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In Reply—Population-wide Sodium Reduction: Reasons to Resist

A discussion of salt intake always generates a vigorous debate. In some part, controversy occurs because of the years of study generally required to determine the effect of dietary modifications on morbidity and mortality. Perhaps as a result, association studies have proliferated in this area to replace randomized controlled trials. However, the field of nephrology in particular has learned a series of "tough lessons" as a result of relying on observational data and is now emphasizing the need for randomized controlled studies to dictate clinical practice. With this background, DiNicolantonio et al bring forward several interesting observations that merit additional comment.

DiNicolantonio et al claim that reducing salt intake may increase renin, aldosterone, adrenaline, noradrenaline, cholesterol, and triglyceride levels. Although the examination of these associations of dietary sodium intake with the renin-angiotensin-aldosterone system is of interest, the primary consideration should be hard end points such as mortality and not just an association with renin levels. The potential issue of the effect of a low-salt diet on insulin levels is also mentioned, but perhaps caloric intake in our modern society should be considered the primary cause of hyperinsulinemia and not a low-salt diet. In addition, clinical outcomes—not necessarily associations with insulin levels—should be the preferred end point when determining the optimal mode of sodium management in patients who have (or are at risk for) diabetes mellitus.

DiNicolantonio et al quote a meta-analysis by Graudal et al[1] that derived data from sudden and large reductions in salt intake and ignored contrary evidence.[2] The underlying rationale for the inconsistent findings in the report by Graudal et al is that a drastic reduction in dietary sodium intake can lead to unfavorable metabolic and neurohormonal alterations, which could promote insulin resistance, lipid abnormalities, and increased cardiovascular risk through a compensatory activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (activation that is proportional to the degree of sodium reduction).[1,3] The theory seems to be valid only for an extremely low sodium intake[4] and not for the 2300 mg/d recommended for the general population.[5]

Moderate salt reduction in the United States is unlikely to have a major impact on iodine status or exercise-associated hyponatremia. Most salt in the diet is derived from processed foods, and the salt used in food processing in the United States is typically not iodized.[6] Encouraging the food industry to use iodized salt during processing or alternative fortification strategies is more likely to yield optimal iodine status in Americans.[7,8] Although careful population monitoring for iodine status is required, countries that mandate iodization of salt used in processing and for personal consumption have found that lower salt intakes do not compromise iodine status.[9] In contrast to the statement by DiNicolantonio et al, most cases of exercise-associated hyponatremia are due to excessive water consumption rather than a lack of salt in the diet.[10-12]

Finally, in regard to the statement on congestive heart failure (CHF), our review excluded studies with CHF as the main outcome. However, a caveat in the discussion section of our article[13] stated that clinicians should be aware that patients with severe CHF take multiple medications that inhibit or block the renin-angiotensin-aldosterone system and thus may not benefit from salt restriction.[14] Less rigorous targets for salt reduction may be appropriate for certain groups of patients with CHF or multiple comorbid conditions.

It is currently impossible for clinicians to provide strictly individualized dietary recommendations to the general population because the approach would require them to have already outlined nutrigenomic interactions and underlying genetic susceptibility

traits, the identification of which, at present, remains an evolving and challenging field of research.[15] Based on the current evidence, and until the response of most individuals to dietary manipulations can be fully distinguished, the hemodynamic benefits of a reduced-sodium diet appear to far outweigh any theoretical metabolic or hormonal perils following dietary salt reduction.[16]

There is evidence that supports a reduction in salt intake for the general population, including outcome trial evidence on cardiovascular disease.[17-20] Historically, salt intake was low in preindustrial societies, and intakes have only reached high amounts in the past century. The food industry has contributed to this increase.[21-23] Of all public health policies, comprehensive salt reduction programs represent some of the most practical and cost-effective strategies for chronic disease prevention,[24,25] but they need the cooperation of the food industry, multiple levels of government, and other diverse stakeholders.[25,26] If this goal was accomplished, the projected health and economic benefits of preventing hypertension, stroke, myocardial infarction, and early mortality would be considerable.[27,28] Consequently, we would like to reinforce the concept that the consumption of a low-sodium/high-potassium diet is a critical strategy for promoting cardiovascular health and for preventing and treating hypertension and cardiovascular disease in the US population.

Kristal J. Aaron, MSPH
Paul W. Sanders, MD
University of Alabama at Birmingham

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