Viewpoint

Unnecessary Controversy Regarding Dietary Sodium: A Lot About a Little

Norman R. C. Campbell, MD, Francesco P. Cappuccio, FRCP, FFPH, FAHA, and Sheldon W. Tobe, MD, FRCPC, FACP, FASH

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Recently, the Journal of the American Medical Association (JAMA) published an article by Stolarz-Skrzypek and colleagues. The cohort study examined 24-hour urinary sodium excretion in relationship to blood pressure, hypertension, and fatal and nonfatal outcomes and concluded low-sodium diets increased cardiovascular disease and should not be recommended on a population basis. The investigative team was strong and indicated a lack of financial conflicts of interest. So what does this mean? In this article we review briefly the evidence supporting the link between higher dietary sodium intake and cardiovascular disease and the smaller body of work purporting that this association is not true, present an abbreviated critique of the study by Stolarz-Skrzypek et al., and add this study to a growing meta-analysis demonstrating that higher sodium intake is associated with worse cardiovascular outcomes.

Reviews of the extensive body of evidence on health risks of high dietary sodium by respected and independent scientific and public health bodies indicate dietary sodium consumed at current levels represents one of the major risks to wellness in the world. In fact, reducing dietary sodium has been estimated to save very substantive health costs in developed countries9,10 and to be even more cost effective than interventions to reduce smoking in middle- to low-income countries.11 The research base for the impact of high sodium intake spans animal and clinical studies and is summarized briefly in Table 1.3-8

In contrast, a few cohort and epidemiologic studies do not confirm the association between high salt intake and hypertension or vascular disease.2,4,7 The fact that a few studies do not support the health risks of high sodium intake is not surprising given the limitations of epidemiologic study designs and in particular the difficulty epidemiologic studies have in addressing confounding factors. A study from Cohen et al.12 designed similarly to the cohort study by Stolarz-Skrzypek et al., suggested that high-sodium diets were associated with less vascular disease. Inspection of Cohen’s results showed those who eat less sodium tended to be less educated, less active, black, and of smaller body size, all factors also associated with low income and associated with poor outcomes. Even with the design limitation, when the cohort studies that assessed the relationship between dietary sodium and cardiovascular outcomes were put in a landmark meta-analysis by Strazzullo et al., increased dietary sodium was associated with increased vascular disease.

The new study by Stolarz-Skrzypek et al.1 has the limitations of the cohort study by Cohen et al.12 While the 24-hour collection of urine is the standard for assessing a population’s sodium intake, if it is not collected correctly, there is a risk of misclassification. Further, a single 24-hour urine collection is not as accurate as multiple-day collection in classifying an individual’s usual sodium intake as there is day-to-day variation in sodium intake.13 Of concern, data from Stolarz-Skrzypek et al.’s electronic Table 1 shows that the lowest-sodium group tended to have higher cardiovascular risk factors, including the lowest educational attainment, higher baseline systolic blood pressure, age, and total cholesterol. Further, the study population was young and hence had a low cardiovascular disease event rate, and the statistical power to detect a dietary sodium effect is low. Sodium intake covaries with caloric intake, and the physically active would tend to be in the high-sodium-intake group, as they have been in other studies.12 The lower-sodium-excretion group (across genders and population samples) also had lower urinary creatinine, potassium, and urinary volumes not explained by differences in body mass, suggesting concurrent illness or an undercollection of the 24-hour urine, rather than consumption of less sodium. The finding in the lower-sodium-excretion group of urinary sodium values of 50 mmol/d (1150 mg/d), levels not found in typical Western populations, is also suggestive of this.14,16 Low adherence even to placebo is strongly associated with poor outcomes.17,18 A usual finding for lower sodium intakes is higher potassium intake, since the main mechanism for lower dietary sodium intake is the consumption of unprocessed foods, which are higher, not lower, in potassium.19 Thus the “inverse” association between
Table 1. Summary of types of studies detailing the effects of sodium on blood pressure, and major outcomes

<table>
<thead>
<tr>
<th>Type of study</th>
<th>Details</th>
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<tbody>
<tr>
<td>Animal studies</td>
<td>Demonstrate high sodium intake increases blood pressure and causes adverse vascular outcomes both directly and through increased blood pressure</td>
</tr>
<tr>
<td>Migration studies</td>
<td>Show increases in blood pressure in people who migrate from areas of low to high sodium intake</td>
</tr>
<tr>
<td>Genetic diseases</td>
<td>Indicate that defects in the ability to retain sodium cause low blood pressure and that defects lead to sodium retention cause hypertension</td>
</tr>
<tr>
<td>Cohort studies</td>
<td>Associate high dietary sodium to vascular disease (meta-analysis)</td>
</tr>
<tr>
<td>Randomized controlled trials</td>
<td>Demonstrate low sodium intake decreases blood pressure in newborn babies, children, and adults and reduces hypertension in adults</td>
</tr>
<tr>
<td>Limited controlled intervention studies</td>
<td>Show reductions in vascular disease with reduced sodium intake</td>
</tr>
<tr>
<td>National intervention studies</td>
<td>Demonstrate that dietary sodium can be reduced and is associated with decreased blood pressure and vascular disease</td>
</tr>
<tr>
<td>Arrays of studies on surrogate markers of outcomes</td>
<td>Show that lower sodium intake is associated with: (1) a reduction in intermediate cardiovascular end points such as left ventricular mass and endothelial dysfunction and (2) a reduction in bone mineral density loss, kidney stone disease, and predisposition to gastric cancer</td>
</tr>
</tbody>
</table>

Table 2. Pooled relative risk estimates of incident strokes and total cardiovascular events associated with 2000-mg difference in sodium intake

<table>
<thead>
<tr>
<th>Participants</th>
<th>Events</th>
<th>Pooled relative risk (95% confidence interval)</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incident strokes (15 trials)</td>
<td>157,963</td>
<td>5,379</td>
<td>Random-effects model: 1.20 (1.03-1.41)</td>
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<td></td>
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<td></td>
<td>Fixed-effects model: 1.14 (1.05-1.24)</td>
</tr>
<tr>
<td>Incident total cardiovascular events (13 trials*)</td>
<td>105,677</td>
<td>5,128</td>
<td>Random-effects model: 1.14 (0.99-1.33)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fixed-effects model: 1.08 (1.02-1.15)</td>
</tr>
</tbody>
</table>

* Without outlier, based on sensitivity analysis. See Strazzullo et al.2
intake should be reduced to prevent cardiovascular disease, the health priority is and should remain how to reduce population sodium intake to save lives.

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References