Abstract

The German Society for Nutrition (DGE) has updated their recommendations for salt intake in January 2017 [1]. According to this press release they consider 1.5g of Sodium (equivalent to 3.5g of salt) as adequate. This is in line with recommendations from other world-wide respected Organizations like the American Heart Association, the Institute of Medicine or the World Health Organization [2–4]. Since only 0.3% of the global population consume such low amounts of salt per day, these guidelines should promote dramatic changes of the world-wide consumer's behavior [5].

Nevertheless these Organizations consider a reduction in sodium intake as an effective health action to reduce blood pressure and improve cardiovascular (CV) health [2–4]. Since higher sodium intake is strongly associated with higher blood pressure, which itself is a leading risk factor for heart disease and stroke, it is believed to be somehow causal for the leading causes of death.

Although clinical trials have undermined the association of reduced blood pressure with reduced sodium intake, the latest Cochrane review, including a total of 185 randomized controlled trials, estimates the impact of salt restriction on blood pressure with less then 1 % as relatively small (the reduction in SBP/DBP in people with normotension was about 1/0 mmHg, and in people with hypertension about 5.5/2.9 mmHg) [6]. Furthermore, to my knowledge, no large randomized trial was intended to investigate favorable effects on CV risk due to salt restriction. Data of epidemiological studies are inconsistent with many showing an increased risk among those consuming less than 3g of sodium per day [7–9]. According to O'Donnell et al., published in 2011, there exists a J-shaped association between estimated sodium excretion of 28,880 patients and CV-events with an increased risk of all CV-events associated with a sodium excretion of greater than 7g (17,5g Salt) per day and less than 3g (7,5g Salt) per day [9].

Since Evolution brought us from a salty marine environment in which sodium chloride is by far the dominant mineral (90%) to where we live now, sodium is an essential nutrient and has some vital functions in every organism [10]. According to any medical textbook, we find chronic hyponatremia to be associated with symptoms like headaches, nausea, poor balance, confusion, seizures, and finally coma [11]. In 1986 it was shown, that pregnant women change their sense of taste in favor of more salty solutions [12]. So it is not surprising that studies found salt restriction in animals and humans to be causal for retardation of fetal growth, leading to low birth weight and the underdevelopment of cardiovascular organs [13]. Hyunwoo et al. examined the relation between salt restriction and insulin resistance, and found a positive association in at least 9 out of 25 studies, with very heterogeneous designs, indicating potential effects of low sodium diets on insulin resistance [14]. Published 1993, Overlack et al. could show an alarming heart rate rising effect of salt restriction [15].

On the other hand it was shown that a sodium loading approach, with 2g of salt, four times a day in a group of 21 patients with primary hypertension, leads to a 8% lowered glycemic response in the oral glucose tolerance test [16]. Furthermore, a second analysis of existing data of two randomized controlled salt trials reveals a significant fall of serum uric acid in the moderate and high intervention group in hypertensive and non-hypertensive subjects [17].

So what are the proposed mechanisms to explain those findings? Since Brunner et al. had published the strong association between sodium excretion and plasma renin and aldosterone excretion in 1972, these findings were replicated over and over again and summarized in the 2017 Cochrane Review on effects of low sodium diets versus high sodium diets [6,18]. The paper showed a significant increase in plasma renin, aldosterone, adrenaline and

noradrenaline which are the main hemodynamic, hormonal responses to sodium shortage. These salt-retaining systems maintain the fragile equilibrium of electrolytes in a salt depleted environment – but at what cost? Chronic elevated aldosterone is well known for its association with numerous symptoms like hypokalemia, osteoporosis, poorly controlled hypertension, sleep apnea and fibrosis of the heart or kidney that can lead to failure of these organs [19–21]. Moreover, the Cochrane paper [6] listed a 2.5% increase in cholesterol, and a 7% increase in triglycerides, two well known risk factors for any CV event.

On the other hand, favorable effects such as the release of natriuretic peptides, a natural aldosterone inhibitor, could be seen in response to a diet rich in salt [22]. These peptides partly control the immune response in an anti-inflammatory and modulatory manner [23].

To shed more light into the controversial findings weather salt reduction results in an elevated or lowered risk for CV events, O'Donnell et al. conducted an investigation within the PURE study in 2014 [7]. With 101,945 fasting morning urine samples from five continents, it was the largest international study to examine the association between sodium intake and health. The estimated associations are based on more than 3000 outcome events, high rates of follow-up and incorporation of a very well validated sodium estimation approach [24]. Their results confirmed J-shaped association published in 2011 with a lower risk of death and CV events correlated with an estimated sodium intake between 3g per day and 6 g per day (7,5g – 15g of salt!).

Furthermore, in 2016 a pooled analysis of 133,118 individuals (63,559 with hypertension and 69,559 without hypertension), from 49 countries out of four large prospective studies undermined these results and found similar associations of urinary sodium excretion with cardiovascular events [25]. In addition there was no association of high sodium intake with an increased risk of CV events or death in the normotensive population, suggesting that salt restriction recommendations should only be aimed at the population with hypertension.

In my personal opinion and summing this up, it appears that the latest history of nutrition guidelines is repeating itself. Similar to the ban of cholesterol and saturated fats, worldwide salt reducing recommendations are based on preliminary observational data with missing trails testing the hypothesis that reducing sodium intake could lower CV risk. Taking the existing evidence into account it is probably harmful to recommend a sodium reduction below 3g per day.

An evolutionary approach would consider the fact that a “hunger” for salt evolved in animals as well as in humans, as studied in isolated indigenous people, for good reasons, since vertebrates were faced with salt scarcity living outside of the oceans. A guideline that turns on our ancient emergency hormonal responses seems to be counterproductive. What’s more important is to reduce the excessive intake of processed foods and emphasize the importance of real food with a favorable sodium to potassium ratio. A liberal salted meal of vegetables and meat must be labeled as nothing but healthy!

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