Salt and arterial hypertension. Epidemiological, pathophysiological and preventive aspects

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Salt and arterial hypertension — epidemiological, pathophysiological and preventive aspects

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Abstract
Arterial hypertension in majority of cases is the result of a poor lifestyle. The table salt intake level has changed over the centuries. In populations characterized by a low level of daily salt consumption, hypertension was a rarity and cardiovascular diseases were much less common. More and more food is sold in processed form, and thus containing large amounts of salt. The amount of salt delivered with food significantly exceeds the actual body's needs. Limitation of salt intake should apply to both prevention of hypertension and cardiovascular diseases as well as treatment of people suffering from hypertension. High sodium intake leads to increased systolic and diastolic pressure, increases the risk of cardiovascular disease as well as other diseases such as stomach cancer etc. There are many mechanisms triggered by excessive intake of table salt that lead to an increase in blood pressure. Excess salt adversely affects the entire human body, and in particular the kidneys and blood vessels. National and International Scientific Societies have developed many documents in the form of guidelines
on the optimal daily sodium intake. Compliance with the recommendations and limitation of salt intake may significantly reduce the incidence of hypertension in the general population.

**Key words:** salt; sodium; arterial hypertension; cardiovascular disease

**Introduction**

Nowadays, arterial hypertension (AH) is considered as one of the major health, social and economic problems throughout the world as well as in Poland. According to World Health Organization (WHO) reports, nearly 1 billion people older than 25 are hypertensive. Increased blood pressure is the most important risk factor of death in the world [1]. Raised BP is a major cause of cardiovascular disease, being responsible for 62% of strokes and 49% of coronary events [2], as well as it is a recognized risk factor for heart and renal failure. In patients with AH, the general risk of cardiovascular disease (CVD) is higher compared to those without AH [3]. According to the data from the NATPOL 2011 study (National Study on the Prevalence of Cardiovascular Risk Factors), the most common CVD risk factors in Poland are hypertension (9.5 million; 32%) and hypercholesterolaemia (18 million; 61%) [4]. High incidence of hypertension, among other things, is associated with a low level of public awareness (adolescents and adults) about the risk factors of this disease [5, 6]. There are two major forms of hypertension – primary hypertension dependent on genetic predisposition and environmental factors (obesity, salt intake, low physical activity) and secondary being the consequence of other diseases (chronic kidney disease, hormonal disorders like the primary hyperaldosteronism, hypercortisolism etc.) [7]. The development of primary hypertension depends on several health determinants (including poor lifestyle) described by Marc Lalonde (Fig. 1).

Among the factors that make up lifestyle one is daily salt intake level. The importance of salt consumption in hypertension development has been emphasized for many years, first by Ambard & Beaujard in 1904 and later by Allen, Meneely and Dahl [8].

Sodium chloride (NaCl) is the main form of sodium intake. The correct concentration of sodium in the body is 135–145 mmol/L [9].

**Level of salt intake**
In Poland, approximately 13 grams of salt is consumed daily with food. This classifies our country at one of the top places in Europe in terms of the amount of salt consumed [10]. The amount of salt ingested differs significantly around the world — from less than 0.5 g of salt a day among Indians from the South American Forests, up to 10–12 g/day in developed countries. Such large salt intake causes increased \( \text{Na}^+ \) retention by kidneys, what partially justifies the high incidence of hypertension in developed countries [11]. Data from the INTERSALT study indicate a positive correlation between the amount of salt excreted in urine and the increase of blood pressure level with progressing age [12]. It is assumed that diurnal sodium excretion in urine, providing normal renal function and correctly collected urine, corresponds to approximately 93% of the daily sodium intake [13]. Sources of dietary sodium include: processed foods, table salt voluntarily added to meals and salt delivered in water and drugs [14].

According to the data from NHANES study (National Health and Nutrition Examination Survey) the highest consumption of salt is noted among men aged 40–49, whereas children aged 2–5 years of age consume the smallest amount of salt [15]. Italy and Spain are the countries with the largest salt intake observed in Europe (Fig. 3) [16].

Published studies indicate that 72–85% of salt comes from the processed foods, 15–20% is added during meal preparation and about 8% comes from drugs and water [17]. Interesting results on the variation in the supply of sodium on the basis of sex were published by Czerwińska et al. among Warsaw population (n = 42, age 20–30 years), it was shown that the amount of sodium consumed by women was 3.1 g/day, while among men 4.7 g/day. In addition, it was shown that the main source of sodium for men were processed products, while for women salt that was added during the preparation of meals [18].

Studies providing information on salt intake suggest even higher daily salt intake in Poland. In 2008, the average daily intake of salt in men was 14.7 g and for women 8.6 g [19]. Data from the EPOGH study showed that in the period 1998–2002 Poles’ average daily intake of salt was 15 g. This was the highest level compared to other countries taking part in the study (Belgium, Italy, Russia, the Czech Republic, Romania) [20]. According to the Polish Institute of Food and Nutrition the salt intake in Poland is 10 g/day [9].

The daily requirement of sodium is about 2 g (86 mmol/l; about 5 g NaCl) for both women and men over the age of 18 [21]. Other sources suggest even smaller daily sodium need in
adults — about 1.3 g [22]. It is worth remembering that in 2.5 grams of NaCl there is about 1 g of sodium (Na; 43 mmol of NaCl there is about 2.5 g NaCl).

**High salt and cardiovascular mortality**

Data showing that increased salt intake increases mortality are from the INTERSALT Study (Fig. 5) [12].

The incidence of strokes in some regions of Japan is directly proportional to the level of table salt intake. These observations have contributed to an intensive campaign supported by the central government to reduce salt intake. The campaign has been successful – over the decade, average consumption for the whole country has fallen from 13.5 to 12.1 grams of salt a day and consumption in the northern regions has fallen from 18 to 14 grams a day. During the same observation period, there was a decrease in blood pressure in adults and children and an 80% reduction in stroke mortality [23]. A meta-analysis conducted by Strazullo et al. covering 12 cohort studies showed that an increase in sodium intake by 5 g/day leads to an increase in the risk of stroke by 23% and cardiovascular disease by 17% [24]. The EPOGH study did not confirm the results of the meta-analysis of Strazullo et al. [25]. However, the inclusion of EPOGH results into the meta-analysis of Strazullo et al. did not change the statistical significance of the results of this meta-analysis. Based on the analysis of the results of Strazullo et al. and EPOGH, it was calculated that reduction of NaCl consumption by 6 g/day reduces the risk of stroke by 24% and cardiovascular diseases by 18% (a reduction in the number of complications of these diseases by 2.5 million during the year) [26]. Sasaki et al. conducted a study in which a reduction in salt consumption by an average of 4 g/day in the population of northern Japan caused a reduction in BP and a reduction in mortality due to a stroke of 80% [23]. In over 11,000 patients participating in Scottish Heart Health Study, aged 40–59, in long-term follow-up the relationship was found between higher salt and an increased risk of coronary events in women, but this relationship did not occur in the observed men [27]. The TOHP I and TOHP II (Trial of Hypertension Prevention) trials were attended by 3,000 people without hypertension. The subjects were divided into two groups: 1. with reduced sodium intake (0.44 mmol/day for 18 months in TOHP I and 0.33 mmol/day for 36–48 months in the TOHP II study); 2. control group. The follow-up period was 10 years in TOHP I, whereas in the TOHP II study it was 15 years. The results of both studies showed a significant reduction (by about 30%) in the incidence of cardiovascular complications and deaths from cardiovascular causes [28].
The results of the DASH and NHANES I studies showed that reduction of NaCl consumption by 100 mmol/day (about 5.8 g/day) reduces the risk cerebrovascular events by 32% [29, 30]. From the blood pressure reduction seen in a meta-analysis [31], it was estimated that a reduction in salt consumption to 5 g per day would reduce incidence of stroke by 24% and coronary heart disease by 18%. This would prevent approximately 35,000 strokes and CHD deaths a year in the UK and approximately 2.5 million deaths worldwide [32]. Hummel et al. reduced sodium intake in 443 patients with heart failure, obtaining a 30-day reduction in the number of deaths [33].

There are well-known observations of the localities in Portugal, where the residents consumed a very large amount of NaCl (21 g/day), while the risk of developing hypertension and stroke was very high. In one of these towns, for two years, there was an intensive educational campaign on reducing salt in food and replacing rich-salt products with less salt. In the place where the consumption of salt decreased by 50%, the mean arterial pressure was 13/6 mm Hg lower than in the city without medical intervention [34]. Based on the analysis of the results of available studies, He et al. estimated that limiting salt intake to 3 g/day leads to a reduction in stroke rates by 13% and coronary heart disease by 10% [26].

Since the beginning of the 1970s, Finland has introduced a policy of reducing salt consumption in the entire population. The main strategy included cooperation with the food industry to produce food products with reduced sodium content and raising public awareness of the harmful effects of salt. Over 30 years, salt intake has dropped by one-third with associated systolic and diastolic blood pressure drop. A reduction in the incidence of stroke and mortality from cardiovascular disease by 75–80% was noted as well as a 6-years life prolongation. Both the body mass index and alcohol consumption increased over the period, therefore lowering salt intake is the main factor that contributed to the reduction of arterial pressure in Finnish population [35].

Salt and blood pressure

Important data on the effect of salt on blood pressure value is summarized in a paper by He et al. In the meta-analysis of randomized studies on moderate salt reduction, BP in hypertensive patients decreased by 5.0/2.7 mm Hg (SBP/DBP respectively) with restriction of dietary salt from 9.5 g/day to 5.1 g/day. In normotensive subjects, the decrease in BP was about half of that observed in hypertensive patients. Therefore, systolic BP decreases about 1 mmHg in hypertensive patients and about 0.5 mmHg in normotensive subjects for each 1
g/day of salt reduction [36]. A decrease of 40 mmol/day (about 2.33 g NaCl) of salt consumption was related to a decrease in systolic/diastolic blood pressure by 4.3/2.0 mmHg. Evaluations of a “no added salt diet” on a hypertensive population with high dietary sodium intake (and on no hypotensive drugs) revealed beneficial effects. 24-hour blood pressure recording and 24-hour urinary sodium consumption were measured before and after 6 weeks of the “no added salt diet”. Despite a modest effect on dietary sodium restriction, the “no added salt diet” significantly decreased systolic and diastolic blood pressure [37]. Lowering the supply of NaCl in both humans and animals is associated with a decrease in blood pressure, while an increased supply of NaCl (250 mmol/day) increases the systolic pressure by 9.4 mm Hg [38]. The analysis of 57 studies performed by Jürgens G. et al. of the influence of sodium intake levels on blood pressure concluded that reduced sodium intake in Caucasians with elevated blood pressure has a beneficial effect on reducing blood pressure in the short term. The results suggest that the effect of low versus high sodium intake on blood pressure was greater in Black and Asian patients than in Caucasians [39]. A meta-analysis of 17 studies in hypertensive patients (n = 734) and 11 studies in subjects with normal arterial pressure (n = 2220) showed that the reduction of diurnal sodium urinary excretion by 78 mmol (4.6 g salt) in patients with hypertension was associated with a decrease of 4.97 mm Hg in systolic blood pressure and 2.74 mm Hg in diastolic pressure. In the subjects with normal blood pressure, a similar reduction in diurnal sodium excretion in the urine led to a decrease in systolic blood pressure by 2.03 mm Hg and diastolic blood pressure by 0.99 mm Hg. In addition, a significant correlation was found between the reduction of urinary sodium excretion and lowering of blood pressure [40].

A study using a 24-hour ambulatory blood pressure monitoring showed that reducing the sodium content in the diet by only 50 mmol (150–100 mmol/day) for 4 weeks is associated with a reduction in blood pressure by 6 mm Hg [41]. Meta-analysis of the reduction of NaCl supply showed that its reduction by 3 g lowers blood pressure in patients with hypertension by 5.6 mmHg, and in people with normal BP by 3.5 mm Hg [26]. Similar results were obtained by the Japanese who reduced the salt intake to 2.3 g/day, thereby lowering the arterial pressure by 3.1 mm Hg compared to the places where the salt intake was high [42]. The studies quoted above show a very strong relationship between the amounts of salt consumed level of arterial pressure.

Salt and the risk of developing arterial hypertension
Salt is one of the major factors that increase blood pressure and is therefore responsible for strokes and heart attacks every year [43]. A number of epidemiological, experimental, and clinical studies have clearly demonstrated that excessive salt intake is related to BP increase and the progression of hypertension [40, 44, 45]. The influence of excessive sodium intake on blood pressure was first described in 1984 by Norman Kaplan. He stated that the diet in developed societies repeatedly rushes the daily provision for sodium, which may explain the higher incidence of hypertension in these areas [46].

The most important clinical trials concerning the influence of the salt intake level on the blood pressure value are: INTERSALT (1996), DASH SODIUM (2001), TONE (2001) [12, 47, 48].

Based on many studies, it is assumed that the threshold of sodium intake affecting the increase of arterial blood pressure is 50–100 mmol/day. Lack of hypertension and no increase of arterial blood pressure with age in populations with a level of sodium consumption < 50 mmol/L/24 hours (< 2.91 g NaCl) justifies the theory of the threshold [49]. Observation of 20 patients with an average blood pressure of 163/100 mm Hg, consuming an approximately 11.2 g NaCl/day showed that lowering the salt intake to 6.4 g/day during the month, reduced blood pressure to an average of 155/95 mm Hg. In the following month, after lowering the level of salt consumed to 2.9 g/day, the arterial pressure was 147/91 mm Hg. After the study, 19 people decided to continue limiting salt intake. After one year their blood pressure was 142/87 mm Hg [50]. Experimental studies indicate that increasing the level of sodium consumed from 137 to 142 mmol/L (physiological concentration range!) reduces the activity of nitric oxide synthase (NOS) by 25% [51]. In experimental studies, it was shown that washing smooth muscle cells (myocytes) with NaCl at a higher than physiological concentration (131–151 mmol/L) leads to hypertrophy of these cells, increased protein synthesis and slowed down protein degradation [52]. Important data come from the INTERSALT study, which investigated the effect and relation of salt intake and systolic blood pressure [12]. The INTERSALT results, which agree with findings from other diverse studies [53, 54] including data from clinical observations, therapeutic interventions, randomized controlled trials, animal experiments, physiologic investigations, evolutionary biology research, anthropologic research, and epidemiologic studies, support the judgment that habitual high salt intake is one of the quantitatively important, preventable mass exposures causing the unfavourable population-wide blood pressure pattern that is a major risk factor for the epidemic of cardiovascular disease. Data from the mentioned INTERSALT study, with 10,079
participants, indicate a positive correlation between the amount of salt excreted in urine and the increase in blood pressure with age, as well as positive correlation of the amount of salt excreted in urine and death from stroke [55]. Based on the results of the INTERSALT study, it was found that an increase in sodium consumption by 6 g/day causes a rise in blood pressure by 9 mm Hg [12]. The Salt Institute (an institution representing kitchen salt producers) criticized the results of the INTERSALT Study. According to this criticism, the rejection of the results of the study from the four centres where NaCl consumption was the smallest causes the disappearance of the correlation between blood pressure values and renal excretion of sodium. The opinion of the salt institute does not agree with the vast majority of authors of studies on the impact of salt on human health [11].

Confirmation of the results of the INTERSALT study was obtained in the Dahl L.K. study. The results of his work confirmed a positive correlation between the level of daily sodium intake and the prevalence of hypertension in selected geographical areas. The authors summarized that among societies or groups habitually consuming low salt diets (perhaps 5 g of NaCl per person per day or less) essential hypertension would be uncommon. Among those consuming high amount of salt (in excess of 10–15 g per person per day) essential hypertension will be common. Individual susceptibility will determine which individual will develop the disease [56].

**Sodium consumption influence on other diseases**

The results of numerous studies indicate a relationship between excessive salt intake and the risk of other diseases (Tab. 1).

A six-week reduction in salt intake from 6.2 to 1.7 g/day gives a noticeable echocardiographic reduction in heart weight (left ventricular mass) [58]. In 9 studies, a significant correlation was observed between the amount of salt in the diet and the size of the left ventricle, and the correlation coefficient ranged from 0.22 to 0.61 [59]. Increased sodium intake impairs diastolic heart function in patients with hypertension or type 2 diabetes [60]. Increasing the amount of salt in the diet leads within a few weeks not only to the hypertrophy of the left ventricle but also to the fibrosis of this chamber [61]. In 2001, a study was completed in Finland, in which 1173 men and 1263 women were observed for more than 12 years using a salt-rich diet. The greater part of ischaemic heart disease and death due to cardiovascular causes was observed. Interestingly, both the incidence of cardiovascular diseases and deaths from this causes did not correlate with the level of blood pressure [62].
In studies on animal models, an increase in sodium consumption led to increased protein loss with urine and significantly impaired kidney’s function. Studies in humans at the population level have shown that sodium intake is associated with proteinuria and albuminuria [63]. The reduction of salt intake resulted in a decrease (regardless of the effect on BP) in the incidence of chronic kidney disease and proteinuria [64]. Experimental studies have shown that a diet rich in NaCl leads to a reduction in the angiotensin-converting type II enzyme level (ACEII) and to an increase in angiotensin II (AngII), which results in renal glomerular fibrosis (glomerulopathy) [65]. In studies conducted in postmenopausal women, a relationship was found between the loss of hip bone density and the diurnal sodium excretion in urine, which indicates the unfavourable role of excessive sodium intake in the pathogenesis of osteoporosis [66, 67]. Excessive salt intake also leads to suppression of superoxide dismutase, which results in an increase of the level of O$_2^-$ . The increase in the concentration of free radicals promotes the development of atherosclerosis [68]. High level of salt intake may also be an indirect cause of obesity by affecting the intake of fluids. It has been shown that in adults, a daily intake of approx. 10 g of salt leads to an increase in liquid consumption by 350 mL per day, compared to the amount of liquids, taken with the recommended intake of 5 g of salt/day. Because sweet drinks have a significant share in the consumption of liquids, salt intake contributes indirectly to the increase in the amount of calories consumed and the development of overweight and obesity [69]. Increased intake of sodium in the diet and urinary excretion of sodium correlate with the greater incidence of stomach cancer. Meta-analysis by D'Elia et al. showed a 68% higher risk of gastric cancer in the population consuming higher amounts of sodium [70]. In other studies, there was a relationship between the level of sodium consumption and the severity of the course of bronchial asthma and the development of cataracts [57]. A study by Gonzalez et al. conducted in 2012 showed higher levels of insulin and C-reactive protein (CRP), lower high-density lipoprotein (HDL) cholesterol levels and higher insulin resistance in people using a high-sodium diet compared to those using a low-sodium diet [71]. Another study conducted in London on a group of 803 men showed no relationship between plasma renin activity and cardiovascular events in both hypertensive and non-hypertensive men [72].

**Low salt intake and risk of cardiovascular diseases**

The multi-centre EPOGH (European Project on Genes in Hypertension) study (n = 1499; 6,1-year observation), based on a six-year observation of healthy populations from five
European countries, aimed to confirm the thesis that lower sodium intake lowers arterial BP. The results of the EPOGH study confirmed that people who increased their daily salt intake (measured by daily sodium excretion in the urine) showed higher values of systolic blood pressure; for diastolic pressure, there was no statistical significance. The increase in systolic blood pressure was 1.71 mm Hg/100 mmol sodium (about 6 g NaCl). Analysis of the results of the EPOGH study (n = 3681, 8-year observation) showed that reduction of salt in the diet (5 g NaCl/day) increases the risk of cardiovascular mortality by 56% [25, 73].

The influence of low sodium consumption on the increase of cardiovascular disease is explained by three pathological mechanisms: 1. activation of the renin-angiotensin-aldosterone system; 2. increase in tissues’ insulin resistance; 3. activation of the autonomic nervous system (sympathetic part) [74, 75]. Three important facts may explain the above research results: 1. Only a significant reduction in the amount of NaCl consumed in a short time leads to an increase in plasma renin activity, an increase in the concentration of angiotensin II and aldosterone. Gradual and slow reduction of NaCl consumption does not lead to the above effects [76]; 2. People taking part in the research could have problems with obeying a salt-poor diet (inadequate compliance); 3. There is a possibility of a J-curve effect that justifies the desirability of conducting further research on determining the optimal daily dose of NaCl [72]. Alderman and Cohen performed an analysis of the results of 32 randomized clinical trials, covering the data of 360,000 subjects, and found that cardiovascular complications were highest among those who consumed less than 2.3 g of sodium and more than 3.4 grams of this element [77]. Analysing the results of the research it can be concluded that there is a J-curve phenomenon of sodium supply and its consumption should fluctuate within certain limits [72].

In the available literature, you can find data confirming three theses:

**Selected pathophysiological mechanisms**

The main pathophysiological mechanisms linking high salt intake with cardiovascular disease are [51, 77–82]:

- increased volume of extracellular fluid — sodium is a strong osmolyte;
- increased stiffness of endothelial cells — epithelial sodium channel (ENaC);
- increase kidney sodium retention — increase in ouabaine secretion;
- reducing the degradation of catecholamines — renalase enzyme;
- hyperactivity of the renin–angiotensin–aldosterone system (RAAS);
— reduced NOS activity.

**Prevention aspects. Recommendations for sodium consumption**

The DASH study (Dietary Approaches to Stop Hypertension) conducted on 412 people showed a beneficial effect of reducing the amount of sodium in the diet on the level of arterial pressure. In the DASH study, subjects were divided into three diet groups: control — a diet low in vegetables and fruits, a large amount of meat, saturated fatty acids, snacks, sweets; **vegetables and fruits** — rich in vegetables and fruits, small amount of snacks and sweets; DASH — rich in fruits, vegetables, defatted dairy products, few snacks and sweets, meat, fats, saturated fatty acids. The observation lasted four weeks. Blood pressure was measured by the ABPM method. In the group using a diet rich in vegetables and fruit, systolic blood pressure dropped by 2.8 mm Hg on average, and diastolic blood pressure decreased by 1.1 mm Hg. In the group that used the DASH diet — reduction in systolic blood pressure was 5.5 mm Hg, diastolic 3 mm Hg. The effect of diet was most visible among people with hypertension, whose systolic blood pressure dropped on average by 11.4 mm Hg and diastolic blood pressure by 5.5 mm Hg. This reduction in blood pressure is comparable with the use of 1–2 antihypertensive drugs [30]. The continuation of the DASH-Sodium study was the Low-Sodium-DASH study in which the effects of diets from the DASH-Sodium study were observed in combination with the reduction of salt intake by: 3.5; 2.3; 1.2 grams a day for 30 days. Irrespective of the level of sodium in the diet, the DASH diet significantly reduced BP. In groups with low, medium and high levels of salt intake, a reduction in systolic blood pressure was observed, respectively of 5.9; 5.0; 2.2 mm Hg, and diastolic of 2.9; 2.5; 1.0 mmHg. The most significant level of blood pressure reduction was observed in the group using the DASH diet together with low salt intake (8.9 mm Hg lower systolic blood pressure, diastolic pressure 4.5 mm Hg), especially in patients with arterial hypertension (lowering systolic blood pressure by 11.5 mm Hg) [76].

So far, the optimal daily sodium intake has not been determined. Experimental, epidemiological and clinical studies indicate the need to gradually reduce the sodium content in the diet. It is recommended to limit the consumption of sodium to about 70–100 mmol/day (4–5.8 g NaCl/day) [83]. The reduction of sodium intake should initially amount to 7–8 NaCl/day followed by 5–6 g NaCl/day. The research results indicate that limiting NaCl consumption below 3 g/day may induce adverse consequences [72].
According to the regulations in Poland and Europe, there is an obligation to add potassium iodide (KI) to the salt in an amount of 30 ± 10 mg/kJ/kg salt. These recommendations have led to a significant reduction in the prevalence of endemic volition in children. The recommendations for salt and iodine consumption are mutually exclusive, therefore it is recommended to provide iodine to the body by alternative methods, such as drinking mineralized waters, milk (200 mL of milk contains 13–30% of the daily requirement for iodine), consumption of fresh fish [86–88].

Summary

Experimental, epidemiological and clinical studies prove that today’s dietary habits lead to the development of hypertension, cardiovascular diseases, and kidney diseases. Excessive supply of salt stimulates many mechanisms involved in the development of hypertension. The reduction of sodium intake is particularly recommended for people with hypertension, type 2 diabetes, acute kidney disease (chronic renal failure with hypertension), and obesity, because these diseases have higher susceptibility to high blood pressure [17]. Limiting the consumption of sodium significantly improves the effects of antihypertensive drugs (in addition to calcium antagonists) and can even replace 1–2 antihypertensive drugs [47] in some cases. Unfortunately, limiting sodium in diet is difficult, because the vast majority of sodium is delivered in processed products (according to various studies — 72–85%). The immediate possibility of reducing sodium consumption is preparation of fresh foods at home. For people who have problems with limiting the amount of sodium consumed, it is recommended to use potassium chloride (KCl) instead of sodium chloride (NaCl) [87]. A study of veterans living in Taiwan (2.5 years of observation) showed that a 17% reduction in NaCl consumption with a 76% increase in KCl consumption resulted in a 40% reduction in the risk of cardiovascular death [89] Recommendations concerning the salt intake by healthy people are not conclusive — it is not known exactly to what values the salt supply is to be reduced.

Potential conflict of interest

The authors do not declare any conflict of interest.

References


**Figure 1.** Health determinants. Marc Lalonde, Ottawa 1978
Figure 2. Sources of dietary salt [14]

![Sources of dietary salt](image)

Figure 3. Level of salt intake in selected countries. WHO 2008–2013 [16]. SDT — suggested dietary target for salt

![Level of salt intake in selected countries 2008–2013](image)

Figure 4. Salt content in selected food products [9, 17]
**Sodium content in fresh and processed products**

![Sodium content graph](image)

**Figure 5.** Results from INTERSALT study [12]

**Table 1.** Effects of salt intake on health conditions [57]

<table>
<thead>
<tr>
<th>Condition</th>
<th>Level of evidence*</th>
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<tr>
<td>Blood pressure</td>
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<td>Stroke</td>
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<td>Cardiovascular diseases</td>
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<td>Left ventricular hypertrophy</td>
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**Table 2.** Effect of sodium on cardiovascular risk — comparison of results of observational studies

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<td></td>
<td>European Project on Genes in Hypertension, 2011</td>
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<td>The level of salt intake does not</td>
<td>Scottish Heart Health Study, 1997</td>
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<td>affect cardiovascular risk</td>
<td>Multiple Risk Factor Intervention Trial, 2000</td>
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<td></td>
<td>Trial of Hypertension Prevention, 2009 (TOHP)</td>
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<td>High salt intake increases cardiovascular</td>
<td>NHANES I (people with obesity),</td>
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<td>Glomerular filtration</td>
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<td>Kidney stones (Ca oxalate)</td>
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<td>Calcium excretion</td>
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<td>Bone demineralization</td>
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<td>Bone fractures</td>
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<td>Stomach cancer</td>
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<td>Fluid retention</td>
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<tr>
<td>Cataract</td>
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<td>Asthma</td>
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<td>Meniere’s disease</td>
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risk 2000

➤ Finish Heart Study, 2001
➤ Trials of Hypertension Prevention, 2007 (TOHP)

Table 3. Recommendations for sodium consumption

<table>
<thead>
<tr>
<th>Organisation</th>
<th>Recommendation</th>
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<tr>
<td>World Health Organisation</td>
<td>5–6 g/day</td>
<td>[83]</td>
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<td>7th Joint National Committee</td>
<td>No more than 2.3 grams/day (6 g NaCl/day)</td>
<td>[19]</td>
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Table 4. Methods of limiting salt intake [9]

Methods of limiting salt intake according to the Institute of Food and Nutrition in Warsaw

1. Limiting the addition of salt during the preparation of meals
2. If you need to add salt — add it only at the end of cooking
3. Using fresh and dried herbs instead of salt
4. Replacing NaCl with sodium and potassium salt (mixture of KCl and NaCl)
5. Choosing products with a lower salt content (GDA — indicated daily intake — %)