Is Dietary Sodium Policy Actually Based on Science?¹

Dear Editor,

I was very interested to read 3 of the recent reviews (1–3) from an ASN Scientific Sessions at Experimental Biology 2014 symposium designed to “review a growing body of evidence examining sodium intake and health outcomes that are highly relevant to sodium DRIs, the DGAs (Dietary Guidelines for Americans), and numerous government- and nongovernmental organization–driven efforts to significantly reduce sodium in the entire population” (1). These are comprehensive reports but they appear to have overlooked water intake in relation to sodium intake, and the physiologic regulation of both with respect to cardiovascular disease and overall health (4). Sodium is one aspect of this regulation that along with water intake can be altered through dietary recommendations. Current emphasis on reducing sodium intake not only does not address the core problem, it makes the solution more difficult. There are a host of practical and pragmatic reasons why consumers, and manufacturers in particular, add sodium to foods. These must be countered or suppressed for such a dietary policy to be effective. There is a far simpler path. Rather than seeking to reduce sodium intake, policy could focus on simply reducing it as a concern.

The issue surrounding dietary sodium arises through a human anomaly. In experiments with animals, increased dietary sodium intake increases plasma sodium, thus increasing plasma osmolality. The appropriate physiologic response is stimulation of the osmoreceptors in the hypothalamus, which increases the release of vasopressin and stimulates thirst (leading to the ingestion of fluids). Increased fluid intake initially leads to increased blood volume, and perhaps increased blood pressure. This can create a pressure diuresis that increases kidney water and sodium excretion. Such a pressure diuresis would be temporary, existing until plasma volume and osmolality return to within the normal range, which would reduce activation of the osmoreceptors, lowering concentrations of vasopressin and switching off thirst (4). However, this does not appear to happen in humans under normal conditions; otherwise we would not be discussing dietary sodium policy. Why this does not happen is a separate question. The fact remains that in most people increased sodium intake is not followed by stimulation of an appropriate fluid intake. The question under discussion is the policy direction that is appropriate in dealing with this anomaly. Water drinking is the easiest part of the regulation of body fluids, and hence of cardiovascular regulation, that could be controlled, and seems in fact likely to be ultimately more effective than measures to reduce sodium intake.

The physiologic response to the osmotic stimulus of current sodium intake amounts is increased drinking. The absence of increased fluid intake, or only low amounts of fluid intake, leads to hypohydration and continual, stimulated release of vasopressin. In the long term this leads to a reduction in kidney function, which could be a component of chronic kidney disease. This chronic, mild hypohydration affects both the intracellular and the extracellular fluid compartments, the latter being associated with hypovolemia. In order to recover blood volume loss, both water and sodium are needed, and the physiologic mechanisms regulating this are thirst and salt appetite. Furthermore, hypovolemia stimulates the release of vasopressin, angiotensin II, and aldosterone, and unless blood volume is restored, even just temporarily, these hormones will continue to be released. Chronic, increased blood concentrations of these hormones are associated with cardiovascular disease (1–5) and possibly kidney disease.

Medications that antagonize the renin-angiotensin (and aldosterone) system make up the overwhelming majority of treatments used to combat cardiovascular disease (5, 6). Yet, as shown by McCarron (2), a high concentration of sodium in the urine is normally associated with low plasma concentrations of angiotensin and aldosterone. The fact that, in humans, high intakes of sodium are associated with high blood concentrations of angiotensin, aldosterone, and vasopressin would indicate that humans are chronically dehydrated and/or hypovolemic. Physiologic regulation indicates further that no matter how much reduction of sodium in the diet takes place, unless blood volume is restored, these hormones will continue to be released, further promoting cardiovascular disease, as well as other health complications (4). Blood volume can be restored relatively easily by drinking sufficient quantities of water.

Animal research has shown also that angiotensin and aldosterone are associated with a centrally stimulated appetite for sodium (2, 7). This has not been demonstrated in humans but the fact that they consume large amounts of dietary sodium could indicate that the hormone-induced appetite may be operating. Once again, no matter what the policies are to reduce sodium intake (3), the physiology of hypovolemia dictates that we will continue to consume sodium in an attempt to restore blood volume. McCarron (2) points out that measures of kidney sodium excretion in humans are similar across ages, sexes, and cultures, and he thus suggests that intakes are also similar. However, work by Maril et al. (8) and Titze (9) would indicate that intake can be greater than the amount excreted and that this excess sodium is taken up and stored in kidneys, skin, and muscle without associated water. This would imply that there is a maximum kidney sodium excretion rate in humans and that to prevent a long-term increase in plasma osmolality (also detrimental for overall health), sodium must be removed from the circulation. This stored sodium is mobile and can be eliminated when appropriate hydration conditions are provided (9, 10).

In conclusion, humans are perfectly well equipped to deal with excess sodium as long as there is an appropriate (increased) fluid intake. Dietary policies aimed at dealing with cardiovascular disease should involve principally recommendations to increase water intake. Once blood volume is returned to normal, the “appetite” for sodium should be reduced.

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Simon N Thornton

*From the University of Lorraine, Nancy, France
(E-mail: simon.thornton@inserm.fr).*

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Reply to SN Thornton¹

Dear Editor:

The comments and concerns expressed by Thornton (1) relate to our recent reviews from the ASN Scientific Sessions at Experimental Biology 2014 symposium regarding sodium intake and health outcomes. His comments appropriately expand the discussion of whether to reduce sodium intakes to low amounts. Our intent with the cited papers was never to exclude critical cofactors such as water. The neural regulation of water and sodium are physiologically interrelated (2), because optimizing both volume and osmolality are essential elements of cardiovascular health. As noted by

Thornton, the hormonal response to decreased perfusion invokes angiotensin II, aldosterone, and vasopressin release, all of which have potent vascular actions that potentially increase cardiovascular disease (CVD) risk.

Thornton's argument that overlooking the role of water intake in understanding the association of sodium with health outcomes raises another question as to the safety of a health policy directed at lowering sodium intake across the entire population. Hyponatremia at hospital admission and in free-living populations has been associated with both increased morbidity and mortality (3–6). Those findings are consistent with the proposition that sodium and water intake must be considered in tandem when assessing health outcomes. As opposed to previous hypotheses that comorbidities accounted for an increased risk of death, recent data indicate that the greatest risk appears in those with few comorbid conditions (5, 6).

Hyponatremia is the most common electrolyte disturbance in the general population (6). Reduced sodium intake in and of itself can impair the healthy kidney's ability to excrete excess free water (7). Therefore, a lower sodium diet would promote the development of a reduced serum sodium concentration, or hyponatremia. Although it is typically assumed that with normal kidney function there is a large excess renal capacity to excrete water, what is not generally appreciated is that reduced solute intake impairs the healthy kidney's capacity to excrete excess water (7). Sodium intake is one of the principle contributors to the renal solute load. Thus, lower sodium intake potentially sets in motion a normal physiologic response that would contribute to the development of hyponatremia in a segment of the general population. If healthy persons lower their sodium intake as recommended by current guidelines, but maintain water consumption that exceeds requirements (as is widely promoted to the general population, especially physically active people), those guidelines may well set the stage for the emergence of hyponatremia even in otherwise healthy people. The hyponatremia-related mortality and morbidity data published to date and the physiology of sodium and water handling by the kidney provide a plausible explanation to account for the increased risk of death from lower sodium intake in healthy, low-risk individuals (8). Furthermore, such a risk would not solely rest on activation of the renin-angiotensin system and aldosterone invoked for those already at risk of CVD.

Perhaps the simplest and most important aspect of the issue raised by Thornton is his reminder that water metabolism is linked to sodium. That obvious and accepted physiologic link should serve as a note of caution that there are many “moving parts” associated with reducing sodium intake in the general population. The longstanding assumption that sodium restriction to amounts below that normally consumed in modern societies is safe and effective fails to consider that complex physiology. The old adage that “it is too good to be true” may well apply to the simplistic policy view that sodium restriction lowers blood pressure and therefore reduces CVD risk.