Letter To The Editor

Reply to Weschler

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TO THE EDITOR: We appreciate Dr. Weschler’s (5) interest in our paper (1) and gladly take the opportunity to address the two points she raises in her letter, i.e., that 1) “Baker et al.’s findings may represent the first demonstration of osmotic activation and inactivation [of sodium] in controlled experimental conditions” and 2) the experiments in Baker et al.’s paper were “inadequate to test the hypothesis that weight maintenance means addition of body water.”

Weschler’s first point misinterprets our results. In her letter, Weschler wrote that “Baker et al. found that the changes in serum [Na] were less than predicted when serum [Na] both increased and decreased.” However, the reported results showed no significant difference between the measured and predicted serum [Na⁺] during the −4%, −2%, or 0% change in body mass (ΔBM) trials (i.e., when measured serum [Na⁺] increased or did not change from baseline). When subjects drank more fluid than they lost during the hyperhydration trials (i.e., gained 2% of BM), the predicted decrease in serum [Na] was greater than the measured decrease in serum [Na]. In the latter case we proposed a more plausible explanation, stating “Because of the high rate of urine excretion during the +2% ΔBM trials, subjects drank a substantial volume (~900 ml) of fluid during the 50-min recovery period to compensate for urine losses and maintain +2% ΔBM. It is possible that fluid absorption was not complete and a portion of the ingested fluid volume remained in the subjects’ stomachs at the time of post-experiment BM measurements. Accordingly, at the end of the 50-min recovery period, subjects rated their level of stomach bloating and sloshing of stomach contents significantly higher during the +2% ΔBM vs. the 0%, −2%, and −4%, ΔBM trials. Consequently, measured serum [Na⁺] was not as diluted as would be predicted by the ΔTBW (total body water) term in the Nguyen-Kurtz equation (1).” We stand behind the veracity of our data and the strength of its interpretation. We do not agree that our findings support the osmotic activation/inactivation of sodium hypothesis.

Dr. Weschler’s second point, that more work (i.e., a longer running protocol) would result in more fuel oxidation and more metabolic water production, is well taken. However, realistically, endurance athletes are going to ingest carbohydrate to replace oxidative fuel losses, especially if more work is performed. Additionally, metabolic water production (~0.13 g/kcal) is offset by respiratory water loss (~0.12 g/kcal) (2, 3), so this results in water turnover with no net ΔTBW, regardless of exercise duration. Furthermore, because glycogen stores are limited (whether glycogen loaded or not), whatever role the release of water complexed to glycogen plays in contributing to total body water would likely occur within our study time frame (2 + h). Therefore, we maintain that our experiment was adequate to refute the notion (4) that 0% ΔBM results in a net increase in TBW during exercise.

REFERENCES


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