Reply to the letter to the editor by Louise Weschler entitled

‘Under what conditions is ingested sodium rendered osmotically inactive’?

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First of all I would like to note that I support every single point Dr. Weschler made in her discussion of the two papers (2; 3).

However, I would also like to underline further the differences in study designs between our examinations in humans and Seeliger’s examinations in dogs. As outlined by Weschler, the dogs in Seeliger’s experiments were given food and fluid once a day in the morning between 8:30 and 9:00 am. In our human study, the test subjects were provided with 3 main meals (8:00 am, 1:00 and 7:00 pm) and 3 snacks (11:00 am, 4:00 and 9:00 pm) in which the daily sodium amount was distributed. The entire amount of sodium was given by dietary intake and fluid supplied together with the meals. This is a significant difference in study design, because the entire sodium regulation system has been challenged six times a day by high salt intake in our study compared to only once per day in Seeliger’s experiment.

As already mentioned by Weschler, fluid intake was kept constant at a level of 40 ml/kg body weight/day in our study, while the dogs received 100 ml/kg body weight/day. Considering our subjects’ daily fluid and sodium intakes, the calculated concentration of ingested sodium was about 214 mEq/L, i.e. a clearly hypertonic saline solution. However, fluid intake of three liters per day, of which two liters are taken as beverages, is already higher than average in Germany. The average day-by-day fluid intake in Germany is rather low, especially in elderly people. One might argue that the chosen level of sodium intake in our study is extremely high. Yes, this is true. But based on salt intake levels in the Western world and the average fluid intakes, it is not uncommon that –calculated as the concentration of sodium- the daily fluid intake be hypertonic. Under the chosen study conditions, which, albeit to some extent extreme, indeed do reflect characteristics of nutritional
habits, positive sodium balances occurred without any changes in potassium excretion or extracellular volume that inevitably led to osmotically inactive sodium storage.

Another difference between these studies is the level of potassium intake. Although in our study we did not balance potassium intake, our test subjects received an intake, independent of body weight, between 50 and 100 mmol/d (meaning 0.65 to 1.3 mmol potassium/kg body weight/d) provided by meals. In the studies by Seeliger, potassium intake was 3.5 mmol/kg body weight/d, which was about 3 to 5 five times higher than in our study and the recommended intake for humans in Germany (1). This high potassium intake could also have influenced Na⁺/K⁺ exchange.

In their experiments, Seeliger et al. (3) went directly from a low salt intake (0.5 mmol/kg body weight/d) to a high salt intake (5.5 mmol/kg body weight/d). A study group with average normal sodium ingestion was apparently not examined. Now, one might speculate that the onset level of sodium intake rather than the total body sodium (TBS) content is important in the regulation of sodium balance because the organism is adapted to a certain level of intake. More precisely, the effect when starting from a low-sodium diet and then switching to an average sodium intake might be different from that of starting from an average sodium intake and then switching to a high-sodium diet. So, it might well be that in the first case the increase in TBS is compensated by fluid retention, i.e. the undisputed physiological mechanism. This would most likely fit very well with the correlation Seeliger et al. (3) found in their dogs. However, in the second case, i.e. when starting from an average intake level and then switching to a high-sodium diet, the mechanism might be different, namely, sodium is stored in an osmotically inactive form. Serum sodium levels would increase
in both cases, and sodium has to be excreted by the kidney, but the maximum excretion capability of the kidney might have already been reached when being on an average intake level. Then, other mechanisms have to compensate for the increased serum sodium level. Because there was no intermediate sodium level in the studies by Seeliger et al. (3), this would mean that increasing sodium intake from low to high intake demands activation of both mechanisms: compensation by fluid retention and storage of osmotically inactive sodium. Obviously, total body fluid content and TBS must correlate in Seeliger's (3) studies because total body fluid is retained up to a certain level.

Certainly, additional studies (animal and human) are mandatory to further improve our knowledge of sodium and water metabolism, including the capability to store sodium in an osmotically inactive way.

Reference List

