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Drinking Water Advisory: Consumer Acceptability Advice and Health Effects Analysis on Sodium

**Drinking Water Advisory:
Consumer Acceptability Advice and
Health Effects Analysis on Sodium**

Prepared for:

U.S. Environmental Protection Agency
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FOREWORD

The Drinking Water Advisory Program sponsored by the Health and Ecological Criteria Division of the Office of Science and Technology (OST), Office of Water (OW), provides information on the health and organoleptic (taste, odor, etc.) effects of contaminants in drinking water. The Drinking Water Advisory documents are a component of the OW Health Advisory program. Drinking Water Advisories differ from Health Advisories because of their focus on esthetic properties (taste, odor, color) of drinking water. A Drinking Water Advisory is prepared when the adverse contaminants cause adverse taste and odor influences at concentrations lower than those for possible health effects.

A Drinking Water Advisory is not an enforceable standard for action. However, it describes nonregulatory concentrations of the contaminant in water that are expected to be without adverse effects on both health and esthetics. Both Health Advisories and Drinking Water Advisories serve as informal technical guidance to assist Federal, State and local officials responsible for protecting public health when emergency spills or contamination situations occur. They are not to be construed as legally enforceable Federal standards. They are subject to change as new information becomes available. This draft supersedes any previous draft advisories for this chemical.

The Advisory discusses the limitations of the current database for estimating a risk level for sodium in drinking water and characterizes the hazards associated with exposure. The document was peer reviewed internally and externally by experts in the field. External peer reviewers were as follows:

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LIST OF ABBREVIATIONS

g	gram
Hg	mercury
kg	kilogram
L	liter
mg	milligram
mm	millimeter
mM	millimolar
mEq	milliequivalents
mmol	millimole
NAS	National Academy of Sciences
Na^+	sodium ion
ppm	parts per million

DRINKING WATER ADVISORY:
CONSUMER ACCEPTABILITY ADVICE AND
HEALTH EFFECTS ANALYSIS ON SODIUM

Executive Summary

The EPA Office of Water is issuing this Drinking Water Advisory to provide guidance to communities that may be exposed to drinking water containing sodium chloride or other sodium salts. The Advisory provides a summary of the current health hazard information and an evaluation of available data on taste problems associated with sodium in drinking water. This Advisory does not recommend a reference dose because data for quantifying risk are limited. The Advisory provides guidance on concentrations at which problems with taste would likely occur.

EPA requires periodic monitoring of sodium at the entry point to the distribution system. Monitoring is to be conducted annually for surface water systems and every 3 years for groundwater systems (40 CFR:141.41). The water supplier must report sodium test results to local and State public health officials by direct mail within 3 months of the analysis, unless this responsibility is assumed by the State. This provides the public health community with information on sodium levels in drinking water.

Conclusion and Recommendation

This Advisory recommends reducing sodium concentrations in drinking water to between 30 and 60 mg/L based on esthetic effects (i.e., taste). This recommendation is not federally enforceable but is intended as a guideline for States. States may establish higher or lower levels depending on local conditions, such as unavailability of alternate source waters or other compelling factors, provided that public health and welfare are not adversely affected. A goal of 2.4 g/day dietary sodium has been proposed by several government and health agencies. Drinking water containing between 30 and 60 mg/L is unlikely to be perceived as salty by most individuals and would contribute only 2.5% to 5% of the dietary goal if tap water consumption is 2 L/day.

At the present time the EPA guidance level for sodium in drinking water is 20 mg/L. This value was developed for those individuals restricted to a total sodium intake of 500 mg/day and should not be extrapolated to the entire population

Sodium in the Environment

Sodium is the sixth most abundant element on Earth and is widely distributed in soils, plants, water, and foods. Most of the world has significant deposits of sodium-containing minerals. Sodium ion is ubiquitous in water because of the high solubility of many sodium salts. Groundwater typically contains higher concentrations of minerals and salts than do surface waters. Sodium is present in road deicing chemicals, in water treatment chemicals, in domestic water softeners, and in sewage effluents. These uses contribute significant quantities of sodium to water.

Sodium is a normal component of the body, and adequate levels of sodium are required for good health. Food is the main source of daily human exposure to sodium, primarily in the form of sodium chloride (salt). Most of the sodium in our diet is added to food during processing and preparation. Various studies have reported that dietary intakes of sodium range from 1,800 to 5,000 milligrams per day (mg/day), depending on methods of assessment and on whether discretionary sodium use is assessed. Discretionary sodium intake is variable and can be quite large. The Food and Drug Administration has found that most American adults tend to consume between 4,000 and 6,000 mg of sodium/day, and therapeutic sodium-restricted diets can range from below 1,000 to 3,000 mg/day.

Studies of Sodium Effects

Cancer Studies. Ingestion of sodium ion is not believed to cause cancer. However, some studies suggest that sodium chloride may enhance cancer risk caused by other chemicals in the gastrointestinal tract. Sodium salts have generally produced inconclusive or negative results in *in vitro* or *in vivo* genotoxicity tests.

Noncancer Studies. Very high oral doses of sodium chloride may cause nausea, vomiting, inflammation of the gastrointestinal tract, thirst, muscular twitching, convulsions, and possibly death. For long-term, lower level exposures, the primary health effect of concern is increased blood pressure (hypertension). A large body of evidence suggests that excessive sodium intake contributes to age-related increases in blood pressure leading to hypertension. Increased blood pressure has also been clearly demonstrated in several animal species given high concentrations of sodium chloride in their diets.

High doses of sodium chloride (about 1,570 mg sodium/kg body weight) have been observed to cause reproductive effects in various strains of pregnant rats. Effects on the dams have included decreases in pregnancy rates and maternal body weight gain. Developmental effects have included increased blood pressure and high mortality. However, these effects were observed only in SHR rat pups (a type of rat specifically bred to be hypertensive) fed high sodium diets for up to 4 months after parturition. This study reported no developmental effects in Sprague-Dawley or WKY rat pups (both normotensive strains). Developmental effects have not been studied in other species.

Studies on Taste and Odor. Several studies are available on the taste threshold of sodium chloride in drinking water. It is not possible to identify precise threshold values for the taste of sodium in drinking water because detectable concentrations vary among individuals and for the same individuals at different times. Age and health status also impact a person's ability to detect the taste of sodium. Other factors affecting taste of sodium in drinking water include possible masking by other dissolved substances, water temperature, and the anion forming the salt. The average taste threshold for sodium in water at room temperature differs substantially among individuals and ranges from about 30 mg/L to 460 mg/L. Sodium in water does not by itself cause odor problems. The World Health Organization has established a drinking water guideline of 200 mg of sodium/L on the basis of esthetic considerations (i.e., taste). When sodium chloride is dissolved in distilled water, it is possible to detect the overall impact on taste prior to recognition of the taste as salty.

Characterization Summary. Although numerous human studies have examined the relationship between sodium intake and blood pressure, these studies are not suitable for defining a quantitative dose-response relationship because (1) the dose-response relationships varied among the different studies, (2) sodium intake measurements were generally indirect (determined by the amount of sodium excreted in the urine), and (3) the results may have been influenced by nutrients in the diet other than sodium, by lifestyle, and by patterns of behavior.

Drinking water does not play a significant role in sodium exposure for most individuals. Those that are under treatment for sodium-sensitive hypertension should consult with their health care provider regarding sodium levels in their drinking water supply and the advisability of using an alternative water source or point-of-use treatment to reduce the sodium. For individuals on a very low sodium diet (500 mg/day), EPA recommends that drinking-water sodium not exceed 20 mg/L. In order to avoid adverse effects on taste, EPA recommends that sodium concentrations in drinking water not exceed 30 to 60 mg/L, a threshold for taste-sensitive segments of the population. Many individuals will not be able to detect the presence of sodium in this concentration range.

EPA requires periodic monitoring of sodium at the entry point to the distribution system. Monitoring is to be conducted annually for surface water systems and every 3 years for groundwater systems (40CFR:141.41). The water supplier must report sodium test results to local and State public health officials by direct mail within 3 months of the analysis, unless this responsibility is assumed by the State. This provides the public health community with information on sodium levels in drinking water.

1.0   INTRODUCTION

The purpose of this Advisory is to provide information to States, local drinking water facilities, and public health personnel on the potential health and esthetic effects resulting from ingestion of sodium-containing potable water, as well as on the concentrations of sodium that are typically found in water.

2.0   SODIUM IN THE ENVIRONMENT

Sodium is a common element in the environment and occurs widely in soils, plants, water, and foods.   Sodium chloride is the most economically and industrially important form of sodium, with an estimated 14,000 direct and indirect uses (Kostick 1993). Sodium chloride use can be broken down into eight major categories: chemical (47%), ice control (25%), food processing (5%), general industrial (5%), agricultural (5%), distributors (5%), water treatment (4%), and miscellaneous (4%). Other sodium salts are used in personal care products, foods, nutritional supplements, and medications.

2.1   Air

Sodium salts are nonvolatile, and sodium does not occur in air except in association with suspended particulate matter or water droplets. Because sodium is nonvolatile, the concentrations of sodium salts in air are usually low, especially in comparison to the concentrations of sodium typically found in water or soil. Ambient air concentrations in coastal areas may be higher than for inland areas because of ocean spray droplets introduced into the atmosphere.

2.2   Soil

Sodium is the sixth most abundant element on Earth, making up about 2.6% of the Earth's crust. Sodium concentrations in soil and other surficial materials of the conterminous United States range widely, from less than 500 parts per million (ppm or mg/kg) to more than 100,000 ppm (Shacklette and Boerngen 1984). Sodium is also transported from the ocean to the atmosphere by spray and is suspended in water droplets until it is either precipitated or introduced to the soil by dry deposition (Fairbridge 1972). The application of fertilizers and other agricultural products that contain sodium salts can increase the sodium in soils.

2.3   Water

Sodium ion is ubiquitous in water, owing to the high solubility of its salts and the abundance of sodium-containing mineral deposits. Seawater contains about 30,000 mg of sodium chloride per liter (mg/L). Sodium chloride can also be found in many rivers and inland lakes and seas, in concentrations varying from 20 mg/L in the Mississippi River to 120,000 mg/L in the Great Salt Lake (Chemistry Explorer 2000). Groundwater typically contains higher concentrations of minerals and salts than surface waters, especially in areas with an abundance of sodium mineral deposits or in areas with sea or estuarine water intrusions (WHO 1979).

There are a number of anthropogenic sources of sodium that can contribute significant quantities of sodium to surface water, including road salt, water treatment chemicals, domestic water softeners, and sewage effluents. Water treatment chemicals such as sodium fluoride, sodium silicofluoride, sodium hydroxide, sodium carbonate, sodium bicarbonate, sodium phosphate, sodium silicate, and sodium hypochlorite provide a relatively small contribution when used individually, but when used together may result in concentrations of up to 30 mg/L (WHO 1979).

The addition of sodium compounds during water treatment for adjustment of pH and water softening are the uses most likely to increase the sodium content of drinking water. Sodium hydroxide, sodium carbonate, and sodium bicarbonate are used for pH adjustment and can contribute from 27 to 57 mg/L sodium to water at their approved maximum use levels (NSF 1997). Domestic water softeners can increase sodium levels to more than 300 mg/L in drinking water (NAS 1977).

Salt that has been used to deice roads can also be a problem for drinking water systems. Salt mixed with ice dissolves and creates a brine with a lower freezing point than water, effectively melting ice (Kostick 1993). Salt is a cheap and effective solution to ice-covered roads, but can become an environmental concern as runoff that affects local vegetation and soil quality, as well as groundwater and surface water supplies.

The National Inorganic and Radionuclide Survey (NIRS) collected national occurrence data on selected radionuclides and inorganic chemicals in drinking water. The NIRS investigated 989 community public water supplies (PWSs) served by groundwater (Cadmus 2001). The PWSs selected were statistically representative of national occurrence. Almost 100% of the PWSs were found to have detectable levels of sodium: 37% had sodium levels greater than 30 mg/L and served approximately 28 million people; 13% had sodium levels greater than 120 mg/L and served approximately 7.1 million people.   The median concentration for all samples was 16.4 mg/L and the 99th percentile was 517 mg/L. The 99th percentile concentration is a summary statistic to indicate the upper bound of occurrence values because maximum values can be extreme values (outliers) that sometimes result from sampling or reporting error.

One limitation of the NIRS study is a lack of occurrence data for surface water systems. To better understand the occurrence of sodium in surface water, occurrence data from Safe Drinking Water Act (SDWA) compliance monitoring were reviewed from States with both surface and groundwater systems (Cadmus 2001). Only Alabama, California, Illinois, New Jersey, and Oregon had occurrence data for sodium. The data represent analytical samples from more than 5,500 PWSs. Sodium was detected in 99.3% to 100% of groundwater PWSs and in 100% of surface water PWSs. The median and 99th percentile concentrations (for both groundwater and surface water PWSs) ranged from 5.26 to 31 mg/L and from 150 to 370 mg/L, respectively. For the five States, the percentage of PWSs with sodium detected in surface water at concentrations greater than 30 or 120 mg/L was generally lower than for groundwater systems.

2.4   Food

Foods and beverages are the largest sources of sodium intake for humans. Of the sodium present in foods, a relatively low amount (10%) occurs naturally (Sanchez-Castillo et al. 1987a,b). The majority of dietary sodium comes from sodium chloride added to food during food processing and preparation. For example, Sanchez-Castillo et al. (1987a,b) estimated that 15% of dietary sodium comes from salt added during cooking and at the table, and 75% comes from salt added during processing and manufacturing. The first National Health and Nutrition Examination Survey (Abraham and Carroll 1981) reported that approximately 32% of the sodium chloride consumed came from baked goods and cereals, 21% came from meats, and 14% from dairy products. Similar results were reported for the FDA Total Diet Study (Pennington et al. 1984). Using data from the 1989 to 1991 Continuing Survey of Food Intake by Individuals, Subar et al. (1998) found that 23.4% of the salt in the diet came from a group of foods that included cold cuts and other processed meats, condiments, snack-type foods (e.g., chips and popcorn), and table salt; 10.9% from yeast bread; 5.6% from cheese; and 4.1% from ham. These foods contributed 44.1% of the total dietary sodium.

Reported dietary sodium intakes range from 1,800 mg/day to 5,000 mg/day in various studies, depending on the methods of assessment and whether discretionary sodium use is assessed (Abraham and Carroll 1981, Pennington et al. 1984, Karanja et al. 1999). Discretionary sodium intake is highly variable and can be quite large. In a 28-day study, Mickelson et al. (1977) found that males added an average of about 5,500 mg of sodium chloride (2,200 mg of sodium) to their food per day. The Food and Drug Administration has found that most American adults tend to consume between 4,000 and 6,000 mg of sodium per day, whereas individuals on sodium-restricted diets usually ingest less than 1,000 to 3,000 mg/day (Kurtzweil 1995).

3.0   CHEMICAL AND PHYSICAL PROPERTIES

Sodium (Na) is the most abundant element of the alkali metal group. Elemental sodium has an atomic weight of 22.99 and is a soft, bright silvery metal. Pure metallic sodium is highly reactive and burns in air to form sodium oxide, which in turn readily hydrolyzes in water to form sodium hydroxide. Because elemental sodium is so highly reactive, it is not found freely in nature. Rather, sodium is found in nature only as the sodium ion (Na^+) combined with a variety of anions to form a number of different salts. Common sodium salts are chloride, carbonate, hypochlorite, and silicate. The physical and chemical properties of five sodium salts are presented in Table 3–1 (Sax 1975, Clayton and Clayton 1981, Sittig 1981, Sax and Lewis 1987, Budavari 1996, HSDB 2000).

4.0   TOXICOKINETICS

Sodium ions are a normal and essential component of the human body, playing a key role in controlling and maintaining the proper osmolarity (concentration) and volume of extracellular body fluids. Both the body content of sodium and its concentration in body fluids are under homeostatic control. In addition to its role in regulating osmolarity and extracellular fluid volume, sodium is important in the regulation of acid-base balance and the membrane potential of cells. As a consequence of these vital functions, the absorption, distribution, and excretion of

Table 3-1. Physical and Chemical Properties of Sodium and Sodium Salts

Chemical Name	Sodium	Sodium Chloride	Sodium Carbonate	Sodium Hypochlorite	Sodium Silicate	Sodium Sulfate
CAS Number	7440-23-5	7647-14-5	497-19-8	7681-52-9	1344-09-8	7757-82-6
Chemical Formula	Na	NaCl	Na_2CO_3	NaOCl	Na_2SiO_3	Na_2SO_4
Molecular Weight	22.99	58.44	105.99	74.44	99.07	142.06
Physical State	Silver metal	Colorless cubic crystal or white crystalline powder	White powder	In solution only	Colorless crystal	White powder or orthorhombic bipyramidal crystals
Boiling Point (°C)	881.4	1,413	Decomposes	—	—	—
Melting Point (°C)	97.83	801	851	—	—	888
Density (g/mL) (20°C)	0.71	2.17	2.53	—	—	2.67
Vapor Pressure (mm Hg)	1	—	—	—	—	—
Specific Gravity	0.71	2.17	2.53	—	—	2.67
Water Solubility (g/100 mL)	React violently	35.7	7.1	Infinitely soluble	Slightly soluble or almost insoluble in cold water.	Soluble in about 3.6 parts water. Max. solubility at 33°: 1 in 2
Taste Threshold (Water) (mg/L)	—	30 - 460 See Section 6.0	—	—	—	180 - 550

sodium ion has been extensively studied in both animals and humans. A brief summary of the most important aspects of sodium toxicokinetics is presented below.

4.1 Absorption

Virtually all (~99%) of the sodium ion ingested in food and water is rapidly absorbed from the gastrointestinal tract (Stipanuk 2000). Sodium crosses the brush border epithelial membrane of the intestine through sodium channels or by carried-mediated diffusion down an electrochemical gradient. During facilitated transport, sodium can carry chloride ion, glucose, amino acids, and other nutrients into the intestinal epithelial cells. Once sodium is in the cytosol of the brush border epithelial cell, it is actively transported into the blood by the Na^+/K^+-ATPase pump located in the basal and lateral membrane of the epithelial cell (Stipanuk 2000).

4.2 Distribution

Once absorbed, the sodium ion is rapidly distributed throughout the body. The concentration of sodium in blood and other extracellular fluids is about 145 mM (3,335 mg/L), whereas the concentration of sodium ion inside cells is about 12 mM (276 mg/L) (Stipanuk 2000). This unequal distribution of sodium between extracellular and intracellular compartments is essential to the normal functioning of all cells and tissues of the body.

4.3 Metabolism

Sodium ion is not reactive and does not undergo any metabolic reactions in the traditional sense (i.e., it is not transformed by enzymic or nonenzymic mechanisms into any altered forms). Sodium does function as a counterion for macromolecules such as DNA, RNAs, proteins, and sulfated polysaccharides that carry a net negative change, and thus, concentrations can be enriched in the microenviorinment surrounding macroion surfaces (Stipanuk 2000).

4.4 Excretion

Sodium is excreted mainly in the urine, although some sodium loss occurs with fecal matter and in perspiration. The kidney, nervous system, and endocrine system maintain very precise control of renal sodium excretion, with approximately 95% to 98% of the sodium being reabsorbed in the kidney (Stipanuk 2000). In the proximal tubule of the kidney, sodium resorption is coupled with organic solutes and anions and protons. Entry into the proximal tubule epithelium is mediated by symporter (e.g., Na^+-glucose, Na^+-PO_4^{-3}, Na^+-lactate, and Na^+-amino acid symporters) and antiporter (Na^+-H^+ antiporter) proteins located on the apical membrane of the proximal tubule. When sodium enters the cytoplasm of the proximal tubule, it is actively transported into the blood by the Na^+/K^+-ATPase pump. Similar sodium resorption mechanisms occur in the loop of Henle and distal tubule.

In response to blood volume depletion (i.e., decreased blood pressure), the sympathetic nervous system stimulates sodium resorption. Hormonal control of sodium resorption is dependent on renal blood flow and nervous system stimulation. Decreased renal pressure in the renal arterioles, as well as sympathetic nervous system stimulation, results in the kidney's production

of renin. Renin cleaves circulating angiotensinogen to form angiotensin I, which is converted to angiotensin II by angiotensin-converting enzyme (ACE), an enzyme that is widely distributed in the body. Angiotensin II stimulates the adrenal gland to synthesize aldosterone, which binds to receptors in the cytoplasm of principal cells located in the collecting tubules of the kidney, and stimulates activity of the apical sodium channel and basal Na^+/K^+-ATPase pump. In response to increased blood and renal pressure, sympathetic nervous system stimulation and aldosterone synthesis decrease and sodium excretion increases. Of the approximately 25,200 mEq of sodium filtered through the kidneys each day, 150 mEq is excreted (Berne and Levy 1993, Stipanuk 2000).

5.0   HEALTH EFFECTS DATA

5.1   Humans

Sodium is an essential nutrient and is needed to maintain body fluid volume and blood pressure. The estimated minimum daily requirement for healthy adults and children 10 years and older is 500 mg/day (NRC 1989a). At birth, the estimated minimum requirement ranges from 100 to 200 mg/day and increases to 225 mg/day at 1 year of age. The minimum requirement increases throughout childhood to 400 mg/day at 9 years of age. Pregnancy and lactation increase the minimum requirement by 69 and 135 mg/day, respectively (NRC 1989a). No optimal level of sodium intake has been established (NRC 1989a). The kidneys have considerable flexibility in removing excess sodium and can accommodate intakes greater than the minimum requirements.

Because sodium is a common constituent of food and water, diseases of sodium deficiency in humans are very rare. However, excess sodium intake can cause acute and long-term health effects, as described in the following sections. The Dietary Guidelines for Americans (USDA 2000) recommend 2.4 g/day as an achievable and reasonable goal that will minimize the risk for sodium-linked hypertension and one that is supported by other recommendations on dietary sodium intake (AHA 2000, NIH 1993, NRC 1989a).

5.1.1   Short-Term Exposure Studies

In general, sodium salts are not acutely toxic because of the efficiency with which mature kidneys excrete sodium. However, acute toxicity and death have been reported in cases of very high sodium intake.

Adults

Acute effects and death have been reported in cases of accidental overdoses of sodium chloride (WHO 1979). Acute effects may include dryness of mucous membranes, violent inflammatory reaction and ulceration in the gastrointestinal tract, along with dehydration and congestion of internal organs, particularly the meninges and brain. Central nervous system disturbances such as convulsions, confusion, and coma may result, and generalized and pulmonary edema are possible. Death may occur from respiratory failure secondary to an acute encephalopathy (MSDS 2000). Two cases are reported by WHO (1979) of individuals who sought medical

attention because of symptoms experienced after using drinking water sources containing greater than 3 g Na/L.

Children

Infants and children are somewhat more susceptible than adults to the effects of acute overdoses of sodium chloride because the kidneys of immature individuals are not as effective in controlling sodium levels as the kidneys of adults (Sax 1975). The accidental administration of infant formula containing high sodium chloride concentrations (dose not reported) resulted in the deaths of 6 of 14 infants (Elton et al. 1963). Kidney lesions, characterized by the shrinkage of tubular complexes in the convoluted kidney tubules, and brain thromboses were noted at autopsy. Gauthier et al. (1969) found that four of five newborn infants who received sodium at concentrations of 2,000 to 2,500 mEq/L (46,000–57,500 mg/L) instead of sugar in formula developed hypernatremia (high blood sodium concentrations) and died. In one reported case (DeGenaro and Nyhan 1971), a 2-year-old boy died 9 days after being given a dose of about 9,200 mg Na/kg as sodium chloride to induce vomiting. Death occurred despite medical intervention. The child was hospitalized within 2 hours of being given the salt solution. He was in a coma. His temperature was elevated, his breathing rapid and heartbeat slow; he was cyanotic and suffering from seizures.

5.1.2   Long-Term Exposure Studies

Adults

A large body of evidence suggests that excessive sodium intake contributes to age-related increases in blood pressure and may contribute to essential hypertension (AHA 2000, NIH 1993, NRC 1989a, USDA 2000). Estimates based on the 1988–1991 National Health and Nutrition Examination Survey (NHANES III) indicate that approximately 50 million adults have high blood pressure (i.e., systolic pressure $\geq$140 mm Hg or diastolic pressure $\geq$90 mm Hg), with the prevalence of high blood pressure increasing with age (NIH 1993). High blood pressure is associated with an increased risk of developing coronary heart disease, stroke, congestive heart failure, renal insufficiency, and peripheral vascular diseases.   However, it must be understood that high blood pressure is a multifactorial disorder, with dietary sodium as one of a number of factors influencing its incidence.

The Intersalt Cooperative Research Group (ICRG) performed a study (generally referred to as the Intersalt study) that suggests blood pressure rises with increasing sodium consumption (ICRG 1988, Elliott et al. 1989). The Intersalt study was a cross-sectional study of the relationship between urinary sodium excretion (as a measure of sodium intake) and blood pressure, involving 10,079 subjects from 52 population centers in 32 countries. The study included approximately equal numbers of men and women ages 20 to 59 years. Estimated regression coefficients of blood pressure change and 24-hour sodium excretion, calculated after adjusting for possible confounders such as age, sex, body mass index, alcohol consumption, and potassium excretion, indicated an increase in systolic pressure of 2.2 mm Hg for every 100 mmol (2,300 mg) increase in sodium intake. The authors indicated that the positive relationship between blood pressure and sodium intake may have been underestimated because some of the subjects included in the

study were (1) following public health campaigns against a high salt intake, (2) taking antihypertensive medications, or (3) collecting inadequate urine samples for sodium intake measurements (Elliott et al. 1989).

Stamler (1991) evaluated the impacts of changes in sodium intake on mortality due to coronary artery disease, stroke, and other diseases. The author estimated that a nationwide reduction of 2.2 mm Hg in average systolic blood pressure would result in a 4% reduction in coronary disease mortality, a 6% reduction in stroke mortality, and a 3% overall reduction in other deaths. This drop in total mortality would result in 12,000 fewer deaths each year in Americans aged 45 to 64. Decreases in sodium intake from average levels to 100 mmol/day (2,300 mg/day) throughout the lifespan would correspond to a reduction of 9 mm Hg in the expected increase in systolic blood pressure from age 25 to 55. This would translate into a mortality rate reduction of 16% for coronary heart disease, 23% for stroke, and 13% for death from all causes. The author concluded that sodium remains the key risk factor for essential hypertension when compared with other risk factors such as body mass index and alcohol consumption.

Frost et al. (1991) performed a meta-analysis of 14 published studies from the United States, Europe, and Asia that measured blood pressure and sodium intake estimated by 24-hour urinary sodium excretion in 12,773 subjects. The analysis indicated that there is a highly significant (p<0.001) positive association between blood pressure and sodium intake within populations. Elliott (1991) performed a similar meta-analysis of 14 observational studies in 16 populations relating 24-hour urinary sodium excretion and blood pressures. This analysis also indicated positive and significant correlations with both systolic and diastolic blood pressure in males and females. For men and women combined (12,503 subjects), the regression coefficient (corrected for reliability) indicated systolic and diastolic blood pressures were lowered by about 3.7 and 2.0 mm Hg, respectively, per 100 mmol (2,300 mg) reduction in 24-hour urinary sodium excretion (p<0.001). This analysis did not include Intersalt data.

Sullivan (1991) analyzed data on 183 subjects to determine sodium sensitivity (increase of mean blood pressure of more than 5% when progressing from low- to high-sodium intake). Using this criterion, sodium sensitivity was detected in 15% of white normotensive subjects, 29% of white borderline hypertensive subjects, 27% of normotensive black subjects, and 50% of black borderline hypertensive subjects. Long-term followup of sodium-sensitive and sodium-resistant individuals with similar blood pressures indicated that a daily sodium intake of about 150 mEq (3,450 mg) resulted in significantly higher blood pressure and forearm vascular resistance in the sodium-sensitive group. Sodium intake may also influence the heart muscle thickness as a secondary response to blood pressure effect. Dietary salt intake, as determined by 24-hour urinary sodium excretion, was significantly correlated (p<0.001) to left ventricular hypertrophy (wall thickness) in a series of 42 hypertensive individuals (Schmieder et al. 1988).

A randomized clinical trial of 2,382 men and women (30 to 54 years of age) with high normal blood pressure (diastolic 83 to 89 mm Hg and systolic below 140 mm Hg) and elevated body weight (110% to 165% of the recommended value) was conducted at nine academic medical centers over a 3-year period (Trials of Hypertension Prevention Collaborative Research Group 1997). Subjects were divided into four groups. One group was treated for weight reduction, the second for reduced sodium intake (1.8 g/day or less), and the third for combined weight

reduction and reduced sodium intake. The last group received usual care and served as the control. Both weight loss and sodium restriction alone or in combination were associated with a decrease in blood pressure at the end of 6 months and at 3 years. Weight loss alone had a greater impact on blood pressure when compared to the usual control group than did sodium restriction at six months (3.7/2.7 mm Hg vs. 2.9/1.6 mm hg). The greatest reduction in blood pressure was observed in the group that combined weight loss with sodium restriction (4.0/2.8 mm Hg). After 3 years, the treatment groups still had lower blood pressures than the usual care group, but the reductions in blood pressure were rather comparable in the weight reduction (1.3/0.9 mm Hg), sodium restriction (1.2/0.7 mm Hg), and combination groups (1.1/0.6 mm Hg). The authors felt that this was a reflection of the difficulties involved with long-term behavior modification to sustain weight loss and low-sodium intakes.

Numerous investigations have analyzed the reduction in blood pressure following a reduction in sodium intake, both in hypertensive and normotensive individuals. A meta-analysis of 56 trials (28 with 1,131 hypertensive subjects and 28 with 2,374 normotensive subjects) found a significant reduction in systolic blood pressure of 3.7 mm Hg ($p<0.001$) in the hypertensives and 1.0 mm Hg ($p<0.001$) for normotensives for a 100 mmol per day (2,300 mg/day) reduction in daily sodium excretion (Midgley et al. 1996). These findings were supported by other studies (Graudal et al. 1998, Cutler et al. 1991). However, other clinical studies have not detected convincing evidence of a protective effect of low sodium intake on the risk of cardiovascular disease (Muntzel and Drueke 1992, Salt Institute 2000, NIH 1993, Callaway 1994, Kotchen and McCarron 1998, McCarron 1998). Even though the experts at the National Heart, Lung and Blood Institute support the policy of universal salt reduction for decreasing the risk for essential hypertension, the scientific experts at the AHA, American Society of Hypertension, and the European and International Societies of Hypertension disagree with the universal salt reduction hypothesis (Taubes 1998).

Dietary studies are difficult to analyze because changing the concentration of one nutrient in the diet changes the balance of all the other nutrients as well. It becomes difficult to determine if the observed effect is the result of the decrease in the target nutrient or the change in the balance of all nutrients. In the mid-1990s, the National Heart, Lung and Blood Institute sponsored a study of hypertension and diet called the Dietary Approaches to Stop Hypertension (DASH) trial. The subjects were 459 adults classified as hypertensives who did not use antihypertensive medications during the trial. The subjects were divided into three groups that received either the control diet, a diet high in fruits and vegetables, or a combination diet that was still rich in fruits and vegetables but had higher amounts of low-fat animal protein and grains than the high fruit and vegetable diet (Vogt et al. 1999). The subjects consumed their respective diets as prepared by the study kitchen for an 8-week period. The sodium content of all three diets was the same (3 g/day) and greater than the present dietary guideline for sodium (2.4 g/day). The high fruit and vegetable and combination diets were 2 to 3 times higher in potassium, calcium, magnesium, and fiber than the control diet. The combination diet reduced average blood pressures by 5.5 mm Hg (systolic) and 3 mm Hg (diastolic) compared with the control diet. The high fruit and vegetable diet reduced blood pressures by 2.8 mm Hg (systolic) and 1.1 mm Hg (diastolic) compared with the control diet. These reductions are similar to those obtained in many of the sodium restriction diets.

In a followup to the original DASH study, the effects of different levels of dietary sodium in conjunction with the combination DASH diet were evaluated (Sacks et al. 2001, Svetkey et al. 1999). All subjects had higher than optimal blood pressure. Systolic blood pressures exceeded 120 mm Hg but were not higher than 159 mm Hg (the cutoff for stage 1 hypertension). Diastolic blood pressures were higher than 80 mm Hg, but not higher than 95 mmHg. After an initial adjustment period on a high-sodium (150 mmol/day) control diet, the 412 subjects were randomly assigned to either the combination DASH diet or a control diet. Diets for both groups had three sodium levels (low, 50 mmol/day; intermediate, 100 mmol/day; or high, 150 mmol/day), which were administered for 30 consecutive days in random order in a crossover design. Meals were supplied to the participants for the duration of the study.

For those in the control group, reducing sodium intake from the high to the intermediate level lowered systolic blood pressure by 2.1 mm Hg; reducing sodium intake from the intermediate to the low level lowered systolic blood pressure by an additional 4.6 mm Hg (Sacks et al. 2001). Subjects on the DASH diet had a 1.3 and 1.7 mm Hg reduction in systolic blood pressure when sodium intake was reduced from high to intermediate level and intermediate to low level, respectively. At the high, intermediate, and low sodium intake levels, systolic blood pressure was reduced by 5.9, 5.0, and 2.2 mm Hg, respectively, in subjects on the DASH diet compared with subjects on the control diet. The effect on systolic blood pressure was greater in hypertensive subjects (systolic 140–159 mm Hg; diastolic 90–95 mm Hg) compared with nominal normotensives (systolic 120–140 mm Hg; diastolic 80–90 mm Hg). The average reduction in blood pressure achieved from sodium restriction was greater for those on the control diet than for those on the DASH diet, for African-Americans on the control diet than for other participants, and for women on the DASH diet than for men on the DASH diet.

Decreases in diastolic blood pressure were correlated with reduction in sodium levels in both the DASH and control diets. Diastolic pressure was lower at all sodium levels in subjects on the DASH diet compared with subjects on the control diet. The magnitude of reduction in diastolic blood pressure was not as great as that reported for systolic blood pressure. The study authors concluded that reducing sodium intake to below the current recommendation of 100 mmol/day and the DASH diet both lower blood pressure substantially, with a greater reduction in blood pressure occurring when the low sodium and DASH diet are combined. They acknowledged the limitation of the 30-day experimental period. Compliance with the low-salt dietary plan might decrease with time because a large portion of the sodium in the American diet comes from processed foods rather than from home use of table salt.

One study suggests that low sodium intake may actually increase the risk for cardiovascular disease in adults. Alderman et al. (1995) reported the relationship between morbidity and mortality due to cardiovascular disease in hypertensive subjects and their urinary sodium excretion. The study cohort included 2,937 hypertensive subjects (1,900 men and 1,037 women). The principal finding was that low urinary sodium excretion was associated with high incidences of heart attacks in hypertensive men and hypertensive subjects (men and women combined), but not in hypertensive women. In men, age- and race-adjusted myocardial infarction incidence was 11.5 versus 2.5 in the lowest versus highest urinary sodium excretion groups. The hypertensive subjects with high sodium intakes did not experience high incidences of myocardial infarction when compared with subjects on normal sodium diets. A group of scientific experts who

commented on this study concluded that future research may be needed to clarify this observation, as the study was not a randomized trial and did not address possible confounders such as smoking and alcohol use (AHA 1995).

Children

A number of studies have investigated the relationship between sodium intake from water and blood pressure in children. Most of these studies have not detected an association between sodium in drinking water (at concentrations ranging from 5 to 583 mg/L) and increased blood pressure in children (Pomrehn et al. 1983, Faust 1982, Armstrong et al. 1982, Tuthill et al. 1980, Colditz and Willett 1985), although a few studies do suggest an increase in blood pressure with the high sodium intake (Calabrese and Tuthill 1977, 1981, Tuthill and Calabrese 1979, Fatula 1967).

Summary

Excessive intake of very high doses of sodium (accidental poisoning) may cause acute effects such as nausea, vomiting, inflammatory reaction in the gastrointestinal tract, thirst, muscular twitching, convulsions, and possibly death. For long-term lower level exposures, the health effect of primary concern is essential hypertension. Although evidence of a positive association between sodium intake and blood pressure and essential hypertension is convincing, numerous studies fail to find a protective effect of low sodium intake in controlling blood pressure in hypertensive subjects. Because of the inconsistencies and uncertainties in the data on the relationship between sodium intake and cardiovascular disease, it is not possible to draw definite conclusions on the benefits of reduced sodium intake. Factors such as increased intake of potassium, calcium, and magnesium, reduced caloric intake, reduced chloride intake, moderate physical activity, and lower alcohol consumption may play a significant role in reducing blood pressure and the risk for cardiovascular disease. Sodium restriction seems to be the most beneficial in lowering blood pressure for older persons who are only mildly hypertensive and are not overweight.

5.1.3   Sensitive Populations

Several studies have shown that children are more sensitive than adults to high sodium intake (Elton et al. 1963, Gauthier et al. 1969, DeGenaro and Nyhan 1971). This increased sensitivity is associated with the lower ability of the immature kidney to control sodium levels compared with that of the adult kidney. However, on a mg/kg basis, the sodium requirement for infants and children is greater than that for adults (NRC 1989a).

In addition to children, the elderly may be more sensitive to adverse health effects resulting from high sodium exposure. This is because the elderly have a higher incidence of cardiovascular disease (including high blood pressure) than do younger subjects (Sowers and Lester 2000). Therefore, the hypertensive effects of sodium may be more severe in the elderly. In addition, because the elderly tend to have a higher taste threshold for salt (Hyde and Feller 1981, Stevens 1996), they may have a higher salt intake. African-Americans are more susceptible to sodium-

induced adverse health effects because of high prevalence of hypertension and increased salt sensitivity among this population (Sullivan 1991, Svetkey et al. 1996, 1999).

Individuals with decreased renal function or renal insufficiency are more sensitive to high sodium intake than are individuals with healthy kidneys. Sodium chloride at 200 mmol/day significantly elevated systolic blood pressure in humans with chronic renal failure (Muntzel and Drueke 1992). In addition, renal tubule defects or alterations in kidney hemodynamics have been postulated to predispose salt-sensitive individuals to retain sodium. Sodium retention has been reported in rats given high doses of sodium chloride following partial nephrectomy. Dietary sodium restrictions are recommended for individuals with acute or chronic renal problems and those with nephritic syndrome (Whitney et al. 1987). Renal problems are associated with about 10% of the hypertension in the population.

5.2   Animal Studies

5.2.1   Short-Term Exposure Studies

Sodium ion (ingested as sodium chloride) has low acute toxicity in animals. Doses that cause lethality in animals range from around 3,000 to 8,000 mg/kg (HSDB 2000, RTECS 2002). Death has been attributed to severe alterations in fluid concentrations and/or fluid volumes (HSDB 2000).

5.2.2   Long-Term Exposure Studies

Hypertension has been clearly demonstrated in several species of animals given high concentrations of sodium chloride in their diets (WHO 1979). Dahl (1967) exposed 21-day-old female Sprague-Dawley rats to a diet containing 8% sodium chloride for 12 to 15 months. This corresponds to a dose of about 1,570 mg/kg sodium ion, based on EPA's reference values of 340 g/bw and a food intake of 17 g/day (assuming a rat consumes 5% of its body weight per day) (U.S. EPA 1988). Within 6 to 9 months, about 75% of the rats exhibited hypertension, and their mean blood pressure increased with age. Rats maintained on the low-salt diet (0.35% sodium chloride, corresponding to a dose of 70 mg/kg sodium ion) did not exhibit a corresponding increase in blood pressure with age.

5.2.3   Reproductive Studies

High doses of sodium chloride (1,570 mg sodium/kg body weight) have been observed to cause reproductive effects in various strains of pregnant rats. Effects on the dams have included decreases in pregnancy rates and maternal body weight gain.

The reproductive effects of sodium ion were studied in three strains of pregnant rats (SHR, WKY, and Sprague-Dawley) ranging in age from 3 months to 1 year (Karr-Dullien and Bloomquist 1979). It should be noted that SHR rats are bred to be hypertensive and serve as a hypertensive rat model. The animals were fed diets containing either 0.4 or 8.0% sodium chloride (corresponding to doses of 79 or 1,570 mg/kg sodium ion, based on EPA's reference values of 340 g bw and 17 g/food/day) throughout gestation and lactation (U.S. EPA 1988).

Pregnancy rates were decreased by 38% in SHR rats and by 66% in WKY rats in the high-salt diet groups compared with those in the low-salt diet groups. The high-salt diet also decreased the maternal body weight gain in SHR and WKY rats. This effect was also noted in SHR rats fed with low-salt diets. No effects were observed in Sprague-Dawley rats.

5.2.4   Developmental Studies

In a continuation to the above-mentioned study, the pups from the low- and high-dose dams were placed on either a 0.4% or 8% sodium chloride diet irrespective of the dams' diets (Karr-Dullien and Bloomquist 1979). This dosing regimen resulted in four dose groups: high-dose pups from high-dose dams (HH), high-dose pups from low-dose dams (HL), low-dose pups from high-dose dams (LH), and low-dose pups from low-dose dams (LL). After 11.5 weeks of exposure, significant increases in systolic blood pressure were noted in SHR HH pups compared with all other pups. This was accompanied by a 63% mortality rate after 4 months of exposure due to peripheral capillary hemorrhage and stroke. No significant changes in blood pressure or mortality were observed in the WKY or Sprague-Dawley pups.

No developmental effects were observed in the offspring of mice administered 189 mg/kg sodium ion or rats administered 147 mg/kg sodium ion on days 6–15 of gestation (Fregly 1981).

5.2.5   Genotoxicity Studies

Sodium (as sodium chloride) produced DNA damage in mammalian assays employing mouse lymphocytes (2.3 g/L), induced unscheduled DNA synthesis in rats (16.8 g/kg), and caused DNA damage in hamster ovaries (6.3 g/L) (RTECS 2002). Tests in *Saccharomyces cerevisia* (46 g/L) and *Escherichia coli* (3.5 g/L) were also positive. However, the overall importance of these findings is questionable because these studies used very high sodium levels that would tend to disrupt the cellular osmotic balance and DNA microenvironment, especially in the *in vitro* studies.

5.2.6   Cancer Studies

Sodium by itself is not believed to cause cancer. However, several studies suggest that sodium chloride may enhance the cancer risk caused by other chemicals (NRC 1989b). For example, the incidences of gastric tumors caused by 4-nitroquinoline-1-oxide and N-methyl-N[1]- nitro-N-nitrosoguanidine were reported to be enhanced by simultaneous sodium chloride administration (10% of diet or in their drinking water) to male Wistar rats (Tatematsu et al. 1975). The authors hypothesized that the promoting effect of the concentrated sodium chloride was a consequence of its ability to disrupt the mucopolysaccharide layer lining the gastric epithelium.

This indirect effect of sodium in enhancing cancer risk might be due to cell death in the gastrointestinal tract and resulting cell regeneration (as measured by ornithine decarboxylase activity and DNA synthesis). For example, a single oral dose of a saturated sodium chloride solution resulted in a 200-fold increase in ornithine decarboxylase activity within 6 hours and a ninefold increase in DNA synthesis within 3 hours in rat stomach mucosa (Furihata et al. 1984). Subsequent studies indicated that sources of sodium other than sodium chloride may also cause

damage to the gastrointestinal tract (Furihata et al. 1989). These sources included the sodium salts of acetic, L-ascorbic, L-glutamic, carbonic, and sorbic acid.

6.0   ORGANOLEPTIC PROPERTIES

Organoleptic properties for contaminants in drinking water refer to odor, color, and taste. Because these characteristics do not cause adverse health effects, they are not used by EPA for developing primary water standards. However, organoleptic properties are used in establishing secondary standards and guidelines.

Several studies are available that report on the organoleptic properties of sodium in drinking water; they focused primarily on taste threshold of sodium chloride in drinking water. None of the studies reported odor thresholds for sodium salts or distinguished between threshold levels and levels that are unpalatable.

The taste threshold concentration of sodium in drinking water depends on the associated anion. For example, Schiffman et al. (1980) reported considerable differences in the overall taste sensation of tested salts due to the associated anions. Taste threshold was tested in 12 students (6 males and 6 females, 18–25 years) using several sodium salts dissolved in deionized water (0.2 M Na^+; 4.6 g Na^+/L). The salts that were perceived to be the most "salty" were sodium chloride and sodium bromide, whereas sodium citrate was perceived to be the least salty. The taste threshold of sodium chloride was dependent on whether sodium chloride was in mixture with other chemicals (e.g., sucrose and/or citric acid). Stevens and Traverzo (1997) reported that the taste threshold for sodium chloride increases three to four times in a mixture with sucrose or citric acid, and more than nine times in a mixture with sucrose and citric acid compared with sodium chloride in water.

Age may also affect the taste threshold. Stevens (1996) reported that the salt threshold is much lower in younger subjects (18–29 years, n=15) than in older subjects (66–90 years, n=15). Subjects were offered 5 mL of distilled water or the salt solution and asked to choose the one with the salty taste. The mean taste threshold values for sodium chloride (as sodium) dissolved in deionized water for younger and older subjects were 1.3 mM (30 mg Na^+/L) and 5.7 mM (131 mg Na^+/L), respectively. The average response for both groups was 2.7 mM (60 mg/L).   Work by Pangborn and Pecore (1982) discussed below suggests that the fact that the participants knew they were to identify a salty taste influenced the results of this study.

Pangborn and Pecore (1982) reported the taste threshold of sodium among 44 females and 13 males (18–22 years old) who were stratified as having high (n=14), medium (n=26), or low dietary intakes of salt (n=17), based on food habit. Solutions of 0.005%-0.12% sodium chloride (20–472 mg Na^+/L) in double-distilled water were tested. The study design was similar to that used by Stevens (1996), except that the directions given to the subjects varied. In one test, the subjects were asked to choose the container that had a taste and then to identify the taste. The mean detection thresholds reported for the sodium chloride in water ranged from 0.015 to 0.031% (60 to 122 mg Na^+/L) and the selection of the container with the salt was correct for 77% to 85% of the trials. The mean taste thresholds for the individual groups are given in Table 6-1.

Table 6-1. Mean Taste Threshold Values in Young Adults with Different Sodium Intake Levels

Sodium Chloride Intake Group[a]	Number of Subjects	Mean Taste Threshold Values[b]	Mean Taste Threshold Values[c]
Low	17	122 mg Na$^+$/L	40 mg Na$^+$/L
Medium	26	87 mg Na$^+$/L	48 mg Na$^+$/L
High	14	60 mg Na$^+$/L	32 mg Na$^+$/L

[a] Denotes the arbitrary assessment of dietary sodium intake through questionnaires. Excerpted from Pangborn and Pecore (1982).
[b] When subjects were not told that the taste to be recognized was salt.
[c] When subjects were told that the taste to be recognized was salt.

Despite the fact that the subjects could correctly identify the container with the salt, they did not initially recognize that taste as salty. For the same group of subjects, the concentration that could be identified as having a salty taste was 2.5 to almost 4 times higher than the concentration that could be tasted. In addition, participants were able to correctly identify the taste as salty only 35% to 49% of the time. These results suggest that taste recognition was greater for the high-salt intake group than for the other groups and that the ability to taste salt and the ability to correctly identify the taste are different.

In a second portion of their study, Pangborn and Pecore (1982) used the same study design but told the subjects to select between distilled water and a solution that contained salt. Under these conditions, the ability to make a correct selection increased to more than 90% and the average taste threshold dropped to a level comparable to that recorded by Stevens (1996). The threshold for the high-salt group was 32 mg/L and those for the low and medium groups were 40 and 48 mg/L, respectively. A hypothesis for the high salt-taste sensitivity in the high-intake group was not provided. Their high salt intake had apparently not acclimatized them to the salt taste.

Weiffenbach et al. (1982) reported the mean taste threshold values for sodium chloride in deionized water as 2.49 mM (57 mg Na$^+$/L), 3.26 mM (75 mg Na$^+$/L), and 6.1 mM (140 mg Na$^+$/L) for subjects ≤45 years, 46–65 years, and >65 years, respectively. The low end of the threshold in this data set is comparable to that in the Pangborn and Pecore (1982) study. The study designs were very similar and it appears that, like Pangborn and Pecore (1982), Weiffenbach et al. (1982) did not reveal the nature of the tastant to the participants. Somewhat higher taste threshold values were reported by Hyde and Feller (1981) for younger adults. The taste threshold for sodium chloride dissolved in deionized water in young adults (mean age 28 years, n=24) and elderly persons (mean age 75 years, n=24) was 10 mM (230 mg Na$^+$/L) and 20 mM (460 mg Na$^+$/L), respectively.

According to WHO (1993), the average taste threshold for sodium (as sodium chloride) at room temperature is about 200 mg/L, a value that was used as the WHO drinking water guideline. However, as illustrated by the data discussed above, the taste threshold may vary substantially among individuals and as a function of other solutes that may be present in the water. For example, Stevens (1996) combined salt with sugar (sweet), citric acid (sour), or quinine

hydrochloride (bitter) in a series of binary mixtures and demonstrated that one tastant influences the ability to detect the second tastant in a mixture.

It is not possible to identify point threshold values for the taste of sodium in drinking water, because the concentration will vary among individuals, for the same individual at different times, and for different water matrices, water temperatures, and many other variables (e.g., age, masking due to other tastants, and the anion forming the salt). However, the data discussed above suggest that 30 to 60 mg/L is a threshold for the ability to taste, but not necessarily to identify, the presence of sodium chloride in water.

7.0   CHARACTERIZATION OF HAZARD AND DOSE-RESPONSE

7.1   Hazard Characterization

Sodium is a physiologically important element needed to maintain normal body fluid volume and blood pressure and normal cell function. The normal sodium level in the blood is 154 mM (3,542 mg/L). Intake from food is generally the major source of sodium, with only a small contribution from drinking water.

The NRC estimated minimum daily requirements for sodium are 120–225 mg for infants (0 months – 1 year), 300–400 mg for children (2–9 years), and 500 mg for individuals 10 years old and older. Requirements increase during pregnancy and lactation. Minimum requirements are easily supplied by the average American diet. The AHA and NAS recommend that for healthy adults 2,400 mg/day is a prudent, achievable dietary intake for sodium that will help lower the risk for hypertension in sodium-sensitive individuals (AHA 2000, NIH 1993, NRC 1989a, USDA 2000). Average daily intakes are closer to 3,500 to 4,500 mg/day (Karanja et al. 1999).

About 3% of the population is on sodium-restricted diets, which sometimes require sodium intakes of less than 500 mg (~1/4 teaspoon) per day. Sodium-restricted diets limit sodium exposure to levels of 250, 500, 1,000, or 2,000 mg/day. Each of these diets achieves sodium restriction by limiting the types and amounts of specific foods that can be eaten. A no-added-salt diet restricts only those foods that are high in sodium (e.g., bacon and potato chips). The sodium content of such a diet averages about 4,000 mg/day (Cataldo and Whitney 1986). The fact that such a diet exceeds the dietary goal for sodium intake is indicative of the degree to which sodium is present in the food supply. Individuals on sodium-restricted diets are advised to find out and consider the amount of sodium in their drinking water supply when planning their diet.

In general, sodium salts are not acutely toxic to humans, and sodium salts generally have low acute toxicity in animals (HSDB2000). However, acute effects and death have been reported in cases of very high sodium intake (WHO 1979). The effects due to ingestion of high sodium concentrations tend to be more severe for infants than adults because of the immaturity of infant kidneys (Sax 1975).

Hypertension due to high sodium intake was originally demonstrated in sodium-sensitive SHR rats (Dahl 1967). Numerous studies conducted in humans suggest that excessive sodium intake contributes to age-related increases in blood pressure, leading to hypertension in sensitive

individuals (WHO 1979, NIH 1993). The Intersalt study suggested that blood pressure rises with increasing sodium consumption. This study, which included 10,079 subjects from 52 population centers in 32 countries, reported an increase in systolic pressure of 2.2 mm Hg for every 100 mmol (2,300 mg) increase in sodium intake (ICRG 1988, Elliott et al. 1989). A positive relationship between sodium intake and blood pressure is also indicated by other investigators (Frost et al. 1991, Elliott 1991). Increases in blood pressure are associated with increases in mortality due to coronary artery disease and stroke (Stamler 1991). Sodium intake may also result in an increase in heart muscle thickness as a secondary response to increased blood pressure (Schmieder et al. 1988).

Reports on blood pressure and sodium intake in children are inconsistent. Several researchers have failed to find an association between sodium concentrations in the drinking water and increased blood pressure in children (Pomrehn et al. 1983, Faust 1982, Armstrong et al. 1982, Tuthill et al. 1980, Colditz and Willett 1985), whereas other studies suggest an increase in blood pressure with a high sodium intake (Calabrese and Tuthill 1977, 1981, Tuthill and Calabrese 1979).

Several clinical trials examining the beneficial effect of decreased sodium intake did not yield convincing evidence of a protective effect of low sodium intake on reducing the risk of cardiovascular disease in normotensive populations (Muntzel and Drueke 1992, Salt Institute 2000, NIH 1993, Callaway 1994, Kotchen and McCarron 1998, McCarron 1998). However, the recent results of the DASH and DASH II trials suggest that dietary changes with sodium restriction are beneficial for many with hypertension (Harsha et al. 1999, Sacks et al. 2001). Heart-healthy diets involve weight reduction, exercise, stress reduction, and adequate dietary intake of potassium, calcium, and magnesium as well as restriction of sodium (Whitney et al. 1987). Evidence suggests that chloride restriction also favors lowered blood pressure (Boegehold and Kotchen 1991, Shore et al. 1988). However, because most of the added sodium in prepared foods is sodium chloride, decreased chloride intake usually accompanies decreased sodium intake. Limiting cholesterol, dietary fat, and alcohol intake are also recommended (Whitney et al. 1987). Older individuals with mild essential hypertension and average body weights seem to have the most successful response to dietary salt restriction (Stipanuk 2000).

Data on the reproductive toxicity of sodium are sparse. High doses of sodium chloride (1,570 mg sodium/kg body weight) have been reported to cause maternal effects in rats. The maternal toxicity included decreased pregnancy rates and maternal body weight gain. Developmental effects included increased blood pressure and high mortality (Karr-Dullien and Bloomquist 1979). However, these developmental effects were observed only in SHR rat pups (a type of rat specifically bred to be hypertensive) fed high-sodium diets for up to 4 months after parturition. This study also reported no developmental effects in Sprague-Dawley or WKY rat pups (both normotensive strains of rat).

There is no evidence that sodium is a carcinogen and it does not appear to be genotoxic. High oral doses of sodium chloride may increase the incidence of gastric tumors in the presence of other carcinogens through mechanisms such as damage to the gastrointestinal tract followed by increased DNA synthesis and cell regeneration (Tatematsu et al. 1975, NRC 1989b, Takahashi et al. 1983).

Populations that are expected to have an increased sensitivity to sodium include individuals with hypertension, the elderly (blood pressure increases with age), African-Americans (the incidence of salt-sensitivity and hypertension is disproportionately high among African-Americans), and individuals with renal problems.

7.2   Characterization of Organoleptic Effects

Studies on the organoleptic properties of sodium in drinking water focus primarily on the taste threshold of sodium chloride. The taste threshold for sodium chloride is dependent on whether sodium chloride is in a mixture with other compounds (e.g., sucrose and/or citric acid), the age of the subjects, and on whether or not the target taste is identified beforehand (Weiffenbach et al. 1982, Stevens 1996, Hyde and Feller 1981, Pangborn and Pecore 1982).

Several studies suggest that taste threshold varies between younger and older subjects (Stevens 1996, Weiffenbach et al. 1982, Hyde and Feller 1981). The taste threshold for sodium chloride dissolved in deionized water in young adults (mean age 28 years, n=24) and elderly persons (mean age 75 years, n=24) was 230 and 460 mg Na^+/L, respectively (Hyde and Feller 1981). Stevens (1996) reported that lower taste threshold values were observed in younger subjects (18–29 years, n=15) compared with older subjects (66–90 years, n=15). The mean taste threshold values for sodium chloride dissolved in deionized water for younger and older subjects were 30 and 131 mg Na^+/L, respectively.

The mean taste thresholds for sodium chloride in water for normotensive young adults ranged from 60 to 122 mg Na^+/L when the target taste was not identified and from 32 to 48 mg/L when the target taste was identified (Pangborn and Pecore 1982). Weiffenbach et al. (1982) reported a mean taste threshold of 57 mg Na^+/L. In the 1982 study by Pangborn and Pecore, individuals who habitually have a high-salt diet had a lower taste threshold than those who habitually consumed less salt.

Because the threshold value for the taste of sodium in drinking water varies as a function of dietary habit, age, temperature, masking by other tastants, the anion associated with sodium, and other factors, no single value can be identified as a unique threshold value. The studies by Pangborn and Pecore (1982) and Weiffenbach et al. (1982) indicate a mean taste threshold of approximately 60 mg/L when the nature of the taste was not identified. When the nature of the taste was identified, the average threshold for young adults was approximately 30 mg/L (Pangborn and Pecore 1982, Stevens 1996). On the basis of these values, 30 to 60 mg/L is the lower end of the taste threshold, and many individuals will not be able to detect the presence of sodium in drinking water except at higher concentrations.

7.3   Dose-Response Characterization

Although numerous human studies have examined sodium intake and blood pressure effects, these studies are not adequate to serve as key studies for dose-response characterization for the following reasons: (1) the dose-response relationships varied among the different studies, (2) sodium intake measurements were generally indirect (determined by the amount of sodium excreted in the urine), and (3) the results may have been influenced by other nutrients in diet,

lifestyle, and behavior in addition to sodium (Muntzel and Drueke 1992, Salt Institute 2000, NIH 1993, Callaway 1994, Kotchen and McCarron 1998, McCarron 1998).

The data on dose-response are fraught with controversy. The AHA (2000), NIH (1993), and NRC (1989a) recommend that healthy adults restrict their sodium intake to no more than 2,400 mg/day. Lowering sodium intake by 100 mmol/day (2,300 mg/day) from average levels (~3,500–4,500 mg/day) lowers systolic blood pressure for sodium-sensitive individuals by 3.7 mmHg compared with 1 mm Hg in normotensive individuals. However, dietary changes that increased calcium, potassium, magnesium, and fiber but did not change sodium (DASH diet) were able to achieve similar reductions in systolic pressure in a hypertensive population (Harsha et al. 1999). A combination of the DASH diet with sodium restriction achieved additional reductions in blood pressure among hypertensive and normotensive subjects (Sacks et al. 2001). However, accomplishing a reduction in population exposure to sodium presents a challenge because most of the sodium comes from processed foods rather than from discretionary use of table salt. Sacks et al. (2001) in their presentation of the DASH-sodium results encouraged incentives that would commercially increase the availability of low-salt products.

Data from NIRS and SDWA monitoring of sodium levels in PWSs have shown that the median levels of sodium detected are generally below 30 mg/L. However, many PWSs reported sodium levels greater than 120 mg/L, and the 99th percentile of the samples in NIRS was 517 mg/L. For persons on sodium-restricted diets, sodium concentrations greater than 120 mg/L could be problematic (i.e., could cause an increase in blood pressure) if sodium levels in water remained elevated for a significant period of time.

This Drinking Water Advisory recommends that the sodium concentration in drinking water not exceed a range of 30 to 60 mg/L because of possible adverse effects on taste at higher concentrations. Concentrations below 30 mg/L contribute less than 1.5% of the sodium in an average American diet and less than 2.5% of the present sodium guideline value, assuming consumption of 2 L of tap water per day. For a concentration of 60 mg/L, the comparable values are 3% and 5%.

EPA requires Public Water Systems that exceed 20 mg/L to notify local and State public health officials(U.S. EPA 1996). The EPA guidance was developed for those individuals restricted to a total sodium intake of 500 mg/day (U.S. EPA 1976) and should not be extrapolated to the entire population.

EPA requires periodic monitoring of sodium at the entry point to the distribution system. Monitoring is to be conducted annually for surface water systems and every 3 years for groundwater systems (40CFR:141.41, US EPA 1996). The water supplier must report sodium test results to local and State public health officials by direct mail within 3 months of the analysis, unless this responsibility is assumed by the State. This provides the public health community with information on sodium levels in drinking water.

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