



Contents lists available at ScienceDirect

# Nutrition

journal homepage: [www.nutritionjrn.com](http://www.nutritionjrn.com)

## Editorial

### The salt debate—More salacious than salubrious

Over the past two decades, the understanding of decreasing salt as a means to reduce blood pressure has evolved from a recommendation for voluntary dietary restriction for individuals at risk to a point where the entire population is in the process of having its food choices limited to products engineered for decreased sodium intake in accordance with the 2010 dietary guidelines.

The *Dietary Guidelines for Americans, 2010* [1], driven by the stepwise approach described in the Institute of Medicine (IOM) study, *Strategies to Reduce Sodium Intake in the United States* [2], declares we should decrease sodium intake to 2300 mg/d (6.0 g of salt) for 50% of the population and further decrease intake to 1500 mg/d (3.9 g of salt) for the remaining 50%, considered to be at higher risk for hypertension. Despite the broad perception that our sodium intakes have increased in recent years, a recent study by Bernstein and Willett [3] indicates that our sodium intake has been stable at approximately 3500 mg/d of sodium (9.1 g of salt) for the past 50 y. This stable figure appears to repudiate any link between salt intakes and the increasing rates of obesity and hypertension.

There is very little documented evidence of salt consumption, but it appears that we may currently be consuming a far lower level than ever before in recent recorded history [4]. Military records spanning the period from the War of 1812 [5,6] to the end of World War II [7,8] indicate that rations for soldiers and prisoners of war varied from 18 to 20 g/d of salt—about double the amount we currently consume in the USA.

The large decrease in salt consumption occurred immediately after World War II, when refrigeration replaced salt as the primary means of food preservation. Before that time, most traditional foods such as cheeses, beef, pork, fish, and vegetables were preserved in salt or with salt brine. After the large decrease in salt consumption that took place in the 1950s, our salt consumption has remained stable.

Decreasing dietary salt as the primary lifestyle strategy to reduce blood pressure has been aggressively promoted by activist groups such as the Center for Science in the Public Interest and World of Action on Salt and Health and by the Centers for Disease Control and Prevention, the World Health Organization, the IOM, and more recently the Food and Drug Administration. Their success in this effort has influenced many food processors, eager to take advantage of perceived public sentiment, to decrease the salt content of their food products voluntarily.

Even the White House has weighed in by placing pressure on many influential food industry stakeholders to decrease the salt content of their foods. Wal-Mart, one of the country's largest food retailers, has publicly indicated that it will require their suppliers to dramatically decrease the salt content of their food products [9]. This type of influence effectively sidesteps the normal regulatory process and the need to provide the scientific justification for such regulations.

With these initiatives actively underway, we can expect the population-wide consumption of salt to start approaching the 2010 dietary guidelines goal of 2300 mg/d of sodium or less. When this happens, Americans will be consuming considerably less salt than they ever have in recorded history and less than in any other country in the world [10,11].

Because this sodium-decrease strategy will be charting new territory, with the goal of achieving highly significant public health benefits, the entire population will, in effect, be placed in a massive dietary intervention trial to demonstrate the impact of low sodium intakes. Indeed, this may have been the understanding of the committee that drafted the IOM's *Strategies to Reduce Sodium Intake in the United States*, because it cautioned that at all stages of its stepwise sodium decrease strategy, efforts to analyze any unintended consequences should be carried out [12].

As the primary lifestyle strategy to reduce blood pressure, decreasing dietary salt differs significantly from other interventions, such as adopting a Dietary Approaches to Stop Hypertension (DASH)/Mediterranean diet or increasing physical activity or decreasing workplace stress, because the potential negative consequences of these latter strategies are known to be very limited. However, as stated by Alderman et al. [13] and repeated in the IOM's dietary recommended intakes [14], renin and consequently aldosterone levels begin to increase significantly when sodium intakes fall below 2800 to 3000 mg/d (~7–8 g/d of salt). It is now generally recognized that increased renin/aldosterone levels are a significant risk factor for cardiovascular disease, just as blood pressure is.

Although decreasing salt intakes may reduce blood pressure for some individuals, it will likely increase renin/aldosterone levels for everyone. As a strategy to decrease blood pressure, unlike the DASH/Mediterranean diet or decreased stress or increased physical activity, decreasing salt does have significant potential for negative consequences because it trades one cardiovascular risk factor (blood pressure) for another (renin/aldosterone). This

tradeoff appears to account for the many studies that demonstrate decreasing salt lowers blood pressure for some and markedly increases morbidity and mortality for many others. This tradeoff in risk factors has never been accounted for in all the models projecting the benefits of decreasing salt and has seldom been mentioned in the arguments for a population-wide salt decrease.

Some recent publications have verified the negative consequences of decreasing salt in the diet [15–21]; therefore, the unintended consequences of decreasing salt cannot be lightly brushed aside as being inconsequential or non-existent.

Considering the potential impact of this significant dietary adjustment, normal prudence should have dictated a large randomized controlled clinical trial before any population-wide intervention. That has not been the case and the current compulsion to decrease salt intake, actively supported by public health authorities, the administration, advocacy groups, and the food industry, must be coupled to a phase IV-type analysis to track the relation of salt intakes (using 24-h urinary sodium measurements) with specific health outcomes and risk markers to ascertain the actual impact of decreasing salt. At a very minimum, subjecting an entire population to an untested dietary regime with known risks requires this analysis be instituted.

This will be the only way for us to learn if decreasing population-wide salt intakes is a benefit or a liability to public health. The public deserves no less.

## References

- [1] US Department of Agriculture and US Department of Health and Human Services. Dietary guidelines for Americans, 2010. 7th ed. Washington, DC: US Government Printing Office; 2010.
- [2] Institute of Medicine. Strategies to reduce sodium intake in the United States. Washington, DC: National Academies Press; 2010.
- [3] Bernstein AM, Willett WC. Trends in 24-h urinary sodium excretion in the United States, 1957–2003: a systematic review. *Am J Clin Nutr* 2011;92: 1172–80.
- [4] Satin M. Mandating regulations in the face of contradictory evidence. *Nutrition* 2011;27:388–9.
- [5] Rations: Conference Notes Prepared by The Quartermaster School For the Quartermaster General, January 1949. *The History of Rations*. US Army Quartermaster Foundation, Fort Lee, Virginia. Available at: [http://www.qmfound.com/history\\_of\\_rations.htm](http://www.qmfound.com/history_of_rations.htm)
- [6] Adams J. Dartmoor prison. A faithful narrative of the massacre of American seamen, to which is added a sketch of the treatment of prisoners during the late war by the British Government. Pittsburgh: S. Engles; 1816.
- [7] Koehler FA. Special rations for the armed forces, 1946–53. QMC historical studies, series II:6: historical branch. Washington, DC: Office of the Quartermaster General; 1958.
- [8] Military Intelligence Service, War Department. American prisoners of war in Germany. Restricted classification removed—STALAG 17B (Air Force non-commissioned officers). 1945.
- [9] Institute of Medicine. Wal-Mart commits to improve the health of its food products. Available at: <http://www.iom.edu/Reports/2010/Strategies-to-Reduce-Sodium-Intake-in-the-United-States/Walmart-Commits-to-Improve-the-Health-of-Its-Food-Products.aspx>. Accessed April 29, 2011.
- [10] Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. *BMJ* 1988;297:319.
- [11] McCarron DA, Stern JS, Graudal N. Public policy and dietary sodium restriction. *JAMA* 2010;303:1916.
- [12] Institute of Medicine. Strategies to reduce sodium intake in the United States. Washington, DC: National Academies Press; 2010. p. 280.
- [13] Alderman MH, Madhavan S, Ooi WL, Cohen H, Sealey JE, Laragh JH. Association of the renin-sodium profile with the risk of myocardial infarction in patients with hypertension. *N Engl J Med* 1991;324:1098–104.
- [14] Institute of Medicine/Food and Nutrition Board. Dietary reference intakes for water, potassium, sodium, chloride, and sulfate. Washington, DC: National Academies Press; 2004. p. 282.
- [15] Garg R, Williams GH, Hurwitz S, Brown NJ, Hopkins PN, Adler GK. Low-salt diet increases insulin resistance in healthy subjects. *Metabolism*; 2010. In press.
- [16] Ekinci EI, Clarke S, Thomas MC, Moran JL, Cheong K, MacLasaac RJ, Jerums G. Dietary salt intake and mortality in patients with type 2 diabetes. *Diabetes Care* 2011;34:703–9.
- [17] Thomas MC, Moran J, Forsblom C, Harjutsalo V, Thorn L, Ahola A, et al. The association between dietary sodium intake, ESRD, and all-cause mortality in patients with type 1 diabetes. *Diabetes Care* 2011;34:861–6.
- [18] Paterna S, Parrinello G, Cannizzaro S, Fasullo S, Torres D, Sarullo FM, Di Pasquale P. Medium term effects of different dosage of diuretic, sodium, and fluid administration on neurohormonal and clinical outcome in patients with recently compensated heart failure. *Am J Cardiol* 2009;103: 93–102.
- [19] Paterna S, Gaspare P, Fasullo S, Sarullo FM, Di Pasquale P. Normal-sodium diet compared with low-sodium diet in compensated congestive heart failure: is sodium an old enemy or a new friend? *Clin Sci (Lond)* 2008; 114:221–30.
- [20] Sowers JR, Whaley-Connell A, Epstein M. Narrative review: the emerging clinical implications of the role of aldosterone in the metabolic syndrome and resistant hypertension. *Ann Intern Med* 2009;150:776–83.
- [21] Stolarz-Skrzypek K, Kuznetsova T, Thijs L, Tikhonoff V, Seidlerová J, Richart T, et al. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. *JAMA* 2011;305:1777–85.

Morton Satin, M.Sc.  
Salt Institute, Alexandria  
Virginia, USA