

Sodium in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

© World Health Organization 2003

All rights reserved. Publications of the World Health Organization can be obtained from Marketing and Dissemination, World Health Organization, 20 Avenue Appia, 1211 Geneva 27, Switzerland (tel: +41 22 791 2476; fax: +41 22 791 4857; email: bookorders@who.int).

Requests for permission to reproduce or translate WHO publications - whether for sale or for noncommercial distribution - should be addressed to Publications, at the above address (fax: +41 22 791 4806; email: permissions@who.int).

The designations employed and the presentation of the material in this publication do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries.

The mention of specific companies or of certain manufacturers' products does not imply that they are endorsed or recommended by the World Health Organization in preference to others of a similar nature that are not mentioned. Errors and omissions excepted, the names of proprietary products are distinguished by initial capital letters.

The World Health Organization does not warrant that the information contained in this publication is complete and correct and shall not be liable for any damages incurred as a result of its use

Preface

One of the primary goals of WHO and its member states is that “all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water.” A major WHO function to achieve such goals is the responsibility “to propose regulations, and to make recommendations with respect to international health matters”

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as International Standards for Drinking-Water. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO Guidelines for drinking-water quality (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared/updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A “final task force” meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO internet site and in the current edition of the GDWQ.

Acknowledgements

The work of the following coordinators was crucial in the development of this background document for development of WHO *Guidelines for drinking-water quality*:

- J.K. Fawell, Water Research Centre, United Kingdom
(inorganic constituents)
- U. Lund, Water Quality Institute, Denmark
(organic constituents and pesticides)
- B. Mintz, Environmental Protection Agency, USA
(disinfectants and disinfectant by-products)

The WHO coordinators were as follows:

Headquarters:

- H. Galal-Gorchev, International Programme on Chemical Safety
- R. Helmer, Division of Environmental Health

Regional Office for Europe:

- X. Bonnefoy, Environment and Health
- O. Espinoza, Environment and Health

Ms Marla Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

The efforts of all who helped in the preparation and finalization of this document, including those who drafted and peer reviewed drafts, are gratefully acknowledged.

The convening of the experts meetings was made possible by the financial support afforded to WHO by the Danish International Development Agency (DANIDA), Norwegian Agency for Development Cooperation (NORAD), the United Kingdom Overseas Development Administration (ODA) and the Water Services Association in the United Kingdom, the Swedish International Development Authority (SIDA), and the following sponsoring countries: Belgium, Canada, France, Italy, Japan, Netherlands, United Kingdom of Great Britain and Northern Ireland and United States of America.

GENERAL DESCRIPTION

Identity

<i>Compound</i>	<i>CAS no.</i>	<i>Molecular formula</i>
Sodium	7440-23-5	Na
Sodium chloride	7647-14-7	NaCl
Sodium carbonate	492-19-8	Na ₂ CO ₃
Sodium hypochlorite	7681-52-9	NaOCl
Sodium metasilicate	1344-09-8	NaSiO ₃

Physicochemical properties (1–5)

<i>Property</i>	<i>Na</i>	<i>NaCl</i>	<i>Na₂CO₃</i>	<i>NaOCl</i>	<i>NaSiO₃</i>
Melting point (°C)	97.83	801	851	–	–
Boiling point (°C)	886	1413	De- composes	–	–
Density at 20 °C (g/cm ³)	0.71	2.17	2.53	–	–
Vapour pressure (kPa)	0.133	–	–	–	–
Water solubility at 0 °C (g/l)	reacts violently	357	71	infinitely soluble	soluble

Organoleptic properties

The taste threshold for sodium in water depends on the associated anion and the temperature of the solution. At room temperature, the threshold values are about 20 mg/litre for sodium carbonate, 150 mg/litre for sodium chloride, 190 mg/litre for sodium nitrate, 220 mg/litre for sodium sulfate, and 420 mg/litre for sodium bicarbonate (6).

Major uses

Metallic sodium is used in the manufacture of tetraethyl lead and sodium hydride, in titanium production, as a catalyst for synthetic rubber, as a laboratory reagent, as a coolant in nuclear reactors, in electric power cables, in nonglare lighting for roads, and as a heat-transfer medium in solar-powered electric generators (3). Sodium salts are used in water treatment, including softening, disinfection, corrosion control, pH adjustment, and coagulation (7), in road de-icing and in the paper, glass, soap, pharmaceutical, chemical, and food industries.

Environmental fate

Sodium salts are generally highly soluble in water and are leached from the terrestrial environment to groundwater and surface water. They are nonvolatile and will thus be found in the atmosphere only in association with particulate matter.

ANALYTICAL METHODS

Sodium concentrations can be determined by direct aspiration atomic absorption spectroscopy (8). Detection limits of 2 and 40 µg/litre can be achieved with flame atomic absorption spectrometry and inductively coupled plasma atomic emission spectrometry, respectively.

ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Air

The sodium levels in ambient air are low in comparison with those in food or water.

Water

The sodium ion is ubiquitous in water. Most water supplies contain less than 20 mg of sodium per litre, but in some countries levels can exceed 250 mg/litre. Saline intrusion, mineral deposits, seawater spray, sewage effluents, and salt used in road de-icing can all contribute significant quantities of sodium to water. In addition, water-treatment chemicals, such as sodium fluoride, sodium bicarbonate, and sodium hypochlorite, can together result in sodium levels as high as 30 mg/litre. Domestic water softeners can give levels of over 300 mg/litre, but much lower ones are usually found (6).

In a survey of 2100 water samples in the USA in 1963–1966, the sodium ion concentrations found were in the range 0.4–1900 mg/litre; in 42% of the samples, the concentrations were in excess of 20 mg/litre, but in 5% they were greater than 250 mg/litre. In a later survey of 630 water-supply systems in the same country, the sodium ion concentrations found ranged from less than 1 to 402 mg/litre, with similar distribution of values (9).

Food

Sodium is naturally present in all foods and may be added during food processing. Fresh fruit and vegetables contain sodium at concentrations in the range <10–1000 mg/kg; cereals and cheese may contain as much as 10–20 g/kg; and human and cows' milk contains 180 and 770 mg/litre, respectively (6,10).

Estimated total exposure and relative contribution of drinking-water

Food is the main source of daily exposure to sodium, primarily as sodium chloride. The estimation of daily intake from food is difficult because of the wide variation in concentrations and the fact that many people add salt to their food. In western Europe and North America, the estimated overall consumption of dietary sodium chloride is 5–20 g/day (2–8 g of sodium per day), the average being 10 g/day (4 g of sodium) (6). People on a low-sodium diet need to restrict their sodium intake to less than 2 g/day (9). The consumption of drinking-water containing 20 mg of sodium per litre would lead to a daily intake of about 40 mg of sodium.

KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

Virtually all of the sodium present in water and foods is rapidly absorbed from the gastrointestinal tract. Sodium is the principal cation found in the extracellular body fluids; only small amounts are found within cells (11). Some is found in bone, where it acts as a sodium reservoir in maintaining the blood pH.

The level of sodium in extracellular fluids is carefully maintained by the kidney and determines the volume of these fluids (9). Sodium balance is controlled through a complex interrelated mechanism involving both the nervous and hormonal systems (12). Sodium is excreted principally in the urine in amounts reflecting the dietary intake (11).

EFFECTS ON LABORATORY ANIMALS AND *IN VITRO* TEST SYSTEMS

Acute exposure

The LD₅₀ values for rats and mice for sodium ion as the chloride salt are 1180 mg/kg of body weight and 1572 mg/kg of body weight, respectively. An LD₅₀ of 3147 mg/kg of body weight was reported for rabbits (13).

Long-term exposure

Hypertension has been clearly demonstrated in different species of animals given high levels of sodium chloride in their diet (6). Despite the usual reservations about extrapolating animal results to humans, the consistency of the animal data suggests that they should not be ignored. Ingestion of a high-salt diet resulted in hypertension in female Sprague-Dawley rats (14). Approximately 75% of 159 rats fed diets containing 8% sodium chloride (equal to 3597 mg of sodium ion per kg of body weight per day) for 12–15 months exhibited hypertension (systolic blood pressure >18.7 kPa (140 mmHg)) within 6–9 months of the initiation of the diet regimen; the mean blood pressure of these animals increased with age. Rats maintained on a low-salt diet (0.35% sodium chloride, equal to 157 mg of sodium ion per kg of body weight per day) did not exhibit a corresponding increase in blood pressure with age.

Reproductive toxicity, embryotoxicity, and teratogenicity

The reproductive effects of the sodium ion were studied in three strains of pregnant rats (SHR, WKY, and Sprague-Dawley) fed diets containing either 0.4 or 8.0% sodium chloride (equal to 208 and 4196 mg of sodium ion per kg of body weight per day) throughout gestation and lactation (15). Their offspring were also placed on low- or high-sodium diets. Pregnancy rates were decreased in the high-salt group, by 38% in SHR rats and 66% in WKY rats. Although slight nonsignificant increases in systolic blood pressure were noted in high-salt WKY dams, significant decreases were observed in their SHR counterparts. SHR pups fed the high sodium level from high-salt dams had significantly higher blood pressure than offspring from all other groups after 11.5 weeks of exposure and exhibited high morbidity and mortality from peripheral capillary haemorrhage and stroke. Maternal high-salt diets caused depression of postnatal growth in all strains of rats studied.

No developmental effects were observed in the offspring of pregnant mice, rats, or rabbits given oral doses of sodium chloride equivalent to 189, 147, and 147 mg of sodium ion per kg of body weight, respectively, on days 6–15 (mice and rats) or 6–18 (rabbits) of gestation (15,16).

Mutagenicity and related end-points

Sodium (as sodium chloride) produced gene mutations in mouse lymphocyte assays, induced unscheduled DNA synthesis in rats, and caused cytogenetic aberrations in hamster ovaries and lung cells as well as DNA damage in hamster ovaries and mouse lymphocytes (13). The overall importance of these findings is reduced because very high dose levels of sodium ion were used.

Carcinogenicity

It is unlikely that sodium alone is carcinogenic. However, a high-salt diet may enhance the carcinogenic potency of chemicals such as *N*-methyl-*N*'-nitro-*N*-nitrosoguanidine in drinking-water by causing irritation of the gastroduodenal tract, thus increasing the exposure of epithelial cells to the carcinogen and resulting in an increased incidence of gastric tumours (17).

EFFECTS ON HUMANS

Although it is generally agreed that sodium is essential to human life, there is no agreement on the minimum daily requirement. However, it has been estimated that a total daily intake of 120–400 mg will meet the daily needs of growing infants and young children, and 500 mg those of adults (18).

In general, sodium salts are not acutely toxic because of the efficiency with which mature kidneys excrete sodium. However, acute effects and death have been reported following accidental overdoses of sodium chloride (6). Acute effects may include nausea, vomiting, convulsions, muscular twitching and rigidity, and cerebral and pulmonary oedema (12,19). Excessive salt intake seriously aggravates chronic congestive heart failure, and ill effects due to high levels of sodium in drinking-water have been documented (6).

The effects on infants are different from those in adults because of the immaturity of infant kidneys. Infants with severe gastrointestinal infections can suffer from fluid loss, leading to dehydration and raised sodium levels in the plasma (hypernatraemia); permanent neurological damage is common under such conditions. Addition of cows' milk or tapwater containing high levels of sodium to solid food may exacerbate the effects (2,6).

The relationship between elevated sodium intake and hypertension has been the subject of considerable scientific controversy. Although short-term studies have suggested that such a relationship does exist (20), most people in western Europe and North America ingest a high-salt diet from infancy yet do not exhibit persistent hypertension until the fourth decade (6). Whereas reducing the sodium intake can reduce the blood pressure of some individuals with hypertension, this is not effective in all cases (21). In addition, some data for both humans and animals suggest that the action of sodium may be at least partly modified by the level of the accompanying anion as well as that of other cations (22,23). Although several studies suggest that high levels of sodium in drinking-water are associated with increased blood pressure in children (24,25), in other studies no such association has been found (26–28).

A particularly striking observation is that, in "nonwesternized" populations, diets are low in sodium, the prevalence of hypertension is very low, and blood pressure does not increase with age. Although it is tempting to conclude that a causal relationship exists, a number of differences between "westernized" and "nonwesternized" populations might account for the difference. However, the good agreement between these results and those of other studies gives further support to a direct link between raised sodium intake and hypertension (6).

Although there is an association between hypertension and certain diseases, such as coronary heart disease, genetic differences in susceptibility, possibly protective minerals (potassium and calcium), and methodological weaknesses in experiments make it difficult to quantify the relationship, and sodium in drinking-water generally makes only a small contribution to total dietary sodium. No firm conclusions can therefore be drawn at present as to the importance of sodium in drinking-water and its possible association with disease.

CONCLUSIONS

Sodium salts are found in virtually all food (the main source of daily exposure) and drinking-water. Sodium levels in the latter are typically less than 20 mg/litre but can markedly exceed this in some countries. On the basis of existing data, no firm conclusions can be drawn concerning the possible association between sodium in drinking-water and the occurrence of hypertension. No health-based guideline value is therefore proposed. However, sodium may affect the taste of drinking-water at levels above about 200 mg/litre.

REFERENCES

1. Stockinger HE. The metals. In: Clayton GD, Clayton FE. *Patty's industrial hygiene and toxicology*. Vol. 2A. Toxicology. New York, NY, John Wiley, 1981:2056-2057.
2. Sax NI. *Dangerous properties of industrial materials*, 4th ed. New York, NY, Van Nostrand Reinhold, 1975:1101.
3. Sax NI, Lewis RJ, eds. *Hawley's condensed chemical dictionary*, 11th ed. New York, NY, Van Nostrand Reinhold, 1987:1050-1051.
4. Sittig M. *Handbook of toxic and hazardous chemicals and carcinogens*, 2nd ed. Park Ridge, NJ, Noyes Publications, 1981:792-793.
5. Budavari S, O'Neil M, Smith A, eds. *The Merck index*, 11th ed. Rahway, NJ, Merck, 1989:1355.
6. *Sodium, chlorides and conductivity in drinking water*. Copenhagen, WHO Regional Office for Europe, 1979 (EURO Reports and Studies No. 2).
7. National Academy of Sciences. *Drinking water and health*, Vol. 3. Washington, DC, National Academy Press, 1980:283-293.
8. Andraea MO, Asmode J, Van't Dack L. Determination of sodium in natural waters by atomic absorption spectrometry with hydride generation. *Analytical chemistry*, 1981, 53:1766-1771.
9. National Academy of Sciences. *Drinking water and health*. Washington, DC, National Academy Press, 1977:400-411.
10. Diem K, Lentner C, eds. *Documenta Geigy. Scientific tables*, 7th ed. Basel, Ciba-Geigy, 1970:688.
11. Guthrie HA. *Introductory nutrition*, 7th ed. St. Louis, MO, Times Mirror/Mosby College Publishing, 1989.
12. Department of National Health and Welfare (Canada). *Guidelines for Canadian drinking water quality. Supporting documentation*. Ottawa, 1992.
13. National Institute of Occupational Safety and Health. *Registry of Toxic Effects of Chemical Substances (RTECS)*. Washington, DC, US Department of Health and Human Services, 1991 (NIOSH Publication 91-101-2).
14. Dahl LK. Possible role of salt intake in the development of essential hypertension. In: Cottier P, Bock KD, eds. *Essential hypertension: an international symposium*. Heidelberg, Springer-Verlag, 1960:53-65.
15. Karr-Dullien V, Bloomquist E. The influence of prenatal salt on the development of hypertension by spontaneously hypertensive rats (SHR) (40462). *Proceedings of the Society for Experimental Biology and Medicine*, 1979, 160:421-425.
16. Fregly MJ. Sodium and potassium. *Annual review of nutrition*, 1981, 1:69-93.
17. Takahashi M et al. Effect of high salt diet on rat gastric carcinogenesis induced by N-methyl-N'-nitro-N-nitrosoguanidine. *Gann*, 1983, 74:28-34.
18. National Research Council. *Recommended dietary allowances*, 10th ed. Washington, DC, National Academy Press, 1989.
19. Elton NW, Elton WJ, Narzarena JP. Pathology of acute salt poisoning in infants. *American journal of clinical pathology*, 1963, 39:252-264.
20. Luft FC et al. Cardiovascular and humoral responses to extremes of sodium intake in normal black and white men. *Circulation*, 1979, 60:697-706.
21. Laragh JH, Pecker MS. Dietary sodium and essential hypertension: some myths, hopes and truths. *Annals of internal medicine*, 1983, 98:735-743.
22. Kurtz TW, Morris RC Jr. Dietary chloride as a determinant of "sodium-dependent" hypertension. *Science*, 1983, 222:1139-1141.
23. Morgan TO. The effect of potassium and bicarbonate ions on the rise in blood pressure caused by sodium chloride. *Clinical science*, 1982, 63:407s.
24. Tuthill RW, Calabrese EJ. Drinking water sodium and blood pressure in children: a second look. *American journal of public health*, 1981, 71:722-729.
25. Fatula MI. The frequency of arterial hypertension among persons using water with an elevated sodium chloride content. *Soviet medicine*, 1967, 30:134-136.

26. Tuthill RW, Calabrese EJ. The Massachusetts blood pressure study. Part 4. Modest sodium supplementation and blood pressure change in boarding school students. In: *Advances in modern environmental toxicology. Vol. IX. Inorganics in drinking water and cardiovascular disease*. Princeton, NJ, Princeton Scientific Publishing Co., 1985:69.
27. Pomrehn PR et al. Community differences in blood pressure levels and drinking water sodium. *American journal of epidemiology*, 1983, 118:60-71.
28. Armstrong BK et al. Water sodium and blood pressure in rural school children. *Archives of environmental health*, 1982, 37:236-245.