The dietary guideline for sodium: should we shake it up? No[1,2]

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ABSTRACT　　　The current US dietary guideline for sodium is a limit of 2.4 g/d or 6 g NaCl/d. This amount of sodium is far in excess of any physiologic need and is likely an essential though not by itself sufficient primary cause of hypertension as well as a contributor to many other cardiovascular and renal abnormalities. The evidence incriminating the current excessive consumption of sodium derives from epidemiologic, experimental, and interventional data, most of which support a threshold of ≈100 mmol/d for the harmful effects of sodium to be expressed. Although the current recommendation may not be low enough to go below that threshold, it is an appropriate and attainable goal for now. *Am J Clin Nutr* 2000;71:1020–6.

INTRODUCTION

The current US dietary guideline for sodium for the general public is no more than 2.4 g/d, equivalent to 110 mmol Na/d or 6.0 g NaCl. This is approximately two-thirds of the average dietary sodium intake of US adults (1), so that an overall average reduction of 40–60 mmol/d is being recommended. This recommendation has been widely made by multiple scientific and governmental agencies and is supported by a large body of evidence. However, an increasingly aggressive campaign was recently mounted against this recommendation in the belief that no benefit and possible harm will occur if it is accomplished. Some have argued that moderate sodium restriction is of no benefit and may possibly harm patients with hypertension, but most who object are concerned about its application to the large nonhypertensive population. In keeping with this position, the lobby for US salt producers, the Salt Institute, has petitioned the Food and Drug Administration to delete the sodium content from the labels of processed foods.

I will argue that the recommendation is correct for now and that the current labeling requirement for sodium is critically important to obtain the desired degree of dietary change. As will be noted, there are multiple benefits of moderate sodium reduction but I will focus primarily on hypertension because it is the condition most closely connected to dietary sodium.

PREVALENCE OF HYPERTENSION

A systolic blood pressure (SBP) >140 mm Hg and a diastolic blood pressure (DBP) of ≈90 mm Hg, or both, is universally considered to connote hypertension. Most who have elevations in both SBP and DBP, ie, ≈18% of the entire adult population, have essential, idiopathic, or primary hypertension (all synonymous

diagnoses). A much larger percentage of those aged >65 y, 50–70%, are hypertensive, but in about two-thirds of the elderly with hypertension only the SBP is elevated—a condition called isolated systolic hypertension (ISH). Although ISH is largely the consequence of age-related sclerosis of the large capacitance arteries, it may share some of the pathogenic mechanisms of essential hypertension, including the contribution of excess sodium. Moreover, the elevated SBP of the elderly responds even more to the removal of sodium from the diet or via diuretics than does the combined hypertension of the young.

There is no way to know whether the incidence of primary hypertension is increasing because this condition has not been easily diagnosed for even 100 y. However, the incidence clearly rises when primitive populations become acculturated, so that it is probable that the overall worldwide age-specific incidence has risen as westernization has spread. Although it is not known whether the incidence of primary hypertension within Western populations has changed, the incidence of ISH is increasing simply because life expectancy is increasing. Because most people now live beyond age 65 y, and because most people aged >60 y have hypertension, the overall incidence of hypertension will clearly continue to increase.

ROLE OF EXCESS SODIUM

After reviewing the available evidence relating sodium intake to hypertension, I conclude that excess dietary sodium is intimately involved in the pathogenesis of primary hypertension, playing a necessary but not sufficient role (2). The view that excess sodium intake induces hypertension reflects the belief of a large number of investigators, as summarized by Denton (3) in the last 87 pages of his book *The Hunger for Salt*. To quote Denton's nearly final words: "There are good grounds, but by no means a proven case, for suspecting excess salt intake, probably associated with reduced potassium intake, in the etiology of hypertension in Western-type communities."

My interpretation of these good grounds can be summarized as follows. Diets in nonprimitive societies contain many times the daily sodium requirement of adults, an amount that is beyond the threshold level needed to induce hypertension. Only part of the population may be susceptible to the deleterious effects of

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TABLE 1
Estimated diet of late Paleolithic humans compared with that of contemporary Americans[1]

Nutrient	Late Paleolithic diet (assuming 35% meat)	Current American diet
Protein (% of energy)	30	12
Carbohydrate (% of energy)	45–50	46
Fat (% of energy)	20–25	42
Polyunsaturated-saturated fat ratio	1.41	0.44
Fiber (g/d)	86	10–20
Sodium (mg)	604	3400
Potassium (mg)	6970	2400
Potassium-sodium ratio	12:1	0.7:1
Calcium (mg)	1520	740

[1] Data from reference 10.

this high sodium intake, presumably because these individuals have an additional renal defect in sodium excretion. Because almost everyone in nonprimitive societies ingests an excess of sodium beyond the threshold needed to induce hypertension, it may not be possible to show a relation between sodium intake and blood pressure in these populations. The absence of such a relation in no way detracts from the possible role of excess dietary sodium in causing hypertension.

It should be obvious that absolute proof of a direct causal role of sodium in hypertension will never be achieved because that would require continual observation of tens of thousands of subjects over 30–40 y in whom nothing but dietary sodium intake is varied and the variation in sodium intake is kept constant. Therefore, the evidence is circumstantial, but I believe it is strong enough to justify action. As noted by Rose (4): "The level of evidence appropriate to a particular decision depends on the consequences of making the wrong decision. For example, there is substantial evidence, but still well short of proof, that a reduction in national salt consumption leads to a somewhat lower mean blood pressure, with important expected health benefits. The change is safe and its cost minimal (except to a small but noisy section of the business community). The evidence for this policy is imperfect, but one may judge it to be sufficient."

Epidemiologic evidence

The first body of evidence is epidemiologic, including the following observations:

1) Primitive people in widely different parts of the world who eat little or no sodium have little hypertension and their blood pressure does not rise with age as it does in all westernized, industrialized populations (5). As an example, the Yanomamo Indians of northern Brazil, who excrete ≈1 mmol Na/d, have an average blood pressure of 96/61 mm Hg (6). Although their low blood pressure has been attributed to poor health and chronic disease or to an absence of stress, Chagnon, who spent many years among the Yanomamo, describes them as vigorous and frequently stressed (6).

2) The absence of hypertension may be related to other differences in lifestyle, but primitive people living under similar conditions who ingest large quantities of sodium develop hypertension. Page et al (7) noted that among the primitive men of the Qash'qai tribe of southern Iran, who ingest an average of 180 mmol Na/d, 18% were hypertensive.

3) When primitive people who are free of hypertension adopt modern lifestyles, including increased intake of sodium, their blood pressure rises and they become hypertensive. When rural Kenyan men moved to Nairobi, their sodium excretion increased from ≈60 to 110 mmol/d and their blood pressure rose significantly over a few months (8).

What has been noted in small groups over a short interval may also apply to the larger population over a much longer period of time. As stated by Denton (9): "Many major diseases are, in reality, 'maladies of civilisation'." These center on the changes in nutrition contingent on the very rapid transition from the predominantly hunter-gatherer life extant over 4–5 million y of hominid evolution to the radically different diet of urban industrialized societies during the past few hundred years. "The set points of appetites, satiation processes and endocrine-biochemical mechanisms were honed over these millions of years of jungle and savannah existence as hunter-gatherers, and they have been inept-maladaptive for diet and lifestyle inherent in Western urban existence. Humans are genetically programmed to a Paleolithic diet" (9).

The likely content of that diet has been contrasted with that of modern humans (10) (**Table 1**). Note that our carnivorous ancestors might have consumed 30 mmol Na/d so that human physiology evolved over millions of years in a low-sodium, high-potassium environment. Modern humans are ill-equipped to handle the current exposure to high-sodium, low-potassium diets. The different intakes of other nutrients shown in Table 1 could, of course, also be involved in the genesis of hypertension alone or in concert with the sodium content of the diet.

The marked increase in sodium intake, largely provided by salt added to processed foods, is so recent that genetic adaptations have not been possible. Moreover, as noted by Trowell (11), modern humans may never be able to adapt successfully to their high sodium exposure because evolutionary changes to preserve Darwinian fitness do not occur if new environmental factors produce disability or death only after the reproductive years are over. The major risks imposed by hypertension afflict mainly those >50 y of age so that adaptation to its pathogenic factors, including excess sodium intake, seems highly unlikely.

4) In large populations, significant correlations between salt intake and blood pressure and frequency of hypertension have been found in most (12–15) but not in all (16) studies. The strongest data come from the Intersalt study, which measured 24-h urine electrolytes and blood pressure in 10 079 men and women aged 20–59 y in 52 places around the world (17, 18). For all 52 centers, there was a positive correlation between sodium excretion and both SBP and DBP, and an even more significant association between sodium excretion and the changes in blood pressure with age. Few populations were found whose sodium intakes were ≈100 mmol/d, the likely threshold above which an effect of sodium on blood pressure is observed. However, the virtual absence of either hypertension or of a progressive rise in blood pressure with advancing age in populations with an average sodium ingestion <100 mmol/d in both the Intersalt study (15) and in separate population studies (6–8) supports the concept of a threshold.

A threshold for sodium

From these data, and others taken from individual studies of different populations with low sodium intakes, little if any hyper-

TABLE 2
Four trials of prevention of hypertension by dietary sodium reduction[1]

Trial name and reference	Duration	Reduction in Na⁺	Incidence of hypertension	
			Intervention group	Control group
	y	*mmol/d*	*%*	
Primary prevention (27) (*n* = 201)	5.3	31	8.8	19.2
Hypertension Prevention Trial (28) (*n* = 196)	3	34	26.9	38.7
Trials of HT Prevention I (29) (*n* = 744)	1.5	44	8.6	11.3
Trials of HT Prevention II (30) (*n* = 594)	4	40	38.1	44.4

[1] Sodium excretion for 8-h urine collections were multiplied by 3. HT, hypertension.

tension is noted until the average sodium intake is > 100 mmol/d, well above the average intake in most industrialized societies. Thus, I believe that when sodium intake exceeds the limits that human physiology evolved to handle, sodium becomes an essential factor in the pathogenesis of hypertension. Obviously, sodium excess must act in concert with other mechanisms, in particular, an inability of the kidneys to excrete all of the excess sodium. This renal defect may be genetic or acquired. There is considerable evidence that an acquired defect, congenital oligonephropathy, associated with low birth weight resulting from intrauterine growth retardation can serve as the major collaborator with excess sodium intake to give rise to hypertension (19).

The likely presence of a threshold implies that attempts to either prevent the development of hypertension or to reduce its degree once it has developed are likely to fail unless average sodium intake is kept below 100 mmol/d for prolonged periods. Other than in some animal models noted in the next section, few such long-term studies have been performed; admittedly, less-conclusive epidemiologic evidence is the main support for the concept of a threshold.

Experimental evidence

In animals

Although a large body of data attests to a prohypertensive role for excess sodium in rats and other small species, its relevance to the human condition remains questionable. However, a study in chimpanzees, the animal species closest to humans, with 98.4% genetic identity, provides the strongest experimental evidence now available for a direct causal role of excess sodium.

Denton et al (20) studied 22 chimpanzees consuming their usual low-sodium, high-potassium diet of vegetables and fruit while in long-standing, socially stable groups. After 1 y of baseline observations with no other changes in diet or social conditions, sodium was added in increasing amounts from 5 to 10 to 15 g/d over 20 mo to half of the chimpanzees, while the other half had no change in sodium intake. In the 10 chimpanzees who ingested at least half of the added salt, blood pressures started to rise progressively, increasing at the end of 84 wk by 33/10 mm Hg above baseline. When the extra salt was no longer provided, blood pressure promptly fell and remained at the same level measured at the beginning of the study. No change in blood pressure occurred in the control animals. As Denton (9) later observed: "These results show unequivocally that increased salt intake causes a large rise in blood pressure in chimpanzees. Thus, in the absence of stress and change of K or Ca status, the increase of salt intake to the range of intake in human societies (100–280 mmol/d) was the sole cause of a

large rise of blood pressure in the species phylogenetically closest to humans."

In humans

Unfortunately, such well-controlled studies cannot be done in people over long periods. Short periods of increased salt intake have been shown to raise blood pressure in normotensive subjects (21, 22). High sodium intake has been shown to activate many pressor mechanisms, including an increase in intracellular calcium (23), a rise in plasma catecholamines (24), a worsening of insulin resistance (25), and a paradoxical rise in atrial natriuretic peptide (26). As will be noted, there are other effects more pronounced in those who are most sensitive to the pressor effects of sodium, ie, in those who are sodium sensitive.

A series of randomized, controlled trials looked at the ability of moderate dietary sodium reduction to prevent or at least delay the development of hypertension (ie, blood pressure > 140/90 mm Hg) in subjects with high-normal blood pressure (ie, 130–140/80–90 mm Hg) over 3–5 y (27–30). With relatively small reductions in average sodium intake, from 31 to 44 mmol/d, statistically significant differences in the percentages of subjects who developed hypertension were seen in all of these trials (**Table 2**).

Of interest, those subjects in phase II of the Trials of Hypertension Prevention who had the AA angiotensinogen genotype had a 43% decrease in the incidence of hypertension whereas those with the GG genotype had no decrease at all (31). These data, along with those noted below, point to the possibility in the near future of more accurately predicting the response to changes in sodium intake.

A particularly relevant trial documented the ability of a moderate decrease in sodium intake to prevent or delay the reappearance of hypertension in the most vulnerable population, the elderly (32). In this trial, successful antihypertensive therapy was stopped and the 975 patients aged 60–80 y were randomly allocated to 4 regimens: sodium restriction, weight reduction, both sodium restriction and weight reduction, or nothing, ie, usual care. Over the next 30 mo, the patients were monitored for the return of hypertension. Despite achieving only a 40-mmol/d decrease in sodium excretion, those assigned to sodium reduction had a 50% decrease in the return of hypertension compared with those receiving usual care.

Although long-term sodium-restriction studies that start with infants and children are not feasible, a short-term study was done. A 6-mo study in almost 500 newborns showed that the half whose sodium intake was reduced by ≈50% had a 2.1-mm Hg lower SBP at the end of 6 mo than did the half who had normal sodium intakes (33). Among the 35% of the participants who could be traced 15 y later, those originally receiving the low-sodium diet had a 3.6/2.2-mm Hg lower blood pressure (34).

Sodium sensitivity

Almost everyone living in westernized societies ingests a high-sodium diet. Although most have blood pressures above optimal values, only about half will develop overt hypertension, which suggests a variable degree of blood pressure sensitivity to sodium. Obviously, both heredity and interactions with other environmental exposures may be involved. After Luft et al (35) and Kawasaki et al (36) described varying responses of blood pressure to short periods of low and high sodium intake, numerous protocols have been used to determine so-called sodium sensitivity. The most extensive studies have been by Weinberger et al (37), who defined sodium sensitivity as a decrease ≥10 mm Hg in mean blood pressure from the value measured after a 4-h infusion of 2 L normal saline compared with the value measured the morning after 1 d of a 10-mmol Na/d diet during which 3 oral doses of furosemide were given at 1000, 1400, and 1800. Using this criterion, these researchers found that 51% of hypertensive persons but only 26% of normotensive persons were sodium sensitive. They noted that sodium sensitivity has a typical bell-shaped distribution with a shift to the right in those who are hypertensive. These investigators observed a further shift with increasing age in normotensive persons and a greater shift in hypertensive persons (38).

Several mechanisms have been proposed to account for increased sodium sensitivity (39). Perhaps the most obvious is a lesser responsiveness of the major mechanism responsible for sodium retention and vasoconstriction, the renin-angiotensin aldosterone (RAA) system. When sodium is restricted, the degree of fall in blood pressure is closely related to the degree of activation of the RAA, as recently reaffirmed in a study of black and white hypertensive people who were prescribed a 10-mmol Na/d diet (40). The black subjects, whose RAA system was less responsive, had a 22/10-mm Hg decrease in blood pressure, whereas the more responsive white subjects had a 17/6-mm Hg decrease.

Whatever the mechanism, those who are more sodium sensitive are also more likely to have several abnormalities that increase their risk of cardiovascular and renal consequences of hypertension (41). These include increased insulin resistance (42); proteinuria, as a likely consequence of increased glomerular capillary hydraulic pressure (43); and left ventricular hypertrophy (44), which may in turn reflect a nondipping of blood pressure during sleep (45). All of these and more features of sodium sensitivity strongly support the need to reduce sodium intake in such patients, not only to achieve a greater decrease in blood pressure, but also to protect against damage to the various vital organs that accompanies sodium sensitivity.

Trials of sodium restriction

The last bit of evidence favoring universal, moderate sodium restriction derives from clinical trials of both normotensive and hypertensive subjects. These trials always measured changes in blood pressure and a few looked at other end points, such as left ventricular wall thickness.

By definition, subjects who are sodium sensitive would be more likely to have a greater decrease in blood pressure when sodium intake is reduced. Most tests of sodium sensitivity involve short-term, abrupt changes in sodium intake but their results have been found to fairly accurately predict the long-term effects of less profound changes in dietary intake (46). Unfortunately, the multitude of trials of dietary sodium restriction reported over the past 30 y rarely, if ever, preclassified the

subjects according to their sodium sensitivity. Because I do not believe there is danger or discomfort from moderate sodium restriction, I do not recommend that a test for sodium sensitivity be done before a lower-sodium diet is prescribed. Nonetheless, the efficacy of sodium restriction in lowering blood pressure would certainly be better delineated if subjects entering such trials had been preclassified as to their responsiveness to the abrupt changes in sodium intake that are used to define sodium sensitivity.

Rigid dietary sodium restriction was first used, often in the form of a rice diet, to lower blood pressure when little else was available (47). Starting in the late 1960s, more moderate sodium restriction was tested; >100 trials were subsequently published. At least 4 meta-analyses of these trials have appeared (48–51). Although these differ in the numbers of trials included and even more in their interpretation of the results, the results agree that blood pressure falls ≈5/2 mm Hg in hypertensive and 2/1 mm Hg in normotensive subjects when they restrict dietary sodium intake to ≈100 mmol/d. The presentation of data by Cutler et al (50) from 32 trials, 22 in hypertensive persons, which met fairly rigid criteria, shows a dose-response relation.

The analysis by Midgley et al (49) showed a greater effect in the elderly, who have been well characterized as being more sodium sensitive. The overall decrease in blood pressure provided by a 100-mmol/d reduction in sodium intake was 6.3/2.2 mm Hg in the 17 trials of older hypertensive subjects, but only 2.4/−0.1 mm Hg in the 11 trials of younger hypertensive subjects. None of the analysts separated subjects by race, but it is likely that black individuals, who are more sodium sensitive, would also achieve relatively greater decreases in blood pressure.

Problems with trials

As shown by Law et al (48), the full effects of sodium restriction are usually not obvious for ≥5 wk; yet many of the trials included in these meta-analyses were only 10–14 d long. As noted by all analysts, in the majority of long-term trials, relatively small decreases in sodium intake were measured so that the full effect of sodium restriction may not have been recognized. As noted by Grandal et al (51), "There were a considerable number of short-term studies of high reduction of sodium intake and long-term studies of low reduction of sodium intake, but very few short-term studies of low reduction of sodium intake and long-term studies of high reduction of sodium intake."

Even in the analysis by Cutler et al (50), which excluded most of the trials included by the other analysts because of defects in design and reporting, the median reduction in sodium intake was 74 mmol/d, bringing the average intake near but rarely below the threshold load that I believe must be breached in order for the full antihypertensive effect of sodium restriction to be manifested.

Difficulty in lowering the dietary intake of free-living subjects to <100 mmol/d for prolonged periods has been noted repeatedly (52). In the trials of hypertension prevention shown in Table 2, reductions of only 30–45 mmol/d were accomplished despite intensive counseling and encouragement, close follow-up, and careful monitoring. As will be noted, the problem revolves around the difficulty in removing the largest component of dietary sodium, that which is hidden in processed foods (53). With current labeling laws, it should be easier to avoid such high-sodium foods and thereby further decrease sodium intake.

EFFECTS OF SMALL CHANGES IN BLOOD PRESSURE

Even the small reductions in blood pressure provided by the relatively modest degrees of sodium reduction previously accomplished could provide significant benefits. Here again, the situation was nicely portrayed by Rose (54): "Supposing that some dietary measure, such as moderation of salt intake, were able to lower the whole blood pressure distribution, we may estimate how the potential benefits might compare with what is currently achieved by the 'high-risk' strategy of detecting and treating hypertension…. We may estimate that all the life-saving benefits achieved by current antihypertensive treatment might be equaled by a downward shift of the whole blood pressure distribution in the population by a mere 2–3 mm Hg. The benefits from a mass approach in which everybody receives a small benefit may be unexpectedly large." A population-based intervention that lowered DBP distribution by 2 mm Hg would reduce the overall number of hypertensive persons by 16% and could reduce the number of heart attacks by 9% and the number of strokes by 15% (55).

ADDITIONAL BENEFITS OF SODIUM RESTRICTION

Even if blood pressure is not lowered, a decrease in sodium intake could provide multiple other benefits. Evidence supports the following effects: regression of left ventricular hypertrophy (56), reduction in proteinuria (57), reduction in urinary calcium excretion and risk of renal stones (58), decrease in osteoporosis (59), protection against stomach cancer (60), protection against stroke (61), enhancement of the antihypertensive effect of other antihypertensive agents (62), and a decrease in diuretic-induced potassium wastage (63).

PUTATIVE DANGERS OF SODIUM RESTRICTION

Extreme degrees of sodium restriction, down to 10–20 mmol/d, have been shown to set off many potentially harmful hormonal and lipid effects that are not seen with more moderate sodium restriction (64). For example, brief, severe sodium reduction raises plasma concentrations of catecholamines (65) whereas prolonged, moderate restriction lowers plasma catecholamines (66). Moreover, if even moderate sodium restriction were accomplished by reduced consumption of grains and dairy products, there could be a decrease in the consumption of calcium, iron, magnesium, and vitamin B-6 to intakes below those recommended. In a review of all current evidence, Morris concluded that "Present data are inadequate for determining the potential nutrient alterations of a broad prescription of sodium restriction" (67). However, the potential problem could easily be avoided if the amount of salt added during processing were gradually reduced.

Even though there are no significant perturbations seen with the degree of sodium restriction that is both feasible and recommended, 2 publications have claimed to expose an increase in myocardial infarctions (68) and cardiovascular mortality (69) in people whose sodium intake was in the lowest quartile within 2 populations. These reports have been roundly criticized but they demand consideration.

Myocardial infarction

Alderman et al (68) reported a 4-fold increase in myocardial infarctions over an average 3.8-y follow-up among treated male hypertensive patients whose initial 24-h urinary sodium excretion was in the lowest quartile, averaging 65 mmol/d. No association was noted for myocardial infarction in women or for stroke in either sex. These data may be faulted for numerous reasons. The number of events studied was small, only 46 myocardial infarctions in all 1900 men, 22 occurring in the 483 in the lowest quartile of urinary sodium excretion. Blood pressures were highest in the men in the lowest quintile of sodium intake. The measure of sodium intake, a single 24-h urine sample, is an inadequate measure of usual intake. Moreover, the one urine sample analyzed may be even less of a reflection of usual intake because it was collected after 4–5 d of avoidance of high-salt foods. No ascertainment of actual sodium intake during the 3.8-y follow-up was attempted so there is no way to know whether the one urine specimen truly reflected usual intake. The dietary intakes of sodium in those who experienced a myocardial infarction may have been well below that generally recommended, thereby activating some of the adverse effects of very low sodium intake noted earlier.

Cardiovascular mortality

As part of the first National Health and Nutrition Examination Survey (NHANES I), which involved a representative sample of 20725 US adults during 1971–1975, a medical examination and one 24-h dietary recall were performed for 11348 of the subjects. In 1992, the status of these 11348 subjects was ascertained: 3923 had died, 1970 from cardiovascular causes. When cardiovascular mortality was correlated with quintiles of sodium and energy intake, there was an inverse association, ie, higher mortality rates among those whose diet was lowest in sodium or energy. However, mortality was directly associated with the sodium-energy ratio, ie, there was higher mortality in those with the highest ratio of sodium to energy.

These results contradict a great deal of evidence that mortality is higher in people who consume the most energy. But the major problem with the data is the use of a notoriously inaccurate 1-d dietary recall to estimate usual sodium and energy intakes. A manifestation of this inaccuracy is the finding that those men who said they consumed 6174 kJ/d (1473 kcal/d) weighed the same as those who said they consumed 12180 kJ/d (2937 kcal/d). Moreover, according to other analyses of the NHANES I data (70), those in the lowest quintile of sodium intake had more preexisting cardiovascular disease, had more hypertension, smoked more, had the lowest body weights, and were more likely to be black—all of which are associated with increased mortality rates.

More recently, when the same data from NHANES I were analyzed more carefully, a lower sodium intake was strongly and independently associated with a reduced risk of cardiovascular disease, particularly stroke, and all-cause mortality in overweight persons, whereas dietary sodium intake was not significantly associated with cardiovascular risk in nonoverweight persons (71). Two additional papers have noted lower cardiovascular mortality rates in people with lower urinary sodium excretion. In the Scottish Heart Study, there was a direct association between urinary sodium excretion and the incidence of coronary events in the 5875 women studied, but no association in the 5754 men (72). A strong, direct correlation between urinary sodium excretion and stroke mortality was found in data pooled from 17 countries (73). I hope that large-scale, long-term prospective data will become available to settle the issue.

In the meantime, the data from Alderman et al (68) have been used as the primary argument by the Salt Institute to petition the removal of sodium from food labels and all restrictions on sodium intake from the US dietary guidelines.

The larger body of evidence described in this article and accepted by most experts in this field supports the likelihood that moderate restriction of dietary sodium intake will provide far more benefits than any possible harm. I agree with the conclusion reached by Kotchen and McCarron (74) on behalf of the American Heart Association Nutrition Committee: "For the general population, the AHA recommends that the average daily consumption of NaCl by adults not exceed 6 g. There is no evidence that limiting NaCl consumption to 6 g per day poses any health risk."

THE NEED FOR LESS ADDED SODIUM IN PROCESSING

As noted, the best that most people can reach is about a 25% reduction in sodium intake, which is often well above the recommended 2.4-g/d level. Because 75% of sodium intake comes from that added in food processing and because most of that sodium is not obvious by taste, the only practical way to reduce intake in the immediate future is to have people read food labels, thereby recognizing the sodium content of foods that is not obvious in any other way. Because this requires active participation by the consumer, food processors should be encouraged to gradually reduce the amount of sodium they add, thereby making it easier for all to moderately reduce their sodium intakes because the preference for sodium is quickly reduced when less sodium is ingested (75). In the United Kingdom, where more aggressive attempts have been made to gain the cooperation of food processors, the Ministry of Agriculture, Fisheries and Food of the Department of Health recently issued this statement: "The Government accepts that there is a large body of authoritative opinion which favours a general reduction in salt consumption. In consequence Ministers have asked officials to initiate discussions with the food industry to explore the scope for broadening public choice in and reducing the salt content of, manufactured foods" (76).

CONCLUSION

The evidence supporting the need for a reduction in dietary sodium intake is convincing and supports the appropriateness of the current US dietary guideline. As noted, this guideline may not be set low enough to prevent the development of hypertension, but it is almost certainly as much as is attainable under current conditions. If this goal can be reached, perhaps even greater reductions will be feasible in the future, particularly if food processors are cooperative.

To deny the benefits of moderate sodium restriction because there is no absolute proof of benefit or safety is akin to faulting John Snow for demanding that the Broad Street pump be closed without knowledge that it was the source of the *Vibrio cholera* responsible for the London epidemic. Snow was able to show that cholera was spread by polluted water only because he could calculate attack rates according to their proximity to the pump. We have even more substantial evidence incriminating excess sodium and we should be as aggressive as John Snow in attempting to stop the epidemic of sodium-related cardiovascular disease.

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