

PART D: SCIENCE BASE

Section 7: Fluid and Electrolytes

This section addresses three major questions related to the intake of fluid and the electrolytes sodium and potassium.

1. What amount of fluid is recommended for health?
2. What are the effects of salt (sodium chloride) intake on health?
3. What are the effects of potassium intake on health?

The Committee placed a strong focus on sodium and potassium because of the substantial body of research linking these electrolytes to levels of blood pressure. Part B, “Introduction,” provides background information on the problem of elevated blood pressure and its control. That information can help the reader appreciate the importance of blood pressure as a modifiable risk factor for cardiovascular and kidney diseases and of dietary factors that can lower and possibly control blood pressure.

The conclusions in this section are largely based on evidence from an extensive, systematic, and very recent review of the scientific literature conducted by an expert panel for the Institute of Medicine (IOM) (IOM, 2004). For topics not covered in the IOM report, we conducted literature searches. The search strategies used to find the scientific evidence related to each of these questions appears in Section C.

QUESTION 1: WHAT AMOUNT OF FLUID IS RECOMMENDED FOR HEALTH?

Conclusion

The combination of thirst and usual drinking behavior, especially the consumption of fluids with meals, is sufficient to maintain normal hydration. Healthy individuals who have routine access to fluids and who are not exposed to heat stress consume adequate water to meet their needs. Purposeful drinking is warranted for individuals who are exposed to heat stress or who perform sustained vigorous activity.

Rationale

Recommendations for water are made to prevent the deleterious, primarily acute, effects of dehydration. These effects include impaired cognitive function and motor control. Although a low intake of water has been associated with some chronic diseases, this evidence is insufficient to establish recommendations for water consumption.

The primary indicator of hydration status is plasma or serum osmolality. Appendix G-1 from the recent IOM report (IOM, 2004) provides the serum osmolality by decile of total water intake in the third *National Health and Nutrition Examination Survey* (NHANES III). Serum osmolality concentrations were essentially identical (the maximum range between the lowest and highest decile was only 3 mOsmol/kg). These data indicate that persons in the lowest and highest deciles of total water intake were neither systematically dehydrated nor

hyperhydrated. Importantly, this pattern of findings also was evident in men and women age 71 and older.

Thirst, which is the desire to drink by both physiological and behavioral cues, may be triggered by a decrease in blood volume or severe dehydration. Over the course of a few hours, body water deficits can occur. However, thirst mechanisms come into play over the ensuing 24 hours to trigger replacement of fluids lost (Johnson, 1964). Such replacement is enhanced by consuming beverages at meals and in other social situations (Engell, 1995; Szlyk, 1990).

Total water intake includes drinking water, water in beverages, and water contained in food. Because normal hydration can be maintained over a wide range of water intakes, the Adequate Intake (AI) for total water was set based on the median total water intake from U.S. survey data (IOM, 2004). The AI for total water intake for young men and women (age 19 to 30 years) is 3.7 L and 2.7 L per day, respectively. In NHANES III, fluids (drinking water and beverages) provided 3.0 L (101 fluid ounces; ~13 cups) and 2.2 L (74 fluid ounces; ~9 cups) per day for men and women age 19 to 30, representing approximately 81 percent of total water intake. Water contained in food provided about 19 percent of total water intake.

The AI should not be interpreted as a specific requirement or recommended intake. Individual water requirements can vary greatly, even on a day-to-day basis, primarily because of differences in physical activity and environmental conditions but also because of differences in diet. A total water intake above the AI often is required by those individuals who are physically active or who are exposed to heat stress. In individuals who are neither physically active nor exposed to heat stress, daily consumption below the AI can be sufficient to maintain normal hydration. Dietary factors also influence water requirements because total water consumption must be sufficient to excrete metabolites of protein and organic compounds, as well as excess electrolytes.

Because healthy individuals have considerable ability to excrete excess water and thereby maintain water balance, the IOM did not set a Tolerable Upper Intake Level (UL) for water. However, acute water toxicity can occur following the rapid consumption of large quantities of fluids that greatly exceed the kidney's maximal excretion rate of approximately 0.7 to 1.0 L per hour.

QUESTION 2: WHAT ARE THE EFFECTS OF SALT (SODIUM CHLORIDE) INTAKE ON HEALTH?

Conclusion

The relationship between salt (sodium chloride) intake and blood pressure is direct and progressive without an apparent threshold. Hence, individuals should reduce their salt intake as much as possible. In view of the currently high levels of salt intake, a daily sodium intake of less than 2,300 mg is recommended. Many persons will benefit from further reductions in salt intake, including hypertensive individuals, blacks, and middle- and older-aged adults. Individuals should concurrently increase their consumption of potassium because a diet rich in potassium blunts the effects of salt on blood pressure.

Rationale

A recent report from the IOM (IOM, 2004) provides the basis for a recommended daily sodium intake (an AI) of 1,500 mg and a UL of 2,300 mg for adults^{1,2}. These recommendations are based on an extensive examination of the scientific literature by an expert IOM (IOM) panel. The primary basis for setting the AI was to ensure overall nutrient adequacy, not to prevent chronic disease. In contrast, the UL was set because of the direct relationship of salt intake with blood pressure.

Review of the Evidence

Studies of the Relationship of Sodium Intake and Blood Pressure. The relationship between sodium intake and blood pressure is direct and progressive. In addition to observational studies, supportive evidence comes from more than 50 clinical trials and meta-analyses (see IOM, 2004, Tables 6-12, 6-13, 6-15, 6-16, and Appendix I). The best available dose-response evidence comes from individual trials that specifically examined this relationship (i.e., randomized trials that tested the effects of three or more levels of sodium intake on blood pressure). In these dose-response studies, the lowest level of sodium intake ranged from approximately 230 to 1,500 mg of sodium per day, while the highest level ranged from approximately 3,200 to over 34,000 mg of sodium per day. The largest and most rigorous of these trials documented statistically significant, progressive dose-response relationships (Johnson et al., 2001; MacGregor et al., 1989; Sacks et al., 2001). Importantly, there was no evidence of a threshold; that is, the direct relationships were evident throughout the range of salt intake.

The trial by Johnson et al. (2001) tested the effects of 5 levels of sodium intake (lowest to highest: 920 mg per day to 7,800 mg per day) in 40 older-aged persons (nonhypertensives, persons with isolated systolic hypertension, persons with combined systolic-diastolic hypertension). The trial by MacGregor et al. (1989) tested the effects of 3 levels of sodium intake (1,100; 2,300; and 4,600 mg of sodium per day) in 20 hypertensive adults. The largest of the dose-response trials, the *Dietary Approaches To Stop Hypertension (DASH) -Sodium* trial, tested the effects of three different sodium intakes separately in two distinct diets—the DASH diet and a control diet. The DASH diet is described in detail in Section D1 and in Table D1-18. In brief, the DASH diet is rich in fruits, vegetables, and low-fat dairy products and reduced in total fat, saturated fat and cholesterol. The control diet is typical of what many Americans eat, that is, relatively high in total and saturated fats and low in fruits, vegetables, and low-fat milk products. Mean achieved levels of sodium intake, as reflected by 24-hour urinary sodium excretion, corresponded to approximate intakes of 1,500; 2,500; and 3,300 mg in the lower, intermediate, and higher doses, respectively.

¹ Previous recommendations from authoritative sources have recommended consuming less than 2,400 mg of sodium rather than 2,300 mg. The limit of 2,400 mg is equivalent to 6 g of sodium chloride, whereas the limit of 2,300 mg corresponds to 100 mmol of sodium. Previous standards had been based on milligrams of sodium chloride rather than millimoles of sodium.

² In view of the form of published data and nutrition labeling, which typically provide milligrams of sodium rather than milligrams of salt, this section will present recommendations in milligrams of sodium.

Of the 3 dose-response trials, the DASH-Sodium trial enrolled the largest and most diverse population; 41 percent were hypertensive, 40 percent were white, and 57 percent were black. However, the DASH-Sodium trial had the narrowest range of sodium intake; approximately half of the U.S. population consumes sodium in excess of the highest level tested in this trial (Rose et al., 1988).

The main results of the DASH-Sodium trial are displayed in Figure D6-1. The blood pressure response to sodium reduction was nonlinear. Specifically, reducing sodium intake by approximately 920 mg per day caused a greater lowering of blood pressure when the initial sodium intake was at the intermediate level than when it was at the higher intake; this pattern of results was especially evident on the control diet. Results from the INTERSALT observational study (Rose et al, 1988) and from the Johnson trial likewise suggest that the blood pressure response to changes in sodium intake is steeper below 2,300 mg per day than above 2,300 mg per day.

In protocol-specified subgroup analyses of the DASH-Sodium trial (Vollmer et al., 2001), a reduced-sodium intake significantly lowered blood pressure in each of the major subgroups on the control diet. On the control diet, reduced-sodium intake led to greater systolic blood pressure reduction among hypertensive individuals, blacks, and persons age 45 years and older compared to their counterparts. Net systolic/diastolic blood pressure reductions associated with reducing salt from the higher to the lower level in hypertensives and nonhypertensives were 8.3/4.4 and 5.6/2.8 mmHg, respectively. On the DASH diet, a qualitatively similar pattern was evident, but the extent of blood pressure reduction was less; net systolic/diastolic blood pressure reductions associated with reducing salt from the higher to the lower level in hypertensives and nonhypertensives were 4.9/2.5 and 1.7/1.1 mmHg, respectively. In each subgroup, the lowest blood pressure was observed on the DASH diet with the lower sodium level.

In subsequent pos-hoc analyses, Bray et al. (2004) presented results in joint subgroups (age and hypertension status, race/ethnicity and hypertension status, and sex and race/ethnicity). In the control diet, sodium reduction significantly lowered systolic blood pressure in each subgroup. In the DASH diet, many but not all blood pressure changes associated with sodium reduction were statistically significant in the subgroups. In all subgroups, the DASH diet significantly lowered blood pressure at the higher sodium level; however, at the lower sodium level, several blood pressure reductions did not achieve statistical significance. Overall, the general pattern of results in subgroup analyses was similar to that of the main results. Deviations between main results and subgroup analyses, especially post-hoc analyses, should be interpreted cautiously because of reduced sample size and hence the potential for false negative associations.

The control diet, in which the blood pressure effect of sodium reduction was the largest, is closer to what most Americans currently eat than is the DASH diet. For instance, in the United States less than 10 percent of adult men and 1 percent of women consume 4.7 g per day of potassium (the potassium goal of the DASH diet), and less than 25 percent of adult men and less than 5 percent of adult women have a daily calcium intake from foods of 1,240 mg per day (the calcium goal of the DASH diet) (IOM, 2004). The low-saturated fat and

total-fat contents of the DASH diet (goals of 6 percent and 27 percent kcal, respectively) are likewise uncommon in the U.S. population. These observations, coupled with the consistency of the findings across subgroups, support recommendations to concurrently reduce sodium intake and consume the DASH diet. Although the duration of each feeding period lasted only one month, it is reasonable to speculate that adherence to the combination of the DASH diet and reduced sodium intake might help blunt the well-documented rise in blood pressure that occurs with age, especially because reductions in systolic blood pressure were greater in the older than younger participants.

As documented above (also, see IOM, 2004 Tables 6-13 and 6-15) the effects of sodium on blood pressure are large and clinically relevant in hypertensive individuals not on medication. Sodium reduction also lowers blood pressure in the presence of antihypertensive drug therapy (Appel et al., 2001). Although the effects of sodium intake on blood pressure are smaller in nonhypertensive individuals, the potential benefits of sodium reduction on blood pressure have substantial public health relevance. Stamler et al. (1989) estimated that a 3 mmHg reduction in systolic BP could lead to an 8 percent reduction in stroke mortality and a 5 percent reduction in mortality from coronary heart disease. In observational studies, a reduced salt intake (as manifest by 24-hour urinary sodium excretion) is also associated with a blunted age-related rise in blood pressure (Rose et al., 1988).

Sodium reduction can also prevent incident hypertension. To date, three trials have explored the effects of a reduced sodium intake as a means to prevent hypertension (*Hypertension Prevention Trial* [HPT], *Trial of Hypertension Prevention Phase I* [TOHP1], and *Phase II* [TOHP2 Collaborative Research Group, 1997]). HPT and TOHP1 were pilot studies that were conducted to inform the design of TOHP2. Each study was a controlled trial in which a behavioral intervention focused exclusively on reducing sodium intake. HPT and TOHP2, also included groups that simultaneously implemented other interventions: increased potassium intake in HPT and weight loss in TOHP2 (1997). Net reductions in urinary sodium excretion on the sodium reduction arm were modest in the three studies, ranging from the equivalent of 300 mg to ~1,300 mg of sodium per day, at the end of followup. In this setting, a reduced sodium intervention that did not include any other lifestyle change led to a decreased relative risk of incident hypertension (range 0.69 to 0.82).

Results from TOHP2 are especially relevant because this trial was designed and adequately powered to test the effects of a reduced dietary sodium intervention as a means to prevent hypertension. TOHP2 was a randomized, controlled 2 x 2 factorial trial that tested the effects of 3 lifestyle interventions (sodium reduction, weight loss, or combined weight loss and sodium reduction) on blood pressure and incident hypertension over 3 to 4 years of followup in overweight individuals aged 30 to 54 years with an initial diastolic blood pressure of 83 to 89 mm Hg and a systolic blood pressure < 140 mm Hg. At 6 months, the height of intervention adherence, the incidence of hypertension was lowest in the combined group (2.7 percent), intermediate in the weight loss only (4.2 percent) and sodium reduction only (4.5 percent) groups, and highest in the control group (7.3 percent). At 18 months, the pattern persisted. By the end of follow-up, the incidence of hypertension was 18 to 22 percent less in each lifestyle group ($p < 0.05$ compared to control) but not different from each other. Results of this trial indicate that lifestyle interventions can prevent hypertension over the long-term.

Also, the pattern of incident hypertension at 6 and 18 months suggests that the effects of weight loss and reduced sodium intake, under optimal conditions of adherence, may be additive.

Relying on behavioral interventions to reduce dietary intake of sodium presents a major barrier to the achievement of greater reductions in blood pressure and to a reduction in the associated CVD complications. In contrast to the short-term (3-day) feeding trials that could achieve contrasts in sodium intake of nearly 34,300 mg per day (Luft et al., 1979), the maximum contrast in the primary prevention trials was 1,300 mg per day in TOHP1. The average contrast in long-term trials lasting 6 months was only 800 mg per day (Hooper et al., 2002). The limited contrast in sodium intake in these trials reflects the difficulties of sustaining behavior change when the most common source of sodium, namely processed foods, accounts for > 75 percent of total sodium intake and when discretionary salt intake accounts for only 11 percent (5 percent added during cooking and 6 percent added at the table) (Mattes, 1997). The sodium that occurs naturally in foods accounts for the remainder (approximately 10 percent).

Salt Sensitivity. Evidence from a variety of studies, including observational studies and clinical trials, has demonstrated heterogeneity in the blood pressure responses to sodium intake. Those individuals with the greatest reductions in blood pressure in response to decreased sodium intake are termed *salt sensitive*. Despite the use of the terms *salt sensitive* and *salt resistant* to classify individuals in research studies, the change in blood pressure in response to a change in salt intake is not binary. Rather, the reduction in blood pressure from a reduced sodium intake has a continuous distribution across individuals. Also, there are no standardized diagnostic criteria and tests. Despite these limitations, it is possible to make some general observations.

Salt sensitivity is modifiable. The rise in blood pressure from increased salt intake is blunted in the setting of a high potassium intake (4.7 g of supplemental potassium per day in one trial, (Morris et al., 1999); 6.7 g per day in another trial, (Schmidlin et al., 1999)). The rise in blood pressure from increased salt intake was also blunted in the setting of the DASH diet, which is rich in potassium (4.6 g of potassium per day) as well as other minerals (Table 6-1) (Bray et al., 2004; Karanja et al., 1999; Sacks et al., 2001; Vollmer et al., 2001); nonetheless, a dose-response relationship between sodium intake and blood pressure persisted.

Individuals with hypertension, diabetes, and chronic kidney disease, as well as middle- and older-aged persons and blacks tend to be more salt sensitive than their counterparts. Genetic factors also influence the blood pressure response to salt. Each of the 14 identified genes that affect blood pressure affects renal salt handling. Such evidence provides indirect support of an etiologic role of sodium in blood pressure homeostasis (Lifton, 2002).

Relationships Between Salt Intake and Health Outcomes Other Than Blood Pressure. As documented by the IOM (IOM, 2004), an increased sodium intake might have adverse effects on additional health outcomes. These include clinical cardiovascular outcomes (i.e., stroke and coronary heart disease), subclinical cardiovascular outcomes (i.e., left ventricular mass), and noncardiovascular outcomes (e.g., urinary calcium excretion,

osteoporosis, and gastric cancer). Cross-sectional studies consistently document an association between urinary sodium excretion and left ventricular mass, but only one small controlled trial assessed the effects of sodium reduction on this endpoint. Numerous trials document that a reduced sodium intake lowers urinary calcium excretion (Table 6-19; IOM, 2004), but urinary calcium excretion, by itself, is not a well-accepted surrogate marker for bone mineral density or dietary induced osteoporosis. Evidence that links sodium intake with gastric cancer is reasonably strong but still insufficient to establish a UL for sodium. No trial has tested the effects of sodium reduction as a means to prevent cardiovascular disease (CVD). However, the most rigorous observational studies (He and MacGregor, 1999; Tuomilehto et al., 2001; see Table 6-17 IOM, 2004) have documented a direct relationship of sodium intake with CVD.

Salt Taste Preferences. At birth, there is no indication that salty substances are distinguishable or preferred (Beauchamp et al., 1986). Preference for the salty taste appears at about four months postnatal (Beauchamp et al., 1994; Beauchamp et al., 1986; Harris and Booth, 1987). Limited evidence suggest that infants' and children's salt preference is shaped by their experience with salt in foods (Beauchamp, 1990; Stein et al., 1996)

Adult salt preferences can be influenced by dietary exposure. Studies have demonstrated that reducing one's dietary sodium intake can decrease one's preference for salty foods and increase acceptance of foods with reduced sodium content (Bertino et al., 1982). Several studies document a temporary increased preference for salt over the initial few weeks when sodium intake is reduced (Bertino et al., 1981; McCance, 1936, Teow et al., 1985–1986; Yensen, 1959). Subsequently, a shift in preference occurs such that by 8 to 12 weeks individuals prefer less salty foods (Bertino et al., 1982; Mattes et al., 1991; Mattes, 1997). This phenomenon also has been demonstrated in long-term studies lasting one year or more (Blais et al., 1986).

On average, the natural salt content of food accounts for only 10 percent of total intake, while discretionary salt use (i.e., table and cooking salt) provides another 5 to 10 percent of total intake. The remaining 75 percent is derived from salt added by manufacturers (James, 2000; Mattes 1991, 1997). When total intake of salt is decreased, discretionary salt use is fairly stable, even when available *ad libitum* (Mattes, 1997). Therefore, any program for reducing the salt consumption of a population should concentrate primarily on a reduction in the salt used during food processing (James, 2000) and on changes in food selection (e.g., more fresh, less-processed items, less sodium-dense foods) and preparation (Mattes, 1997). Previous guidelines have focused on decreasing the intake of foods and beverages high in salt (HHS 1985, 1990, 1995, 2000) because of their large contribution of salt intake in the diet.

Recommendations for Salt (Sodium Chloride) Intake

The IOM set the AI for sodium for adults at 1,500 mg per day to ensure that the overall diet provides sufficient amounts of other nutrients and to cover sodium sweat losses in unacclimatized individuals who are exposed to high temperatures or who are moderately physically active (IOM, 2004). This amount of sodium does not apply to highly active individuals, such as endurance athletes and certain workers (e.g., foundry workers) who lose large amounts of sweat on a daily basis and thus require a higher sodium intake.

The IOM set the UL at 2,300 mg of sodium per day (IOM, 2004). In dose-response trials, this level of sodium intake commonly was the next tested level above the AI. The UL of 2,300 mg of sodium daily is not a recommended intake. There is no benefit to consuming sodium in an amount that exceeds the AI. For members of groups that are most sensitive to the blood pressure effects of increased salt intake (that is, middle- and older-aged persons, blacks, and individuals with hypertension, diabetes, or chronic kidney disease), it is advisable to consume an amount of sodium that is less than the UL. These groups also have higher levels of blood pressure

Positions Taken by Other Policymaking Groups. Numerous policymaking organizations have recommended a reduced salt intake as a means to lower blood pressure in the general population. In the United States, the National High Blood Pressure Education Program set a sodium intake goal of 100 mmol (2,300 mg) per day as a means to prevent hypertension in nonhypertensive individuals (Whelton et al., 2002) and as first line and adjuvant therapy in hypertensive individuals (Chobanian et al., 2003). The American Heart Association set an intake of 6 g of salt (2,400 mg of sodium) per day as a recommended upper limit for healthy Americans. In Great Britain, the Scientific Advisory Committee on Nutrition in 2003 conducted an independent review of available evidence and also set an upper limit of 6 g of salt (2,400 mg of sodium) per day. Recently published Canadian recommendations for the prevention and treatment of hypertension are to restrict salt intake to 65 mmol to 100 mmol (1,500 mg to 2,300 mg) per day in hypertensive individuals and to 100 mmol (2,300 mg) per day in normotensive individuals at risk for becoming hypertensive (Touyz et al., 2004). Note that in the United States, 90 percent of adults will develop hypertension (Vasan et al., 2002). In its report, *Diet, Nutrition and the Prevention of Chronic Diseases* (2003), the World Health Organization set an upper limit of 70 mmol (1,600 mg) of sodium per day as a means to lower blood pressure.

Sodium Intakes

According to data from NHANES III (IOM, 2004), the median intakes of sodium among adult men and women age 31 to 50 are 4,300 mg and 2,900 mg of sodium per day, respectively. One quarter of adult men exceed 5,200 mg of sodium per day, and one quarter of women exceed 3,500 mg per day. Approximately 95 percent of adult men and 75 percent of adult women exceed the UL of 2,300 mg of sodium per day, and 100 percent exceed the AI of 1,500 mg of sodium per day. On average, blacks and non-blacks consume similar amounts of sodium. The reported sodium intakes probably are underestimates of total sodium intake because the NHANES III did not ask about discretionary salt intake.

QUESTION 3: WHAT ARE THE EFFECTS OF POTASSIUM INTAKE ON HEALTH?

Conclusion

Diets rich in potassium can lower blood pressure and lessen the adverse effects of salt on blood pressure, may reduce the risk of developing kidney stones, and possibly decrease bone loss. In view of the health benefits of potassium and its relatively low intake by the general population, a daily potassium intake of at least 4,700 mg is recommended. Blacks are especially likely to benefit from an increased intake of potassium.

Rationale

Review of the Evidence

Effect of Potassium on Blood Pressure and Salt Sensitivity. Supportive evidence for the conclusion that an increased potassium intake lowers blood pressure appears in the IOM report (IOM, 2004), as follows:

- Table 5-2 covering 17 observational studies
- Tables 5-3 through 5-5 covering 36 clinical trials

Most trials tested pill supplements, typically in the form of potassium chloride (Tables 5-4 and 5-5, IOM, 2004). Three meta-analyses of these trials document that, on average, increased potassium intake lowers blood pressure in nonhypertensive and hypertensive individuals (Cappuccio and MacGregor, 1991; Geleijnse et al., 2003; Whelton et al., 1997). In the meta-analysis by Whelton et al. (1997), average net systolic/diastolic blood pressure reductions from a net increase in urinary potassium excretion of 2 g per day (50 mmol per day) were 4.4/2.5 mmHg among hypertensive individuals and 1.8/1.0 mmHg among nonhypertensive individuals. No dose-response trial tested the effects of more than two levels of potassium intake on blood pressure.

Relatively few trials tested the effects of potassium as provided in foods (Table 5-3, IOM, 2004). The potassium in fruits and vegetables is accompanied by bicarbonate precursors rather than chloride. In the initial DASH trial, a diet rich in fruit and vegetables (and therefore rich in potassium) lowered blood pressure (Appel et al., 1997). Another trial documented that increased fruit and vegetable consumption can significantly lower blood pressure (John et al., 2002), but that trial did not report the potassium intake of participants on the fruit and vegetable intervention.

Because virtually all trials used potassium chloride supplements while observational studies assessed dietary potassium intake from foods (paired with nonchloride anions), the effect of potassium on blood pressure appears to result from potassium rather than its conjugate anion. No single trial tested the effects of three or more levels of potassium intake on blood pressure; hence, the dose-response relationship is unclear. Still, blood pressure reductions from supplemental potassium occurred when baseline intake was low (e.g., 1.3 to 1.4 g of potassium per day in Brancati et al., 1996) and when baseline intake was much higher (> 3.1 g of potassium per day in Naismith and Braschi (2003)).

Evidence from the observational studies and clinical trials has demonstrated heterogeneity in the blood pressure responses to potassium intake. Blacks and hypertensive individuals are more sensitive to the effects of potassium than their nonblack and normotensive counterparts, respectively. Dietary salt intake also modifies the effects of potassium on blood pressure. Specifically, the effects of potassium on blood pressure are greater when salt intake is high than when salt intake is low (see Table D6-1).

Some trials have assessed the effects of increased potassium intake on salt sensitivity, that is, the pressor response to increased salt intake. Study populations included nonhypertensive predominantly black individuals (Morris et al., 1999; Schmidlin et al., 1999) and hypertensive

individuals (Morgan et al., 1984). These trials are consistent in documenting that potassium blunts the pressor (blood-pressure raising) effects of salt. One dose-response trial documented that increasing potassium intake to 4.7 g per day reduced salt sensitivity in nonhypertensive blacks (Morris et al., 1999). In aggregate, these trials highlight the potential benefits of increasing potassium intake in blacks, a group of individuals with a high prevalence of hypertension and of blood pressure-related cardiovascular-renal disease.

To date, no trial has tested the effects of increased potassium intake on blood pressure-related clinical outcomes. However, observational studies suggest that increased potassium intake may prevent stroke and perhaps coronary artery disease (see Table 5-6, IOM, 2004).

Effect of Potassium in Preventing Bone Loss and Kidney Stones. A diet rich in potassium from fruits and vegetables favorably affects acid-base metabolism because these foods also are rich in precursors of bicarbonate (Sebastian, et al., 1994, 2002). Acting as a buffer, the bicarbonate-yielding organic anions found in fruits and vegetables neutralize acids generated from meats and other high-protein foods. In the setting of an inadequate intake of bicarbonate precursors, excess acid in the blood titrates bone buffer. This results in demineralization of the bone. Increased bone breakdown and calcium-containing kidney stones are adverse consequences of excess acid derived from the diet. Therefore, diets rich in potassium with its bicarbonate precursors might help prevent kidney stones and bone loss.

To date, two observational studies have documented that high intakes of potassium (median of 4.0 g per day in men and 4.7 g per day in women) are associated with a reduced risk of incident kidney stones (Curhan et al., 1993, 1997). In a third observational study conducted in Finland, the relationship was statistically nonsignificant, perhaps because of the much higher usual levels of potassium consumed in this population (Hirvonen et al., 1999). In addition, one trial (Barcelo et al., 1993) documented that approximately 3.6 to 4.7 g of supplemental potassium citrate reduced the risk of recurrent kidney stones. The potassium added to processed foods and the potassium in supplements typically has chloride as the conjugate anion. Since chloride cannot neutralize excess acid in the body, this form of potassium is not expected to help prevent kidney stones or bone loss.

Observational studies, including both cross-sectional studies and longitudinal studies, suggest that increased potassium intake is associated with increased bone mineral density (See IOM, 2004, Table 5-7). Trials also have documented that supplemental potassium bicarbonate can reduce bone breakdown and increase bone formation (Sebastian et al., 1994). However, no trial has tested the effect of increased potassium or diets rich in potassium on bone mineral density or on clinical outcomes related to osteoporosis.

Recommendations for Potassium Intake

The IOM set the AI for potassium for adults at 4,700 mg per day. This level of intake should maintain lower blood pressure levels, mitigate the adverse effects of salt on blood pressure, reduce the risk of developing kidney stones, and possibly decrease bone loss. At present, dietary intake of potassium by all groups in the United States is considerably lower than 4,700 mg per day. In recent surveys, the median intake of potassium by adults in the United States was approximately 2,900 to 3,200 mg per day in men and 2,100 to 2,300 mg per day in

women. On average, blacks consume less potassium than non-blacks. Among men, age 31 to 50 years, median potassium intake was approximately 2,600 mg in blacks and 3,300 mg in non-blacks. Corresponding figures in women were 1,900 mg and 2,400 mg, respectively (see Table D1-7). Because blacks have a relatively low intake of potassium and a high prevalence of elevated blood pressure and salt sensitivity, this subgroup of the population would especially benefit from an increased intake of potassium.

In the generally healthy population with normal kidney function, a potassium intake from foods that exceeds 4.7 g per day poses no potential for increased risk because excess potassium is readily excreted in the urine. Hence, the IOM did not set a UL for potassium (IOM, 2004). However, a potassium intake below 4.7 g per day is indicated for individuals whose urinary potassium excretion is impaired. Adverse cardiac effects (arrhythmias) can result from hyperkalemia, which is a markedly elevated serum level of potassium. Common drugs that can substantially impair potassium excretion are angiotensin converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), and potassium-sparing diuretics. Medical conditions associated with impaired potassium excretion include diabetes, chronic kidney disease, end stage renal disease, severe heart failure, and adrenal insufficiency. As a group, elderly individuals are at increased risk of hyperkalemia because they often have one or more of these conditions or take one or more of the above medications.

SUMMARY

Healthy persons who have routine access to fluids and who are not exposed to heat stress consume adequate water to meet their needs. Thus, the Committee makes no special recommendations concerning water intake. To help lower blood pressure, the Committee recommends that individuals reduce their salt intake as much as possible, aiming for less than 2,300 mg of sodium daily. The Committee recommends a concurrent increase in potassium intake to 4,700 mg daily. In addition to helping lower blood pressure and blunting the effects of salt on blood pressure, this amount of potassium intake may reduce the risk of developing kidney stones and possibly reduce bone loss. Blacks are especially likely to benefit from reductions in sodium intake and increases in potassium intake.

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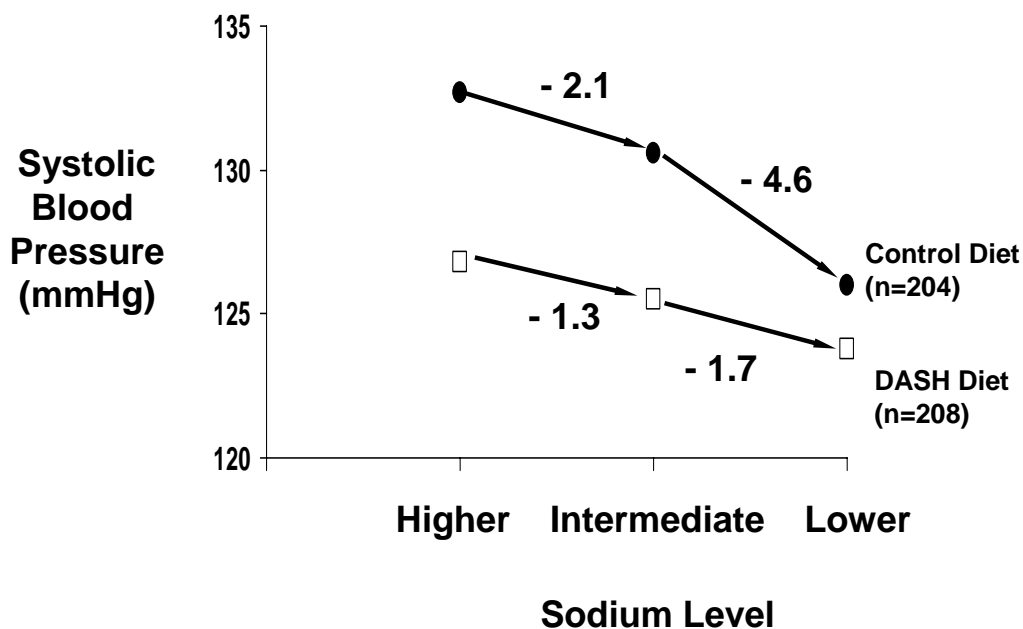
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Figure D7-1. Dose-Response Relationship Between Systolic Blood Pressure and Sodium Intake in Two Diets: Main Results From the DASH Sodium Trial (Sacks FM et al., 2001)



Control Diet represents the typical American diet. DASH diet emphasizes fruits, vegetables, and low-fat dairy foods, includes whole grains, poultry, fish, and nuts, and is reduced in fats, red meat, sweets, and sugar-containing beverages. The 3 sodium levels are defined as higher (3,450 mg/d), intermediate (2,300 mg/d) and lower (1,150 mg/d).

Table D7-1. Trials That Assess the Main and Interactive Effects of Salt and Potassium on Blood Pressure

Citation	Design	Population	Sodium Levels Tested	Potassium Levels Tested	Duration	Main Effects of Sodium	Main Effects of Potassium	Interactive Effects	Interpretation
Sacks et al., 2001	Crossover trial of three sodium levels, parallel trial of 2 diets	412 adults, % women, % AA, 135/86 mmHg	50 (L), 100 (I), 150 (H) mmol/day	1700 mg/d in control diet, 4700 mg/day in DASH diet	30 days	SBP: -7.9 mmHg (H to L) in control diet, -3.0 mmHg in DASH diet; DBP: -3.5 mmHg (H to L) in control diet, -1.6 mmHg in DASH diet	SBP (DASH diet net of control): -5.9 mmHg at H, -2.2 mmHg at L; DBP (DASH diet net of control): -2.9 mmHg at H, -1.0 mmHg at L;	Subadditive effect of sodium reduction and DASH diet (p<0.001)	The DASH diet (rich in potassium) blunts but does not eliminate the effects of increased sodium on blood pressure. A low sodium intake blunts the effects of the control diet (low in potassium) on blood pressure.
Morris et al., 1999	parallel, sequential phases; baseline (I) with low K+Na, then 'Na loading' low K and high Na, then 'K loading'	38 men, 63% AA, <140/<90 mm Hg	15 mmol/day (basal diet), then 250 mmol/d (high salt)	30 mmol/day (basal diet), then 70 mmol (A), 70 mmol (B), 120 mmol (C), placebo (D)	6 weeks total (2 week basal, 1 week Na loading, 3 weeks with K loading)	Pre-Post Na loading: SBP: +8.8 mmHg in Blacks (basal to high salt), +2.9 mmHg in Whites (basal to high salt); +DBP: 5.6 mmHg in Blacks (basal to high salt), +1.4 mmHg in Whites (basal to high salt)	Pre-Post K loading: SBP: -4.9 mmHg in blacks (basal to A or B tx), -2.5 mmHg in whites (basal to A or B tx); DBP: -3.3 mmHg in Blacks (basal to A or B tx), -1.9 mmHg in whites (basal to A or B tx)	Supplementing dietary potassium at 70 mmol/d attenuated moderate salt sensitivity in both blacks and whites (p<0.01) and at 120 mmol/d abolished salt sensitivity and suppressed the frequency and severity of salt sensitivity in blacks (n=5) to levels similar to those observed in whites.	Effects of sodium and potassium based on pre-post BP change, not net of placebo. K appears to blunt the rise in blood pressure from sodium and do so in a dose-dependent fashion in blacks.
Skrabel et al., 1981	2 x 2 factorial	20 men, 21-25, all non-hypertensive, 125/73.1 mmHg	50, 200 mmol	80, 200 mmol	2wk	SBP: -2.7, DBP:-3.0, both NS	SBP: -1.7, DBP:-4.5, both NS	SBP: -2.3, DBP:-3.5, both NS	Small trial, all BP change NS
Chalmers et al., 1986	2 x 2 factorial	212 adults with hypertension, DBP: 90-100 mmHg	~80, ~150 based on 24 hr urines	~70, ~90 based on 24 hr urines	12 weeks	SBP: -3.9, DBP:-3.1, both sign	SBP: -5.1, DBP:-4.2, both sign	SBP: -4.2, DBP:-2.6, both sign; no formal test but BP effects of increased K and reduced Na appear subadditive	Increased potassium or reduced sodium, alone or together, reduce blood pressure to the same extent.