Making Sense of the Science of Sodium

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Despite the Institute of Medicine's commitment to base its nutrient intake recommendations in evidence, the 2004/2005 Dietary Reference Intakes for sodium were not supported by evidence, as the subsequent 2013 Institute of Medicine review admitted. In this review, I suggest an approach to setting nutrient intake requirements based in physiology. Briefly, the requirement of a given nutrient can best be said to be the intake that calls for the least adaptation or compensation by the intact organism. For sodium, evidence indicates that such an intake is typically between 3000 and 5000 mg/d.
Nutr Today. 2015;50(2):63–66

DIETARY REFERENCE INTAKES: THE BACKGROUND

In the early 1990s, a decision was made by the Food and Nutrition Board of the Institute of Medicine (IOM) to the effect that, in the future, nutrient intake recommendations (ie, Dietary Reference Intakes [DRIs]), would be "evidence based." Any implication that previous editions of the intake recommendations had not been based in evidence would certainly not be correct. What was to be different, going forward, was how the evidence was gathered and evaluated.[1,2] The expectation was that this process would be explicitly set forth so that the basis for the recommendations would be transparent, and subsequent revisions would be built upon what the previously available evidence had supported. Although the objectives of this process seemed sensible, and even ideal, its implementation over the past 20-plus years has often been inconsistent and even internally contradictory. And, rather than eliminating controversy, both the process and the outcomes have, in many cases, been hotly contested. The casualties of the several controversies have been the American public, patients with chronic disease whose diets were altered to accord with the DRIs, and, for some nutrients (such as sodium), the food industry.

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The author has no conflicts of interest to disclose.

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DOI: 10.1097/NT.0000000000000084

SALT CONCERNS

As has been exhaustively reviewed previously,[3] the general populations of the United States and United Kingdom have been subjected to a steady program aimed at decreasing sodium intake, dating back to at least the early 1980s, and probably a decade or 2 earlier. The justification for this policy effort was the fact that blood pressure is, to some extent, dependent upon sodium intake, particularly in individuals deemed "salt sensitive."[4] It had long been recognized that high blood pressure is a risk factor for cardiovascular disease (CVD) and cardiovascular mortality and that reducing *elevated* blood pressure lowers that risk. These general relationships are widely accepted and are not in question. However, these findings derived from patients with hypertension were extrapolated to conclude that lowering sodium intake in nonhypertensive individuals would lower blood pressure in them as well and would thereby reduce risk of heart disease. There are 2 components to this presumption: (1) that lowering salt intake in normotensive individuals will, in fact, lower blood pressure meaningfully and (2) that lowering salt intake in normotensive individuals will reduce adverse health outcomes. Both are false. It is important to recall that in a recent Cochrane analysis of nearly 170 randomized controlled trials, Graudal et al[5] found not a single example of a study with a blood pressure effect from sodium intake reduction in normotensive individuals. There were no studies of health benefits accruing to the reduction of salt intakes of healthy adults to levels below those prevailing in Europe and North America (~3450 mg/d; 150 mmol/d).[4,6] Thus, the presumptions expressed in the 2004/2005 DRIs have never been supported by evidence. Nevertheless, a presumption of benefit continues to be expressed in the sodium intake recommendations of numerous otherwise authoritative bodies.[7] Reflecting this lacuna, Drummond Rennie, editor of *JAMA*, was quoted in an interview in *Science* as saying that the "…authorities pushing the 'eat-less-salt' message had made a commitment to salt education that goes way beyond the scientific facts."[3]

The problem with drawing any conclusion from the limited evidence available—and this cannot be stressed too strongly—was that the studies concerned had been done in individuals who already had a metabolic abnormality (ie, hypertension) and who often, as well, had above average sodium intakes. In its review and evaluation of the more recent evidence (up to 2012), the IOM published a revised analysis in 2013,[8] stating belatedly that there was no evidence of benefit for reduction of sodium intake below 2300 mg/d (100 mmol/d).

Nevertheless, the 2004/2005 DRIs from the IOM have been allowed to stand and remain the basis for federal salt policy today.

In brief, blood pressure reduction is a reasonable proxy for health outcomes in hypertensive individuals on high sodium intakes, but it simply does not track health outcomes in normotensive individuals at average or below average sodium intakes. Finally, it is important to note that the 2004/2005 IOM task force did not evaluate the risk of adverse health outcomes, if any, produced as a consequence of lowering salt intake in nonhypertensive individuals,[9] and it was in part to rectify this omission that the 2012/2013 IOM panel was convened.

SHIFTING SALT POLICY

To review, the 2004/2005 sodium intake recommendation, termed "AI" (for adequate intake), was set at 1500 mg/d (65 mmol/d) for adults up to age 50 years, 1300 mg/d (57 mmol/d) for adults between 50 and 70 years, and 1200 mg/d (52 mmol/d) for adults older than 70 years.[5] As has been pointed out previously,[10] an AI is specifically defined as the intake found in a healthy population.[1,2] However, no first-world population has a sodium intake anywhere near the 2004/2005 IOM targets. Surprisingly, in his letter transmitting the 2013 report of the special panel of the IOM to the secretary of the Department of Health and Human Services,[11] Harvey Fineberg, president of IOM, stated "…the evidence linking sodium intake to health outcomes supports current efforts by the Centers for Disease Control and Prevention (CDC) and other authoritative bodies to reduce sodium intake in the U.S. population below the current average adult intake of 3400 mg/d." There is essentially nothing in the actual report to back that statement. Certainly, as already noted, there were no randomized controlled trials showing improved health outcomes as a result of reducing sodium intake from the current average (3450 mg/d) to 2300 mg/d, let alone 1500 mg/d.[6]

Here is where the evidence-based medicine (EBM) approach should have come to the rescue. In the absence of reliable evidence for benefit accruing to the intended reductions, the use of EBM would logically have led to the conclusion that no such reduction was justified. Despite Fineberg's assurances, the evidence summarized in the IOM's 2013 report does not support current efforts to reduce sodium intake in the US population below the average adult intake. However, it may be argued that absence of evidence supporting reduction does not, in itself, mean that doing so would not produce benefit. Thus, lacking hard evidence, one must then ask whether evidence of countervailing harm is associated with reduction in sodium intake below current population average values.

As it turns out, several recent population-based studies have shown persuasively that risk of both CVD and mortality follow U-shaped (or J-shaped) curves relative to sodium intake, with risk of mortality and CVD rising both as intakes drop below 3000 mg/d and as they rise above 7000 mg/d.[12–15] In this behavior, sodium acts exactly like most nutrients.[2,10] The reports concerned have dealt with patients with diabetes and CVD, as well as the general population. In all these studies, risk was lowest at a sodium intake in the range between 2800 and 6000 mg/d. Concordant with these findings is a report by Stolarz-Skrzypek et al,[16] who pooled 2 large prospective European studies, with up to 15 years' follow-up. They found a nearly 4-fold increase in cardiovascular mortality as sodium intake decreased from the highest tertile to the lowest and an approximate doubling of CVD events across the same decrease in intake. The long duration of observation in these studies virtually excludes reverse causality (ie, the possibility that the low-sodium-intake groups were such because of preexisting CVD). Finally, even in a large cohort of hypertensive patients (N = 398 419), lowering of blood pressure below "normal" values (130–139 mm Hg) prospectively resulted in increased risk of both mortality and end-stage renal disease.[17]

Although these papers[11–16] are relatively recent and would not have been accessible to the panel formulating the 2004/2005 guidelines, their findings were not actually without precedent. For example, Alderman et al,[18] in their 1995 publication of the Work-Site Hypertension Study, reported an approximate doubling of both CVD events and myocardial infarctions as sodium intake fell from the highest quartile to the lowest. In this study and in the pooled European[16] studies, the highest sodium intake was actually not particularly high. In the Work-Site Hypertension Study, the boundary of the fourth quartile began at about 4000 mg/d, and in the European studies, the third tertile began at about 4600 mg/d, well within the range of lowest risk in the population-based studies.[13–16]

Based on the criterion of the intake that requires least adaptation or compensation by the intact organism, intakes of 3000 to 5000 mg/d seem to be optimal.

These studies focused mainly on the rise in risk of adverse outcomes at sodium intakes *lower* than prevailing intakes in the general population. Risk associated with substantially *higher* intakes, of course, is not to be discounted or dismissed. Nevertheless, in the current context, the emphasis is on intakes lower than current population averages. Here, the evidence from all the studies cited indicates that there is probable increased risk of harm at intakes below 2800 mg/d in normal-weight adults. In addition, it is now becoming

clear also that sodium restriction in patients with congestive heart failure can actually worsen outcomes.[19] As an aside, one must ask, if it is not good for sick people, why would one think it good for the well? Thus, considering this diverse body of evidence, and as the most recent IOM report indicated, lowering population-level intake recommendations below current intakes, and certainly below 2300 mg/d, cannot be defended by available evidence.

MECHANISMS OF RISK

The reason for the observed increase in cardiovascular risk at low sodium intakes is not hard to discern. In fact, consideration of the underlying physiology shifts the focus constructively from the phenomena concerned to the relevant body control systems. It also directs attention to the criteria used for the establishment of a "normal" or "ideal" intake. As I have pointed out elsewhere,[20] and as others have suggested also,[21] perhaps the best criterion for the determination of "normal" (at least where it can be applied) is the intake that requires the least day-to-day compensation or adaptation by the intact organism—adaptation needed precisely for homeostasis at prevailing intakes. It is important to keep in mind that the ultimate physiological purpose of sodium intake is precisely the maintenance of blood pressure. Demonizing sodium is not only unsupported by evidence but is counterphysiological as well, as it ignores sodium's most basic function in mammalian bodies.

Adequate total body sodium content (and extracellular fluid sodium concentration) is necessary for maintenance of central blood volume and renal perfusion. For this reason, these variables are strongly defended by the body's homeostatic apparatus. On the low intake side, these defense mechanisms include salt hunger and reduction of urine and sweat sodium losses.[22,23] Together, these effects function to increase sodium intake and reduce losses and are mediated by the renin-angiotensin-aldosterone system (RAAS), which begins to be engaged as sodium intakes drop below 3000 mg/d in an average-weight adult.[24,25] And on the high intake side, the salt receptors on the tongue "flip" from positive to negative; that is, they produce an aversive sensation that is quite unlike the response of the other 4 basic taste sensors and that thereby tends to decrease intake of salty foods.[22]

There is no disagreement about the importance or necessity of homeostatic compensation, as intakes and losses from the body fluctuate widely and adjustments must be made to offset these perturbations. However, the RAAS, in defending central blood volume, exacts a toll on several body systems when it is continuously deployed. This toll has been shown to include increased risk of myocardial infarction and cardiac death.[26,27] The RAAS is, in effect, a "rescue" mechanism,[22] invoked in response to serious threat and hence necessary for survival. One might reasonably argue that an intake that did not constantly evoke such compensation would be not only more salubrious for the organism but, in fact, the one that is to be recommended. The recently published U-shaped risk curves[12–15] would place that intake in the range between roughly 3000 and 6000 mg sodium/d (Figure). Such a U-shaped curve is, in fact, the standard model used by the IOM for all nutrients.[2] It is, in a sense, reassuring to note that sodium is thus like most other nutrients in that there is potential harm at both extremes of intake.

As an illustration of the role of compensatory responses to reduced sodium intake, one may recall the observation of McCarron et al,[28] more than 30 years ago, showing that sodium intake in National Health & Nutrition Examination Survey (NHANES) was inversely correlated with blood pressure, not directly as conventional wisdom asserts. This finding has been widely ignored or criticized[29] as it simply could not be "correct." But, in fact, such a relationship is precisely what one would expect as an expression of extracellular fluid homeostasis. The sodium requirement, as with essentially all nutrients, varies considerably from individual to individual. Those with a higher sodium requirement unconsciously choose higher sodium intakes, not for taste, but for maintenance of central volume.[22] And those with lower requirements choose diets lower in sodium, again not for taste but as expression of the body's wisdom. The finding of high salt intake in NHANES in individuals with lower blood pressure is thus not so much paradoxical, as an expression of homeostasis at work.

In conclusion, it is worth noting that the approach to "normal" nutrition based upon minimizing the need for compensation, which I have articulated here, constitutes, in effect, an aesthetic criterion, one testable through the methods of physiology, but not easily susceptible of testing by the methods of EBM. But then, as noted above, EBM had not actually been used in setting recent sodium intake recommendations in the first place.

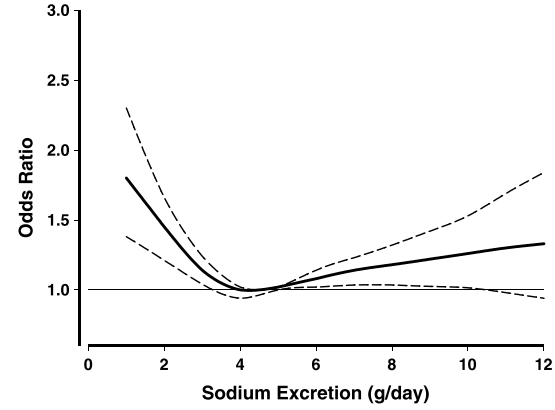

FIGURE. Observed risk of cardiovascular events plotted as a function of sodium intake, redrawn from the data of O'Donnell et al.[13] Note that risk rises both as sodium intake falls below 3 to 4 g/d and rises above intakes of 6 to 7 g/d and also that, in this dataset, the risk is numerically higher at lower rather than at higher intakes. Copyright 2014, Robert P. Heaney, MD. Used with permission.

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Commentary on Making Sense of the Science of Sodium

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Sodium reduction is an important component of a healthy dietary pattern to reduce cardiovascular disease risk. Numerous authoritative scientific bodies and professional health organizations have issued population sodium intake recommendations, all of which are at least 1000 mg/d lower than the current average American sodium intake of nearly 3500 mg/d. Recent research has called these recommendations into question, but a number of methodological issues may account for the inconsistency of results in observational studies examining the relationship between sodium intake and health outcomes. Health and nutrition professionals must consider that public health recommendations are made after weighing all of the evidence, including studies of greater and lesser strength of design and some with conflicting results. Nutr Today. 2015;50(2):66–71

SODIUM AND HEALTH

Blood pressure–related diseases (eg, coronary heart disease, stroke, heart failure, and chronic kidney disease) are leading causes of morbidity and mortality worldwide. The strong relationship between excessive sodium intake and high blood pressure forms the basis for recommendations to reduce sodium intakes. Numerous authoritative scientific bodies and professional health organizations have issued current population sodium intake recommendations, all of which are at least 1000 mg/d lower than the average American sodium intake of 3478 mg/d[1] (Table).

Sodium recommendations are based on results from animal studies, epidemiological studies, clinical trials, and meta-analyses of trials that demonstrate the adverse health effects of excess sodium intake. Feeding studies evaluating sodium intake ranging from 1500 to 2300 mg/d demonstrate blood pressure–lowering effects.[2,3] They also indicate that the relationship is direct and progressive, but nonlinear. For example, decreasing sodium intake by a given amount can reduce blood pressure more when starting sodium intake is lower, compared with when sodium intake is decreased by the same amount from a higher starting sodium intake.[4–6]

Several trials that evaluated long-term effects of sodium reduction on blood pressure demonstrated a consistent trend for fewer cardiovascular disease (CVD) events and/or mortality among those on a reduced sodium intervention.[7]

The impact of sodium reduction on blood pressure is greater in people with hypertension, but people with blood pressure in nonhypertensive ranges also benefit.[5] The GenSalt feeding study[2] and DASH (Dietary Approaches to Stop Hypertension)-Sodium are 2 trials that demonstrated blood pressure–lowering effects of sodium reduction in people without clinically defined hypertension. Furthermore, the effect of sodium reduction is greater in older versus younger study participants, and in the DASH-Sodium trial, effects of sodium reduction are increased when it occurs in tandem with good diet quality such as the DASH diet.[8]

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This study has no sources of support that require acknowledgment.

The authors have no conflicts of interest to disclose.

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DOI: 10.1097/NT.0000000000000086

In addition to a study that observed an inverse relationship of sodium intake and blood pressure,[9] other studies have suggested that sodium intakes in the range recommended by authoritative scientific bodies and professional health organizations are more harmful than higher sodium intakes.[10,11] These studies are inconsistent with findings from the majority of observational studies and randomized clinical trials. In addition, possible explanations for these inverse findings include measurement error from assessment tools that rely on self-report, reverse causality, and lack of adjustment for total kilocalorie intake or other nutrients that influence blood pressure.

In addition to the strong science on the effects of sodium on blood pressure, excess sodium has also been linked to kidney stones, asthma, osteoporosis, and gastric cancer.[12] Emerging research suggests that higher sodium intakes may be a risk factor for development of autoimmune diseases,[13] and preliminary research has also associated higher sodium intakes with increased adiposity and inflammation in healthy adolescents, independent of calorie intake.[14]

WHY POPULATION SODIUM REDUCTION?

Hypertension is a major public health problem affecting nearly 78 million US adults (about 1 in 3) and more than 40% of African Americans, and nearly half of those affected have uncontrolled hypertension.[15] Easing the burden of blood pressure–related disease warrants a comprehensive approach of both treating diagnosed hypertension and addressing underlying causes of high blood pressure in the population, such as a poor diet, to prevent future cases.

Average sodium intakes in the United States for those 2 years or older is 3478 mg/d, at least 1000 mg higher than any of the recommendations given by expert groups.

Given the significant number of people in the United States who would benefit from blood pressure lowering, reducing sodium intake in the American population is expected to have widespread benefits. People with hypertension, diabetes, and chronic kidney disease, as well as middle- and older-aged persons and African Americans, tend to exhibit a greater blood pressure response to reduced sodium intake than their healthier, younger, white counterparts.[16] These populations represent approximately 50% of the US

TABLE Recommendations for Sodium Intakes

Issuing Organization	Document	Guideline, mg/d	Notes
Domestic			
American Heart Association and American College of Cardiology	Guideline on Lifestyle Management to Reduce Cardiovascular Risk, 2013	• No more than 2400 • 1500 can result in even greater reduction in blood pressure	The guideline also states that even without achieving these goals, reducing sodium intake by at least 1000 mg/d lowers blood pressure
Institute of Medicine	Dietary Reference Intakes for Sodium, 2004	• 1500—Adequate Intake • 2300—Upper Level	
US Department of Health and Human Services and US Department of Agriculture	Dietary Guidelines for Americans, 2010	• <2300 • 1500 among persons who are ≥51 y old and those of any age who are African American or have hypertension, diabetes, or chronic kidney disease	
International			
Australia—National Health and Medical Research Council and New Zealand—Ministry of Health	Nutrient Reference Values for Australia and New Zealand, Including Recommended Dietary Intakes, 2006	• 460–920—Adequate Intake • 2300—Upper Level	
UK—National Institute for Health and Care Excellence	Public Health Guidance—Prevention of Cardiovascular Disease at the Population Level, 2010	• 2400 by 2015 • 1200 by 2025	
World Health Organization	Guideline: Sodium Intake for Adults and Children, 2012	• <2000	The WHO classifies adults as ≥16 y old
Average American sodium intake (population ≥2 y old) = 3478 mg/d—at least 1000 mg/d greater than any of the recommendations.			

population 2 years or older.[17] In addition, the blood pressure–raising effects of excess sodium are more pronounced in overweight and obese people, who comprise nearly 70% and 32% of the US adult and child/youth populations, respectively.[15] Furthermore, lowering dietary sodium can significantly blunt the rise in blood pressure that occurs with age.[4] This is important given that 90% of all Americans are expected to develop high blood pressure in their lifetime.[18]

INSTITUTE OF MEDICINE 2013 REPORT: WHAT IT DID AND DIDN'T SAY

A 2013 Institute of Medicine (IOM) report on Sodium Intake in Populations examined evidence published since 2003 concerning the potential benefits and adverse effects on health outcomes of sodium intake, particularly intakes of 1500 to 2300 mg/d.[19] The health outcomes examined included CVD, heart failure, myocardial infarction, diabetes, mortality, stroke, bone disease, fractures, falls, headaches, kidney stones, skin reactions, immune function, thyroid disease, and cancer but did not include intermediate outcomes such as blood pressure. The IOM committee did not conclude that blood pressure is not an acceptable surrogate for health outcomes, and blood pressure was characterized as a valid surrogate marker in a 2010 IOM report.[20] The Food and Drug Administration also recognizes blood pressure as a valuable biomarker for increased CVD risk.[21]

The IOM report generated much interest and debate, and some news stories contained inaccuracies and misrepresentations about its conclusions. Some stories suggested that sodium reduction was unnecessary or harmful, which likely created consumer confusion. An editorial authored by several committee members was published in the *Journal of the American Medical Association*,[22] and it summarized key points from the report including the following:

- There is a positive relationship between sodium intake of 2300 mg/d or greater and risk of CVD, supporting efforts to reduce current population sodium intakes.
- An association between sodium intake and CVD outcomes persisted after adjusting for blood pressure in some studies, suggesting that other factors (such as potassium intake) could mediate the relationship.

- Studies on direct health outcomes were of inconsistent quality and insufficient quantity to conclude whether sodium intakes less than 2300 mg/d were associated with greater or lesser risk for CVD. This statement does not convey that there is no benefit to further sodium reduction; rather, better-quality studies are needed before a more definitive conclusion can be drawn about these intake levels and direct health outcomes. Research is needed to determine associations between sodium intakes of 1500 to 2300 mg/d and health outcomes in the general population and in individuals with various health conditions (eg, diabetes and chronic kidney disease).

The IOM committee was not tasked with identifying a target sodium intake level (and the report explained that the heterogeneity of the available data precluded the committee from doing so), although its report fueled much debate on this topic. These debates highlighted the importance of focusing on the totality of the evidence base: Americans are consuming much more sodium than any public health recommendation, and lowering current intakes confers benefits on blood pressure and CVD.

A J-SHAPED RELATIONSHIP BETWEEN SODIUM AND CVD COULD RESULT FROM METHODOLOGICAL LIMITATIONS IN OBSERVATIONAL STUDIES

Recently published observational epidemiologic studies suggest that sodium intakes less than 2300 mg/d may increase risk of adverse outcomes, particularly in individuals with some health conditions (eg, diabetes and chronic kidney disease). These findings have led to disagreements among some in the public health and medical community about recommended sodium intake targets and ultimately about the importance of *any* reduction from current levels. Interpreting the results of observational studies is challenging because they can be highly dependent on the types of data collected (and not collected) and the statistical analytic approach. For example, a recent large observational study that assessed sodium intake by analyzing spot urines found greater risk of death and cardiovascular events with sodium intakes of less than 3000 mg/d (or >6000 mg/d),[11] whereas another recent long-term follow-up study that assessed sodium intake by multiple 24-hour urine collections documented a reduced risk of CVD with intakes less than 3000 mg/d.[23]

A number of methodological issues may account for the inconsistency of results in observational studies examining the relationship between sodium intake and CVD, many of which use data sets that were not specifically designed to test the relationships between sodium intake and CVD. Key issues include the following:

- Use of unreliable measures of sodium intake, such as spot urines. A single urine sample is not an ideal measure to predict health outcomes that occur decades later. Because of intraindividual variation in day-to-day sodium intake and diurnal sodium excretion, multiple 24-hour urine collections are the criterion standard for assessing sodium intake. These collections place a greater burden on the investigator and study participants, so inferior measures are often used. Recent evidence suggests that there are major variations in urinary sodium output in tightly controlled settings where dietary intake is known,[24] underscoring the importance of using multiple 24-hour urine collections for greater accuracy.
- Reverse causality, that is, inclusion of sick people who may have reduced their sodium intake in response to medical orders to limit sodium or who decreased overall food/calorie intake as a result of medication use or because of their disease state. The low sodium levels in these groups may not be the cause of their adverse health outcomes; instead, the low sodium intakes are prompted by the comorbid conditions.
- An insufficient number of cardiovascular events to definitively support the conclusion that a low-salt diet increases a person's chances of dying of heart disease.

Methodological issues are common. An empirical analysis of methodological issues in 26 cohort studies that found a mix of direct, inverse, null, and J-shaped associations between sodium intake and CVD detected an average of 3 to 4 methodological problems per study.[25] Many of these studies were recently included in a meta-analysis that concluded that sodium intakes less than approximately 2500 mg/d increase health risk,[10] but averaging lower-quality studies does not improve quality or strengthen inferences.

Methodological concerns limit the usefulness of these observational studies in setting dietary recommendations. It has been recommended that until well-designed cohort studies in a representative sample of the population are available, it remains appropriate to base recommended levels of sodium intake on the robust body of evidence linking sodium with elevated blood pressure and the few existing general population trials of sodium reduction on CVD.[25] Feasibility concerns explain the lack of randomized controlled trials showing improved health outcomes as a result of reducing sodium intake from current average intakes to recommended levels in the 1500- to 2300-mg/d range. It is unlikely that this evidence will be available soon because trials that examine clinical end points are notoriously expensive, requiring large numbers of participants and taking years to achieve an adequate number of study outcomes.

PHYSIOLOGICAL RESPONSE TO REDUCED SODIUM INTAKE

Dr Heaney states that continual deployment of the renin-angiotensin-aldosterone system in response to reduction in sodium intake leads to increased risk of myocardial infarction and cardiac death.[26] The renin-angiotensin-aldosterone system response is known to be greater with large and abrupt changes in sodium intake, but this may not be relevant to the gradual, sustained sodium reductions that are advocated.[27,28] It is not known with certainty how much and for what duration various degrees of sodium reduction

increase renin or aldosterone levels, or the clinical relevance of modest increases in plasma renin activity (whether acute or sustained) resulting from sodium reduction.[29] Renin also rises in response to blood pressure–lowering therapies that reduce CVD risk. There is greater certainty of the rise in blood pressure as a biomarker for future CVD than how the rise in renin affects CVD.[28]

FEASIBILITY OF ACHIEVING SODIUM INTAKE RECOMMENDATIONS

Education and counseling to promote individual behavior change are important, but reducing the sodium in the food supply is critical. Nearly 80% of Americans' sodium intake comes from sodium added to packaged and restaurant foods.[29] This food environment makes it challenging for people to attain recommended sodium levels unless they prepare much of their food from scratch, in contrast to trends in preferences for convenience foods and meals.[30] Less sodium pre-added to consumers' food purchases would help them decrease sodium intake and provide more control over their sodium intake.

While the numerous roles sodium plays in foods contribute to the complexity of the issue, sodium reduction can be achieved by industry and be accepted by consumers.[31] Some progress may occur by simply reducing salt, and beyond that, there is opportunity for innovation with new food manufacturing technologies and culinary techniques. The broad span of sodium content within similar food categories indicates that reduction to lower levels is feasible. A recent survey showed remarkable variability of sodium content in the same branded foods across countries. No single country consistently had the highest salt products; thus, regional taste preferences may not be responsible for the variation in salt content.[32]

Taste is an important driver of food choices, but is a malleable trait. Because most people eat considerably more salt than the body needs, salt preference appears to drive consumption rather than physiological need. Preferred levels are likely the result of the normative levels in the food supply. People who begin eating lower-sodium diets acclimate to prefer the lower levels, even eventually finding previously enjoyed foods to taste too salty. If the sodium they eat decreases gradually, they are usually not able to detect a difference in taste. Although more sensory research is still needed, it seems likely that consumers' preferences for salty foods will shift downward without a decline in taste enjoyment if sodium in the US food supply is reduced gradually in a stepwise fashion.[29] Stepwise reduction will likely take several years to achieve average American sodium intake that is within the range of what is acceptable to the public health community.

Standards for sodium levels in each food category would provide a level playing field for the industry, because all companies would have a common target as they reduce sodium in their products. This would help mitigate the potential for a given company to lose a competitive edge if they reduce sodium in their products while other companies do not. Such standards have been implemented in the United Kingdom, which recently observed a reduction in blood pressure, heart disease and stroke events, and deaths at the same time that sodium was reduced in the food supply by 15%.[33]

A comprehensive approach to cardiovascular health promotion and disease prevention must be multifactorial. In addition to a healthy diet, it should include regular physical activity, maintaining a healthy body weight, managing blood pressure and cholesterol, controlling blood sugar, and avoiding tobacco. Sodium reduction is not an isolated recommendation; it is an important component of a healthy dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, nontropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats.[6] Diets aligned with this pattern will likely have less sodium and more potassium, magnesium, and calcium than standard American diets. Combined, these factors will decrease CVD risk.

Successful sodium reduction requires action and partnership at all levels—individuals, healthcare providers, professional organizations, public health agencies, governments, and industry. Health and nutrition professionals must consider that public health recommendations are made after weighing all of the evidence, including studies of greater and lesser strength of design and some with conflicting results. To echo the conclusion of more than 30 leading nutrition scientists in a June 2014 statement: "Population-wide reduction of sodium intake is an integral approach to reducing CVD events and mortality in the United States."[34]

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