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Science trumps politics: urinary sodium data challenge US dietary sodium guideline[1,2]

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For the past 3 decades, the US government has promoted a policy of reduced dietary sodium intake as the principal nutritional means of reducing blood pressure and its attendant cardiovascular disorders in adults. Early on, this policy targeted at-risk individuals such as people with chronic arterial hypertension; however, in the past decade it has been applied to the population at large. Despite a litany of well-intended strategies from mandatory sodium labeling to extensive educational and social marketing efforts, there is little evidence that sodium intake has changed. In fact, some advocates of the policy have argued that sodium intake actually has increased, reaching extreme levels in some people (1). The failure of the government's efforts has been typically attributed to the food industry's excessive use of sodium in their products (1). Both the application of such a government policy to the entire population and the simplistic assessment that its failure to date can be attributed to the food industry's reluctance to provide lower sodium foods belie the scientific complexity of the issues, including sodium's role in health and disease.

In this issue of the Journal, Bernstein and Willett (2) provide a valuable analysis of 24-h urinary sodium (UNaV) data extracted from the medical literature published between 1957 and 2003. Their findings from the 38 US studies that met rigorous search criteria and involved 26,271 people confirm and extend the conclusions we published a year ago (3). Our analysis involved 19,151 people from 33 countries and 62 survey sites between 1984 and 2008. Like that of Bernstein and Willett, our analysis revealed a remarkably narrow range of UNaV across very diverse populations and eating habits, without the extreme levels often purported to exist by advocates of lower sodium intake (1) and no evidence of a change over time. The latter was best shown by the data of the UK Food Standards Agency between 1986 and 2008, which we noted (3) offered no evidence that an intense social marketing effort begun in 2005 had been successful.

One possible explanation, first raised by our report last year (3), is that human sodium intake is a parameter that even the most well intentioned public policy cannot modify in most people. An extensive body of basic science research, dating from Richter's seminal observations (4), has characterized an integrated network of peripheral hormonal signals interfaced with complex neural networks specific to regulating sodium intake of experimental animals (5). Although those basic research findings have not as yet been extrapolated to humans, they should not be completely ignored because they may yet provide a model of what is feasible in humans.

The current report extends our observations by documenting that, likewise, all the efforts in the United States over the past 3 decades have had no effect on the population's sodium intake. An alternative possibility for the stability of sodium intake is that sodium has been largely consumed in association with food intake, motivated by hunger and appetite. To the extent that caloric intake has been stable over populations and decades, so too has sodium. Thus, a potential benefit of reducing food sodium content would be a concurrent reduction of sodium. Working against that theoretical outcome, however, is the reality that over the millennia, before the introduction of processed foods, sodium was added to foods at the time of preservation, cooking, or consumption. An individual in our society has the identical options today as the food industry moves to offering more products whose ratio of calories to sodium is increased (ie, lower sodium content per serving). This individual choice could abrogate any effect on average sodium intake in society as these data indicate has happened.

Regardless of why sodium intake has been so stable, the data of Bernstein and Willett (2), as well those of McCarron et al (3), suggest that it is not a readily modifiable nutritional parameter for the population at large. Furthermore, a substantial body of research in humans provides evidence as to why this latest attempt to modify the general population's sodium intake is doomed to failure. Sodium has a critical role in extracellular fluid volume regulation as well as being of fundamental importance in cellular function across virtually all organ systems. Thus, it is unlikely that as an organism, humans would have evolved without the development of failsafe mechanisms to ensure sufficient sodium availability. Sodium is 1 of only 3 nutrients whose excretion in urine is recognized as regulated; water and glucose are the other 2. Consequently, deficits in these nutrients elicit immediate, potent, counterregulatory physiologic responses.

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The renin-angiotensin-aldosterone system (RAAS) lies at the heart of such protective mechanisms in humans. The reduction of sodium intake below ≈120 mmol/d (2760 mg/d) in humans activates the RAAS cascade (6, 7), producing angiotensin II (AII), the most potent vasoconstrictor in humans. Among the many actions of AII is the stimulation of aldosterone secretion by the adrenal cortex, whose subsequent renal action is to promote sodium reabsorption by the kidneys. Aldosterone is so effective that it can eliminate virtually all filtered sodium from the urine when sodium intake is not sufficient to meet physiologic needs. The intuitive sense of such a hormonal system that is stimulated when sodium intakes fall below 120 mmol/d (2760 mg/d) is clear: sodium is the backbone of extracellular fluid and ensures adequate blood volume, arterial pressure, and ultimately, organ perfusion. Thus, a recommendation below 120 mmol/d (2760 mg/d) assumes *1*) that the basic biology of the organism should be ignored; *2*) that intakes below this threshold are not potentially harmful (8); *3*) that the food industry can produce foods of such reduced sodium content and its attendant changes in texture, taste, and stability that people will change their eating habits; and *4*) that people will desire to make these changes rather than see them as unwarranted constraints on a fundamental element of human behavior, ie, choosing the foods they eat.

The extensive 24-h UNaV data from the United States that Bernstein and Willett (2) report in this issue, combined with our remarkably similar findings across 33 societies worldwide (3), add a new dimension to the public dialog surrounding current and proposed government guidelines calling for population-wide reductions in dietary sodium (9, 10). These combined data from >40,000 people, roughly half of whom live in the United States, spanning 1957–2008, document that daily human sodium intake fluctuates within a very narrow, reproducible range of between 120 mmol/d (2760 mg/d) at the lower end and 210 mmol/d (4830 mg/d) at the upper limit. Given the time span involved and the changes in the US food supply and that in other societies that have occurred over this 50-y period, it is highly unlikely that this range of human sodium intake is a fact that should be dismissed. The average 24-h UNaV that the combined data describe, ≈155–160 mmol/d (3565–3680 mg/d), is 50% higher than the current upper limit of the US government recommendation of 100 mmol/d (2300 mg/d) (9), and 2.5 times higher than the proposed 2010 guideline of 1500 mg/d (10). In fact, as noted in our earlier publication (3), the US government's largest randomized trial (11) and the only double-blind, randomized, controlled cross-over trial of lower sodium intake in mild hypertension (12) reported similar lower limits of achievable sodium intake of ≈120 mmol/d (2760 mg/d), which is well above the upper limit currently recommended and nearly identical to the absolute lower limit defined by the 24-h UNaV data. The significance of these data is that they provide a stark perspective on just how dramatic a change in our food supply would have to be executed, and the commensurate alteration in human behavior, to meet even the current guidelines (10), let alone the proposed new guidelines (9).

The analyses of extensive measurements of 24-h UNaV, which these 2 reports have collected from the medical literature over the past 5 decades, are compelling. They provide plausible, scientific evidence of a ''normal'' range of dietary sodium intake in humans that is consistent with our understanding of the established physiology of sodium regulation in humans. This scientific evidence, not political expediency, should be the foundation of future government policies, thus respecting the known and unknown scientific complexities surrounding sodium's role in health and disease. Guidance for sodium intake should target specific populations for whom a lower sodium intake is possibly beneficial. Such an approach would avoid broad proscriptive guidelines for the general population for whom the safety and efficacy are not yet defined (8). An appropriate next step is not to lower the sodium guideline further. Rather, the scientific community should commit to the continued evolution of the science underlying sodium's role in normal and abnormal human physiology.

DAM has consulted with the food industry and the Salt Institute in the past. TBD has consulted with the European Union's Salt Committee as well as the Comité des Salines de France in the past. EMS had no conflicts of interest to report.

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