motivated salt appetite found in rodents do actually apply in humans because, as Hendriksen et al (1) report, sodium intake of humans is very high. However, humans do not appear to increase drinking in response to the increased osmolality that should follow an increase in sodium intake. This is a confounding problem because blood volume can be restored easily by drinking sufficient quantities of water.

Furthermore, until blood volume is restored, the regulatory hormones mentioned above will continue to be released. Interestingly, the overwhelming majority of medications used to combat cardiovascular disease are blockers of the renin-angiotensin system (3). More recently, antagonists of the aldosterone receptor have shown their efficacy when given in association with the other medications (9). The presence of increased plasma concentrations of angiotensin and aldosterone would suggest that humans are chronically dehydrated, or hypohydrated, and yet they continue to consume large quantities of sodium as if they were trying to repair the volume loss. Why humans do not drink despite the combined physiologic signals of increased osmolality and plasma concentrations of angiotensin could be considered the central problem of cardiovascular disease as well as of increased sodium intake.

Perhaps the more appropriate recommendations would be to increase fluid intake, mainly water, with an industry-associated modest reduction in the sodium content of processed foods. The principal objective would be to restore blood volume and decrease concentrations of the regulatory hormones, especially those associated with motivated salt appetite and cardiovascular disease. Furthermore, early rodent work suggests that increased drinking is associated with increased effectiveness of the principal cardiovascular treatment (10). Once concentrations of the hormones are decreased, excretion of sodium in the urine would regulate physiologically most of the dietary intake in excess of need.

Neither of the authors declared a conflict of interest.

Simon N Thornton
Patrick Lacolley

REFERENCES


Reply to SN Thornton and P Lacolley

Dear Sir:

We appreciate the interest of Thornton and Lacolley in our study on the potential health impact of salt reduction in processed foods. They state that an increase in fluid intake is an important measure to deal with high salt intakes. We agree that fluid intake is essential for many physiologic processes in the human body (1) and that consumers should drink enough fluid, preferably water. This view is also expressed in the Dutch dietary guidelines for healthy nutrition, which indicate that fluid consumption should be ~1500–2000 mL/d (2). However, in our opinion, the specific theory that an increase in fluid consumption is crucial for reducing the health burden related to an excessive salt intake has not been proven. This hypothesis must first be tested in long-term randomized controlled trials in humans, and the potential underlying mechanisms need to be supported by physiologic studies. For the moment, a population-based approach to reduce sodium amounts in processed foods can be considered an effective intervention to lower blood pressure and to decrease the burden of cardiovascular disease in the general population (3).

None of the authors declared a conflict of interest.

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Erratum


An author’s name was incorrectly included in the list of authors for this article. Andrea Neiman was not a coauthor, and her name should not have appeared in the list of authors.


Erratum


The conflict of interest statement is incorrect. It should read as follows: “IAM consults for Mars and Coca-Cola and sits on the UK government’s Scientific Advisory Committee on Nutrition. STF, PAG, CAH, and JCGH declared no potential conflicts of interest in relation to the contents of this editorial.”


Erratum


In Table 1, the values for calcium absorption and the associated P value are incorrect. In addition, the P value (0.6) for BMI appears incorrectly in the row for “Age.” A corrected version of Table 1 appears below.

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Control (n = 19)</th>
<th>800 IU (n = 19)</th>
<th>2000 IU (n = 20)</th>
<th>4000 IU (n = 18)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>60 ± 4.5</td>
<td>57 ± 4.5</td>
<td>59 ± 5.8</td>
<td>60 ± 4.7</td>
<td>0.4</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.2 ± 3.8</td>
<td>26.4 ± 3.6</td>
<td>27.6 ± 4.9</td>
<td>26 ± 4</td>
<td>0.6</td>
</tr>
<tr>
<td>Calcium intake (FFQ – dietary + supplement)</td>
<td>1156 ± 580</td>
<td>1027 ± 469</td>
<td>1160 ± 499</td>
<td>1196 ± 518</td>
<td>0.8</td>
</tr>
<tr>
<td>Calcium absorption</td>
<td>33.6 ± 15.2</td>
<td>31.1 ± 16.9</td>
<td>32.1 ± 14.8</td>
<td>31.1 ± 10.5</td>
<td>0.95</td>
</tr>
<tr>
<td>Serum calcium (mg/dL)</td>
<td>9.6 ± 0.3</td>
<td>9.6 ± 0.3</td>
<td>9.5 ± 0.4</td>
<td>9.5 ± 0.4</td>
<td>0.95</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.89</td>
</tr>
<tr>
<td>25(OH)D (nmol/L)</td>
<td>61.7 ± 15.3</td>
<td>64 ± 13.8</td>
<td>64.8 ± 15.1</td>
<td>62.1 ± 14.2</td>
<td>0.90</td>
</tr>
<tr>
<td>1,25(OH)D (pmol/L)</td>
<td>97.9 ± 27.7</td>
<td>104.6 ± 26.3</td>
<td>111.7 ± 38.1</td>
<td>117.3 ± 64.9</td>
<td>0.52</td>
</tr>
</tbody>
</table>

1 All values are means ± SDs. FFQ, food-frequency questionnaire; 1,25(OH)D, 1,25 dihydroxyvitamin D; 25(OH)D, 25-hydroxyvitamin D.
2 P values are based on an overall F test from an ANOVA comparing differences between dose groups.