

More Mixed Messages in Terms of Salt

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In this issue of *CJASN*, McCarron *et al.* (1) again take on the salt and health issue. The group seems to be in the minority this year, because the salt admonishers have been out in force (2–4). The salt party-line opposition has been relatively quiet as of late, although Graudal and Galløe (5) pooh-poohed salt restriction as a component of antihypertensive therapy, stating that, “Population studies have not been able to show an association between salt intake and unfavorable health outcome.” On the contrary, in their review, He and MacGregor (3) showed a relationship between urinary sodium excretion and death from stroke with $r = 0.832$. They then showed a similarly robust relationship between urinary sodium excretion and stomach cancer with $r = 0.702$. Cardiac left ventricular mass takes a hefty beating with $r = 0.61$. A redrawn plot from Intersalt shows an increase in BP with age against salt weighing in at $r = 0.56$, a topic that affects me directly, although my wife thinks it’s the booze. Dr. He, one of the authors, makes his living from epidemiology, so it must all be true. It is surprising that anyone is left alive. Nothing brings on more emotions than this topic!

Why are the loins being girded? I do not know exactly; however, McCarron *et al.* (1) point out that the Institute of Medicine committee has been “at it again.” This most learned pundit body (in the United States, at least) has issued new strategies to reduce salt intake (6). The means or strategies the committee may include but are not limited to are the initiation of, “1 actions by food manufacturers such as new product development and food reformulation, 2 actions at the government level such as special initiatives and regulatory or legislative options, and 3 actions by public health professionals and consumer educators.” So it seems that something is coming down at the national level. He and MacGregor certainly agree with this strategy (3). They show a marvelous figure in their review showing signaling cycles that are driven by “demand for very salty foods,” “soft drinks and mineral waters,” and “weight at no cost.” All three of these parameters are driven by that capitalist evil, “profit.” It makes one nostalgic for the planned economy and those great collective farms! Oddly, the socialist countries do not seem to eat less salt. The authors go on to state, “The reasons that the food industry adds large

amounts of salt to processed foods is mainly because it makes cheap, unpalatable food edible at no cost.” So, there you have it.

What is new in the commentary by McCarron *et al.* (1)? Not too much. The theme seems to be that humans eat the amount of salt that they do because the intake is a “physiologically set parameter with a relatively narrow range intended to assure optimal function of a myriad of physiologic systems.” This contention is very difficult to prove. Indeed, 48 of 52 Intersalt centers (7) ate more salt than the Institute of Medicine would wish, but as He and MacGregor would maintain (3), the intake might be “industry driven,” even in the socialist countries. As evidence for their “set point” argument, McCarron *et al.* (1) cite a review by one of the authors (8). The review is excellent, aside from the range of salt intake studied (up to 8%), and draws attention to novel findings regarding aldosterone-sensitive neurons in the nucleus tractus solitarius that express hydroxysteroid dehydrogenase 2. The neurons are activated when salt is restricted and shut down when salt is abundant; however, these findings do not directly address the issue of why, in woman and man, sodium intake generally revolves around 150 to 170 mmol/d. As a matter of fact, Brown *et al.* (4)—the authors include a major Intersalt investigator—claimed that we only need 10 to 20 mmol/d (4). I am not sure why they would allow us the 20 mmol/d, because the Yanamamo Indians (record holders on sodium reticence) ate but 10 mmol/d, and surely those with relish. The McCarron group (1) then show a figure from the British Foods Standards Agency that conducted six surveys of salt intake within the United Kingdom since Intersalt. Salt intake in the past 25 yr has not changed, although He and MacGregor would attribute that to the greed of British food manufacturers (3). Finally, McCarron *et al.* (1) show a graph depicting a mean sodium intake of 160 mmol/d spanned by 2 SD (120 to 220 mmol/d); however, this figure is no evidence that God intended things to be this way. The group also cites the TOPH studies, favorites also of He and MacGregor (3). The latter authors used the studies to document the long-term reduction of cardiovascular events (15 yr later). The TOPH protocol called for a sodium reduction from 180 to 80 mmol/d for 3 yr (1); however, try as they may, the TOPHers could not lower sodium below 138 mmol/d after 3 yr. Those whose minds are made up will not be swayed by these findings.

There are other dietary issues confronting the treatment of our patients. Since I left the United States, the rate of obesity in adults has roughly tripled in the country from 11.5% in 1990 to 34.1% in 2004 (9). The problem is not confined to the United States. Could it be the gustatory effects of salt? To argue that the

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calorie-appetite set point has suddenly been increased would be disingenuous, although the serving size in the United States seems to have doubled, a state of affairs that allows my wife and me to order only one dinner in restaurants when we come to visit. In any event, aside from surgical treatment, compared with usual care, dietary counseling interventions for obesity produced “only modest weight losses that diminish over time” (10). Thus, there are big-time food problems that confront us, for which I certainly have no answer; however, I am glad that I do not belong to the Institute of Medicine.

Disclosures

None.

References

1. McCarron DA, Geerling JC, Kazaks AG, Stern JS: Can dietary sodium intake be modified by public policy? *Clin J Am Soc Nephrol* 4: 000–000, 2009
2. Mohan S, Campbell NR: Salt and high blood pressure. *Clin Sci (Lond)* 117: 1–11, 2009
3. He FJ, MacGregor GA: A comprehensive review on salt and health and current experience of worldwide salt reduction programmes. *J Hum Hypertens* 23: 363–384, 2009
4. Brown IJ, Tzoulaki I, Candeias V, Elliott P: Salt intakes around the world: Implications for public health. *Int J Epidemiol* 38: 791–813, 2009
5. Graudal N, Galløe A: Should dietary salt restriction be a basic component of antihypertensive therapy? *Cardiovasc Drugs Ther* 14: 381–386, 2000
6. Institute of Medicine: Strategies to Reduce Sodium Intake, 2009. Available at: <http://www.iom.edu/CMS/3788/59128.aspx>. Accessed September 1, 2009
7. Intersalt: An international study of electrolyte excretion and blood pressure—Results for 24 hour urinary sodium and potassium excretion. Intersalt Cooperative Research Group. *BMJ* 297: 319–328, 1988
8. Geerling JC, Loewy AD: Central regulation of sodium appetite. *Exp Physiol* 93: 177–209, 2008
9. Jeffery RW, Sherwood NE: Is the obesity epidemic exaggerated? No. *BMJ* 336: 244, 2008
10. Dansinger ML, Tatsioni A, Wong JB, Chung M, Balk EM: Meta-analysis: The effect of dietary counseling for weight loss. *Ann Intern Med* 147: 41–50, 2007

See related article, “Can Dietary Sodium Intake be Modified by Public Policy?” on pages 000–000.