine collection and other biological parameters. Cross-centre analyses showed that stroke mortality was significantly positively related to the 24-hour sodium excretion rate in men and to the sodium/potassium ratio in both sexes.

Alderman et al. (58) reported an increased risk of myocardial infarction in association with a lower sodium intake among male hypertensive subjects who had been treated with blood pressure reducing drugs. The trend for women was the opposite, although not significant. The sodium intake was measured using single 24-h urine, which was collected 5 days after the subjects had been asked to avoid consumption of foods with a high salt content. One can therefore question whether the assessment provided a representative measure of the subjects' usual sodium intake. The results could also have been biased due to that confounders, e.g. alcohol, were not accounted for. In another study Alderman et al. (59) reported a significant negative correlation between sodium intake estimated by 24-h recalls and all-cause and CVD mortality in a follow-up of the first U.S. NHANES I study. Based on these results, the authors concluded that sodium restriction might lead to negative health effects and that advice to reduce sodium intake in the general population is not justified. A critical examination of the data (60), however, favoured the opposite interpretation since the authors also found a positive correlation between the sodium content of the diet expressed as mg/kcal and mortality. A major weakness of the NHANES I data was the low energy intake, which was on average below levels associated with bed-bound or wheelchair activity. When the study population is classified into sodium density (mg Na/kcal), the energy intakes are more comparable among the quartiles, indicating that underreporting is more evenly distributed. The energy adjusted sodium intakes are thus more reliable, and only these data can, in the absence of 24-h urine data, be used with some confidence in the analysis of a possible relationship between sodium intake and mortality. The result of this analysis, which the authors briefly mention, is that there is a weak, but significant, positive association between the sodium content of the diet and both total and CVD mortality.

He et al (61) examined the risk of cardiovascular disease associated with dietary sodium intake in 2,688 overweight (BMI) and 6797 non-overweight persons in the first National Health and Nutrition Examination Survey Epidemiological Follow-up Study (NHANES I). Subjects were aged 25 to 74 years when the survey was conducted in 1971–1975. Dietary sodium and energy intakes were estimated at baseline using a single 24-hour dietary recall method. The average follow-up was 19 years. Among overweight persons, a 100 mmol higher sodium intake was associated with a 32 % increase in stroke incidence, 89 % increase in stroke mortality, 44 % increase in coronary heart disease mortality, 61 % increase in cardiovascular disease mortality, and 39 % increase in mortality from all causes. Dietary sodium intake was not significantly associated with cardiovascular disease risk in non-overweight persons. The limitations of the study are the same as for the earlier mentioned study by Alderman et al. (60) on the same population.

In a prospective study by Tuomilehto et al. (62) on Finnish men and women aged 25-64 years, 24-h urinary sodium excretion, divided into quartiles, was directly related to the incidence of coronary and stroke events, and death from coronary heart disease, cardiovascular disease, and any cause. There was a significant elevated risk for coronary heart disease, cardiovascular disease, and all-cause mortality, associated with a 100 mmol increase in 24 h urinary sodium excretion in both men and women. The frequency of acute coronary events, but not acute stroke events, rose significantly with increasing sodium

excretion. In separate analyses for each sex, the risk elevations were significant in men only. There was also a significant interaction between sodium excretion and body mass index for cardiovascular and total mortality; sodium excretion predicted mortality in men who were overweight, but not in normal weight subjects. The increase in risk was independent of blood pressure and potassium excretion (63). The sodium intake in the lowest sodium excretion groups was below 159 mmol/L in men and 119 mmol/L in women, corresponding to approximately 4 g sodium (10 g salt) and 3 g sodium (7.5 g salt) per day, respectively.

According to a meta-analyses of prospective studies with 19 independent cohorts, 177,025 participants and over 11,000 vascular events, higher salt intake of approximately 6 g per day was associated with a 23 % higher incidence of stroke and a 14 % higher incidence of cardiovascular events. (64)

In a review Perry (65) concludes that available studies suggest that sodium intake is independently related to left ventricular hypertrophy, a condition that is associated with increased risk of coronary mortality. Long-term sodium restriction decreases left ventricular hypertrophy of hypertensive subjects (66, 67).

Several studies indicate a positive relationship between sodium and calcium excretion and that the sodium intake may play a role in the aetiology of osteoporosis and kidney stones (68).

Salt reduction and cardiovascular risk

A review of controlled studies in which the sodium intake was restricted did not reveal any evidence of adverse effects of moderate sodium restriction (69). The analysis included 20 randomised intervention studies with at least 6 months follow-up and urinary excretion data. A Cochrane review did not find any benefits of reduced dietary salt reduction for the prevention of cardiovascular disease. The meta-analysis included seven randomized trials with a follow-up at least 6 months, 6250 participants and 665 deaths, and the analyses were done separately for normotensive and hypertensive subjects (70). One of the included trials was done for subjects suffering from severe heart failure (71). The participants were severely salt and water depleted due to medication with large doses of diuretics and fluid restriction to 1000mL per day. A re-analysis of these studies were done combining data for hypertensives and normotensives together and excluding the study done for subjects suffering from severe heart failure (72). The analysis showed that a decrease in salt intake of 2 – 2.3 g per day decreased cardiovascular events by 20 % ($p<0.05$) and all-cause mortality non-significantly by 5-7 %.

Recommended intake

According to epidemiological studies hypertension is practically non-existent in populations with low salt intake. A lower sodium intake will attenuate the usual blood pressure increase with age. Data from individual trials and meta-analyses of previous trials show that reduction of sodium decrease blood pressure. The effect is greater among hypertensives subjects. The magnitude of blood pressure decrease of sodium restriction also depends on the dietary composition, and seems to be more pronounced when the diet is less optimal, e.g. with respect to the balance between the energy providing nutrients, fibre, potassium and calcium and possibly other constituents, provided by e.g. fruit and vegetables.

Observational studies suggest that population blood pressures, cardiovascular morbidity and mortality have declined together with decreased salt intake. Blood pressure is a strong independent risk factor for CVD. A lower sodium intake is associated with decreased risk of CVD morbidity and mortality. A reduction in salt intake might decrease cardiovascular events. It has been estimated that cardiovascular benefits of reduced salt intake are on par with benefits of a population-wide reductions in tobacco use and would be highly cost-effective (73).

Adults

There is a progressive dose-response relationship between sodium intake and blood pressure. Any recommendations on the sodium intake thus have to be based on practical and public health considerations, rather than on a precise estimate of an optimal physiological intake. Based on a pragmatic evaluation of the available data, a sodium intake of less than 100 mmol (2.3 g) per day (5.8 g salt) would be feasible at the population level.

The current average sodium intake in the Nordic countries can be estimated at 3-4.5 gram per day (8-11 g salt). The proposed population targets would therefore require a reduction in the average population intake of approximately 1-2 g sodium (3-5 g salt) per day.

Children

Blood pressure rise with age begins in early childhood. Systolic blood pressure level exceeds already at the age of 10 years the systolic blood pressure level observed in low-salt populations. Blood pressure measured in childhood tracks with the level measured in adulthood and predicts early atherosclerosis in adulthood. Available data suggest that a reduction in sodium intake in young age is associated with a lower blood pressure in later life. A use of a lifelong low salt diet beginning in early childhood is recommended. It is also prudent to limit sodium intake in childhood in order to avoid preference for a diet with a high salt level. The recommended sodium intake for children up to 13 years age is set to 0.25 g per 1000 kJ, which is based on the energy-adjusted recommended levels for adult women.

Pregnancy and lactation

Pregnancy as well as lactation are associated with a small increase in the physiological requirements for sodium, i.e. about 0.07 g or 3 mmol per day (pregnancy) and 0.12 or 5.2 mmol per day (full lactation). These amounts are small and can apparently be handled by the homeostatic system of the body. There is a lack of evidence to suggest that sodium requirements during pregnancy and lactation differ from that of non-pregnant women.

International expert reports

As early as 1982, a WHO report on prevention of cardiovascular disease (74) recommended that the salt intake should not exceed 5 g/d. This recommendation was based on various clinical and epidemiological data. Since then, several international and national expert bodies including WHO (75, 76), U.S. Food and Nutrition Board (77), American Heart Association (78, 79), and a British Expert Panel (80) have published recommendations to limit salt intake to 6 g/d among adults. The Panel also sets recommendations for children and adolescents. A joint report from three German institutes recommends that salt intake in the German population should be reduced to between 3.5 g/d and a maximum of 6 g/d (BfR 2011). The importance of population-wide sodium reduction as means to prevent

cardiovascular disease and stroke has been pointed out by American Heart Association (81) and Nice (82).

Reasoning behind the recommendation

There is a progressive dose-response relationship between sodium intake and blood pressure. Any recommendations on the sodium intake thus have to be based on practical and public health considerations, rather than on a precise estimate of an optimal physiological intake. Based on a pragmatic evaluation of the available data, a sodium intake of less than 100 mmol (2.3 g) per day (5.8 g salt) would be feasible at the population level. Thus, the long-term recommendation in NNR 2004 is maintained.

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