The following is the abstract of the article discussed in the subsequent letter:

**Vrijens, D. M. J., and N. J. Rehrer.** Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. *J Appl Physiol* 100: 1847–1851, 1999.—This study assessed whether replacing sweat losses with sodium-free fluid can lower the plasma sodium concentration and thereby precipitate the development of hyponatremia. Ten male endurance athletes participated in one 1-h exercise pretrial to estimate fluid needs and two 3-h experimental trials on a cycle ergometer at 55% of maximum O2 consumption at 34°C and 65% relative humidity. In the experimental trials, fluid loss was replaced by distilled water (W) or a sodium-containing (18 mmol/l) sports drink, Gatorade (G). Six subjects did not complete 3 h in trial W, and four did not complete 3 h in trial G. The rate of change in plasma sodium concentration in all subjects, regardless of exercise time completed, was greater with W than with G (−2.48 ± 2.25 vs. −0.86 ± 1.61 mmol·1·h−1, P = 0.0198). One subject developed hyponatremia (plasma sodium 128 mmol/l) at exhaustion (2.5 h) in the W trial. A decrease in sodium concentration was correlated with decreased exercise time (R = 0.674; P = 0.022). A lower rate of urine production correlated with a greater rate of sodium decrease (R = −0.478; P = 0.0447). Sweat production was not significantly correlated with plasma sodium reduction. The results show that decreased plasma sodium concentration can result from replacement of sweat losses with plain W, when sweat losses are large, and can precipitate the development of hyponatremia, particularly in individuals who have a decreased urine production during exercise. Exercise performance is also reduced with a decrease in plasma sodium concentration. We, therefore, recommend consumption of a sodium-containing beverage to compensate for large sweat losses incurred during exercise.

**What can be concluded regarding water versus sports drinks from the Vrijens-Reher experiments?**

**To the Editor:** Although there is no evidence that sports drink ingestion reduces the risk of hyponatremia in those athletes who overdrink (2, 7), it might still be argued that sodium ingestion offers some protection against hyponatremia in those athletes who drink only enough to maintain weight during exercise. A paper cited in support of this latter hypothesis is Vrijens and Rehrer 1999 (6). However, the conclusion of this paper, that maintaining euhydration via ingestion of a sports drink resulted in a smaller reduction in plasma sodium concentration than ingestion of water under the same conditions, can be questioned for several reasons.

Of 10 fit subjects (maximum O2 consumption = 63 ml O2·min−1·kg−1) subjects, only 4 could complete both water (W) and Gatorade (G) trials. Moreover, “several blood samples from two subjects were lost; therefore, these two subjects were excluded from the statistical analyses of blood parameters.” The authors state that only data from the four finishers were used for plasma sodium, but it is not clear whether this applies to Table 1, (where “n” is reported as 10), or Fig. 1, or both.

It is important to note the high rate of nonfinishing. In similar harshly warm experimental conditions, fit athletes have been observed to reach “volitional exhaustion” as a function of increase in core temperature (4, 5). Thus the possibility that subjects’ core temperatures increased despite euhydration should be explored. Indeed, one subject’s trial was terminated for this very reason: the core temperature reached 39.5°C.

The authors’ most important conclusion is that “plasma sodium decrease was greater (P = 0.0007) with W than with G (Fig. 1)” (p. 1848, results) is inconsistent with another of their conclusions, that “a lower rate of urine production correlated with a greater rate of sodium change” (p. 1847, Abstract). G had a lower rate of urine production (0.082 ± 0.085 l/h) than W (0.119 ± 0.1 l/h), requiring, according to the authors, a greater rate of sodium decrease for G.

In the discussion, the authors erroneously predict that ingesting 2.4 liters of Gatorade with its 43 mmol sodium “would theoretically provide an increase of 3.9 mmol/l of sodium” (p. 1851). To arrive at this number, the authors have divided the 43 mmol by the extracellular fluid volume. Thus their calculation is based on the incorrect premise that extracellular fluid volume will remain constant. Extracellular fluid volume will increase, as required by the restoration of osmotically, via the redistribution of body water from intracellular fluid to extracellular fluid (1, 3, 7). The Nguyen-Kurtz equation (3) (which is based on osmotic equilibrium) predicts only an ~1 mmol/l increase in plasma sodium concentration in this instance (7) instead of the authors’ 3.9 mmol/l (change in total body water is assumed to be nil per the experimental protocol).

**REFERENCES**


**REPLY**

We agree with Dr. Weschler that the legend to Fig. 1 in our paper (2) could have been clearer. All subjects (n = 10) were included in the figure. Although we had not intended for this to be a performance trial, a number of subjects could not finish the 3-h cycling protocol. Thus numbers varied at the various time points because not all subjects completed the 3 h, and for two subjects, in one treatment, plasma samples were lost. This is clearly stated in the results section on plasma sodium. The repeated-measures ANOVA was conducted on those who completed 3 h in both trials and for whom we had plasma at all time points (n = 4). Despite this small number, the difference in plasma sodium concentration between beverages was so consistent and substantial that the effect was highly significant (P = 0.0007).

Table 1 does not contain plasma sodium, nor should it. Table 1 contains data from all 10 subjects, as stated. To be able to

Louise B. Weschler
161 Richdale Rd.
Colts Neck, New Jersey
e-mail: weschler@optonline.net
make comparisons, we therefore reported pertinent data as rates of change; thus all subjects’ data could be used irrespective of the duration of exercise.

Our subjects were well trained but not heat adapted, living in the Dunedin region, which has a cool to temperate climate year round. Some subjects stopped early because of volitional exhaustion, and one was stopped due to the ethical constraint that we stop subjects at rectal temperature \( (T_{re}) = 39.5^\circ C \). There is substantial evidence from the literature to support the finding that hyperthermia can occur without dehydration. González-Alonso et al. (1) observed that trained cyclists all fatigued at a similar level of hyperthermia \( [\text{esophageal temperature (Tes)} = 40.1\text{–}40.2^\circ C] \) when exercising (60% maximal \( O_2 \) uptake) in the heat (40°C). Tes is typically lower than \( T_{re} \), and among our subjects only one reached \( T_{re} = 39.5^\circ C \).

The ANOVA over time conducted on plasma sodium concentration clearly demonstrated a greater decrease with pure water than with the typical sports drink, which contained 18 mM of sodium. There was no significant difference in the mean rate of urine production between the two treatments, as was shown in Table 1. There was large interindividual variation in urine production. However, regression analysis including all subjects across both treatments demonstrated a clear inverse correlation between rate of urine production and rate of change in plasma sodium concentration. We never implied causation and did not state that “G had a lower rate of urine production” or that this required “a greater rate of sodium decrease for G.”

Dr. Weschler is correct in that there would be some shift from the intracellular fluid to the extracellular fluid and that our rough calculation of the expected change in plasma sodium concentration was inaccurate. However, her calculation also contains error because sodium lost from the extracellular fluid via sweat and urine was not taken into account. A precise calculation would be extremely difficult without accurate quantification of these losses. However, the minor error in calculation of expected sodium change does not alter the conclusion drawn, which remains substantiated by the changes in measured plasma sodium concentration.

REFERENCES


N. J. Rehrer
School of Physical Education
Otago University
Dunedin, New Zealand
e-mail: nancy.rehrer@stonebow.otago.ac.nz