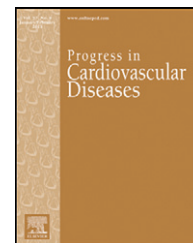


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## Hypertension Due to Toxic White Crystals in the Diet: Should We Blame Salt or Sugar?

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### ABSTRACT

The "Salt Hypothesis" is the notion that an increase in salt intake will increase blood pressure and thus increase the risk of cardiovascular disease (CVD), which has been a point of contention for decades. Despite this, numerous health organizations, dietary guidelines, and government policies advocate population-wide salt restriction. However, there is no conclusive proof that restricting salt intake reduces the risk of hypertension (HTN) and/or CVD events; sodium restriction in fact may paradoxically lead to adverse health outcomes. Importantly, another white crystal, sucrose (or table sugar) but also high-fructose corn syrup are much more detrimental food additives. Indeed, added sugars have the ability to induce hypertension via the promotion of inflammation, oxidative stress, insulin resistance, and obesity. Considering that there is no physiologic requirement for dietary carbohydrate, there is little reason to suspect adverse health consequences from cutting back on sugar. This paper reviews the evidence relating to salt and sugar on HTN and CVD. Based on our review of the scientific literature, guidelines should focus more on reducing sugar rather than salt for the prevention and treatment of HTN and its consequences.

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The authorities pushing the 'eat-less-salt' message had "made a commitment to salt education that goes way beyond the scientific facts."<sup>1</sup>

[Drummond Rennie (JAMA editor)]

Numerous government bodies and health institutions recommend population-wide sodium restriction to reduce cardiovascular disease (CVD).<sup>2-4</sup> Starting with the least restrictive, Canada's 2010 Sodium Working Group recommends reducing the average population sodium intake to 2300 mg/day mainly by advocating a reduction in the sodium content of processed/packaged foods.<sup>2</sup> The World Health Organization (WHO) recommends that everyone around the globe consume less than 2000 mg of sodium per day.<sup>3</sup> And the American Heart Association (AHA) goes even further by recommending that all Americans reduce

their sodium intake to <1500 mg/day.<sup>5</sup> Although this level of sodium restriction is advocated by many scientists, it is based largely on expert opinion that this level of salt restriction will lead to further reductions in blood pressure (BP).<sup>6</sup>

The previous 2010 Dietary Guidelines for Americans recommended a sodium intake of <2300 mg per day for the general population, and <1500 mg/day among certain "higher risk" populations, such as African Americans, people 51 years of age and older and people who have hypertension (HTN), diabetes mellitus (DM), or chronic kidney disease (CKD).<sup>4</sup> However, the Institute of Medicine (IOM) recently released an evidence-based report in May 2013 assessing the 2010 DGAC recommendations and concluded that there is insufficient evidence to recommend reducing the consumption of sodium to below 2300 mg/day, and even acknowledged that salt

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## Abbreviations and Acronyms

AHA = American Heart Association

BP = blood pressure

CKD = chronic kidney disease

CVD = cardiovascular disease

DM = diabetes mellitus

HTN = hypertension or hypertensive

IOM = Institute of Medicine

restriction may lead to adverse health risks.<sup>7</sup> A few salient points have been summarized from the IOM report below

- “Studies on health outcomes are inconsistent in quality and insufficient in quantity to determine that sodium intakes below 2300 mg/day either increase or decrease the risk

of heart disease, stroke, or all-cause mortality in the general U.S. population.

- “The committee found some evidence suggesting risk of adverse health outcomes associated with sodium intake levels in ranges approximately 1,500 to 2,300 mg/day among those with diabetes, kidney disease, or CVD.”
- “There is no evidence on health outcomes to support treating population subgroups differently from the general U.S. population”
- “The evidence on health outcomes is not consistent with efforts that encourage lowering of dietary sodium in the general population to 1,500 mg/day.”

The above IOM summary is likely why the 2015 Dietary Guidelines for Americans eliminated the <1500 mg/day sodium recommendation in those at-risk subgroups. The 2015 Dietary Guidelines for Americans however still recommend a sodium restriction of <2300 mg/day.<sup>8</sup>

## Salt intake

“There is, after all, abundant evidence that the intrinsic salt appetite in mammals—and hence in *Homo sapiens* as well—is firmly set so as to best handle risks at both ends of the spectrum.”<sup>9</sup>

[Bjorn Folkow]

The brain’s ability to detect and react to sodium depletion, to seek it out (sodium appetite), to detect sodium in the environment (taste receptors) and to consume a sufficient amount of sodium are key evolutionary adaptations that has allowed many animals to survive across various geographic environments.<sup>10</sup> While most health organizations and government programs recommend population-wide sodium restriction this is likely a futile effort, potentially wasting money and resources.<sup>2</sup> This is because sodium intake is regulated by the hypothalamus<sup>10</sup> and trying to force a population to reduce their sodium intake to <2300 mg/day may be practically impossible. In fact, the intake of sodium across numerous countries over the last 50 years has remained in a very tight range (approximately 3.5–4 g of sodium per day) despite population-wide policies recommending sodium

restriction.<sup>10,11</sup> This suggests that the intake of sodium is indeed driven by physiology not by behavior or a hedonic drive.

The approximate average intake of sodium hovers around 3400 mg/day (or 8.5 g of salt) in Canada, the United States, and the United Kingdom.<sup>2,12</sup> This amount is likely the ideal level for optimal health, sitting at the center of our homeostatic salt set-point.<sup>13</sup> In fact, when adjusted for size and metabolic rate, animals such as sheep and rats consume very close to the average amount of salt humans do, suggesting a similar salt-set point in animals.<sup>13</sup> Sodium is the predominant ion in the extracellular fluid, and is essential for maintaining normal osmotic pressure, plasma volume (and hence tissue perfusion), and nerve impulses, and is also critical for optimal acid–base balance, ionic gradients and trans-membranous potential. Accordingly, it makes sense that the body would have a system (the hypothalamus and the renin–angiotensin aldosterone system for example) in place to ensure optimal dietary intake and internal regulation of sodium.

In summary, sodium intake seems to be physiologically driven in both humans and animals. The average intake across mammalian species is similar when given free access to salt; suggesting an ideal sodium set-point that is controlled by physiology. The human body is able to: a) identify sodium depletion; b) drive increased sodium intake and c) maintain normal sodium homeostasis. This tight physiological regulation of salt intake/homeostasis has allowed species to survive across diverse habitats and environmental stressors (heat, excessive loss of fluid, stress).<sup>10</sup> Other types of dietary interventions, such as focusing on improving the overall diet (e.g., with the Mediterranean or DASH diet) are more likely to bestow greater health benefits without the need to restrict salt intake.<sup>14,15</sup>

## Salt and BP

In the early 1900s, Allen and Sherrill found prolonged BP reduction in only 60% of 180 patients with hypertension when salt intake was severely restricted to less than 2 gram per day (around 800 mg of sodium per day or less).<sup>16</sup> Surprisingly, Pines and Perera<sup>16</sup> did not find significant increases in BP with the administration of 10 to 20 g of salt for two weeks in 3 normotensive patients. Grant and Reischman<sup>17</sup> gave normal males 20 to 30 g of salt per day and found similar effects as Pines and Perera. Berger and Feinberg<sup>18</sup> provided as much as 25 g of salt per day to HTN patients without a BP effect. And Kawasaki et al.<sup>19</sup> stated that “salt loading alone or with injection of desoxycorticosterone acetate (DOCA) virtually never increases BP significantly in normal subjects.” Hence, even the combination of corticosteroids and a high-salt intake in normal patients does not seem to be enough to induce HTN.

Paradoxically, other studies showed that salt loading actual can reduce BP in salt-resistant patients.<sup>20</sup> Luft and colleagues<sup>21</sup> concluded in one of their studies that, “sympathetic nervous system activity appears to decrease with sodium loading in normal subjects. These responses may have facilitated the excretion of massive salt loads in normal subjects and may have modulated the increases in BP.” Low-salt diets can increase vasoconstriction<sup>22</sup> and giving salt can lead to vasorelaxation, particularly in those who are “salt-resistant” (i.e., those who do

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