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Publication Date	2016-12-15
Publisher	Massachusetts Medical Society
Repository DOI	10.1056/NEJMc1612304

Dietary Sodium and Cardiovascular Disease Risk

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To the Editor:

Cogswell et al[1] argue that the reported association between low sodium intake and increased cardiovascular risk does not fulfill the criteria for causality, and they therefore conclude that low sodium intake should be recommended. We wish to call attention to several issues in their assessment of the available data.

First, the criticism by Cogswell et al. with regard to fasting urinary sodium estimates of group-level sodium intake is misleading, because this method has been validated in a large international study (intraclass correlation vs. 24-hour urine samples, 0.71), has a similar association with blood pressure as that for sodium-intake estimates derived from 24-hour urine samples, and has been suggested by the World Health Organization (WHO) as a method of estimating sodium intake in populations. Furthermore, an increased risk of cardiovascular disease or death with low sodium intake is not confined to studies that use spot or fasting urinary estimates; studies that have used a variety of methods to measure sodium intake (including dietary assessments and 24-hour urine samples) in diverse populations have reached the same conclusion.[2] We recognize that the assessment of sodium intake in an individual person requires multiple 24-hour measurements, but that is not required to assess the association between categories of sodium intake and cardiovascular disease in populations.

Second, in their application of the Hill criteria, it is surprising that in a discussion of the plausibility criterion the authors would not include the fact that sodium is an essential mineral. J-shaped associations are expected and common for physiologic factors, such as other minerals (e.g., calcium), vitamins (e.g., vitamin D), and hormones (e.g., thyroxine). Sodium is essential to life and is required for cellular action potentials and volume balance. Although excess sodium intake is a risk factor for hypertension, low sodium intake activates the renin–angiotensin– aldosterone system and is a modifiable risk factor for orthostatic hypotension. We think that the suggestion that sodium intake can be very low and yet be entirely safe contradicts our understanding of the role of sodium in human physiology.

Third, the case presented by Cogswell et al. is predicated primarily on disproving the harm, rather than on establishing a cardiovascular benefit, of low sodium intake. It is not known whether low sodium intake results in a lower risk of cardiovascular disease than moderate sodium intake does, but there is generally consistent epidemiologic evidence that high sodium intake (>5 g per day) is associated with a higher risk of cardiovascular disease.[2] None of the studies referenced by Cogswell et al. show that the risk of cardiovascular disease with low sodium intake (<2.3g per day) is significantly lower than the risk with moderate intake in general populations. They highlight two cohort studies to support their case,[3,4] but neither study showed that the risk of cardiovascular disease with an intake of less than 2.3 g per day was significantly lower than the risk with moderate intake ranges. One study describes a J-shaped association between sodium intake and the risk of myocardial infarction, with a nadir of risk of all cardiovascular disease events at a moderate sodium intake of 2.9 to 3.6 g per day,[4] findings that are entirely consistent with a J-shaped association shown in other prospective cohort studies. The interpretation by Cogswell et al. of a 2014 Cochrane review[5] contradicts the conclusions reached by the authors, with the latter reporting insufficient power to confirm clinically important effects. The authors reference an Institute of Medicine (IOM) review[6] as supporting a low sodium intake (<2.3g per day), but it did not. The IOM committee found that the evidence from studies of direct health outcomes was insufficient and inconsistent for the conclusion that the lowering of sodium intake to below 2.3 g per day either increases or decreases the risk of cardiovascular disease, and it recommended better-quality cohort studies and randomized trials.

Proof of the benefit and safety of low sodium intake on health outcomes is needed before extreme reduction in sodium intake can be recommended for entire populations. Until large, randomized trials clearly show a lower risk of cardiovascular disease with low sodium intake, there is no solid basis for recommending low sodium intake for the prevention of cardiovascular disease. Current public health efforts should focus on reducing sodium intake in populations with high sodium intake (>5 g per day) rather than on diverting resources in the pursuit of an unproven strategy of low sodium intake for the entire population.