

Exercise-induced sweating decreases 24-h sodium balance compared to rest in recreational exercisers

H. Z. Macrae¹, T. Cable¹, L. Mougin¹, H. Nuttall¹, E. Liddell¹, B. Bellisario¹, D. Locke¹, D. Miller¹, A. J. McCubbin², M. P. Funnell³, V. L. Goosey-Tolfrey⁴ and L. J. James¹

¹School of Sport, Exercise and Health Sciences, Loughborough University, UK

²Department of Nutrition, Dietetics and Food, Monash University, Australia

³NIHR Applied Research Collaboration Centre - East Midlands, Leicester Diabetes Centre, UK

⁴Peter Harrison Centre for Disability Sport, Loughborough University, UK

Daily sodium intake in England is ~3.3 g/day⁽¹⁾, with government and scientific advice to reduce intake for cardiovascular health purposes having varying success⁽²⁾. Eccrine sweat is produced during exercise or exposure to warm environments to maintain body temperature through evaporative cooling. Sweat is primarily water, but also contains appreciable amounts of electrolytes, particularly sodium, meaning sweat sodium losses could reduce daily sodium balance without the need for dietary manipulation. However, the effects of sweat sodium losses on 24-h sodium balance are unclear.

Fourteen active participants (10 males, 4 females; 23 ± 2 years, 45 ± 9 mL/kg/min) completed a preliminary trial and two 24-h randomised, counterbalanced experimental trials. Participants arrived fasted for baseline (0-h) measures (blood/urine samples, blood pressure, nude body mass) followed by breakfast and low-intensity intermittent cycling in the heat (~36°C, ~50% humidity) to turnover $\sim 2.5\%$ body mass in sweat (EX), or the same duration of room temperature seated rest (REST). Further blood samples were collected post-EX/REST (1.5-3 h post-baseline). During EX, sweat was collected from 5 sites and water consumed to fully replace sweat losses. During REST, participants drank 100 mL/h. Food intake was individually standardised over the 24-h, with bottled water available adlibitum. Participants collected all urine produced over the 24-h and returned the following morning to repeat baseline measures fasted (24-h). Sodium balance was estimated over the 24-h using sweat/urine losses and dietary intake. Data were analysed using 2-way ANOVA followed by Shapiro-Wilk and paired t-tests/Wilcoxon signed-rank tests. Data are mean (standard deviation).

Dietary sodium intake was 2.3 (0.3) g and participants lost 2.8 (0.3) % body mass in sweat (containing 2.5 (0.9) g sodium). Sodium balance was lower for EX (-2.0 (1.6) g vs -1.0 (1.6) g; P = 0.022), despite lower 24-h urine sodium losses in EX (1.8 (1.2) g vs 3.3 (1.7) g; P = 0.001). PostEX/REST blood sodium concentration was lower in EX (137.6 (2.3) mmol/L vs 139.9 (1.0) mmol/L; P = 0.002) but did not differ at 0-h (P = 0.906) or 24-h (P = 0.118). There was no difference in plasma volume change (P = 0.423), urine specific gravity (P = 0.495), systolic (P = 0.324) or diastolic (P = 0.274) blood pressure between trials over the 24-h. Body mass change over 24-h was not different between trials (REST +0.25 (1.10) %; EX +0.40 (0.68) %; P = 0.663).

Sweat loss through low-intensity exercise resulted in a lower sodium balance compared to rest. Although urine sodium output reduced with EX, it was not sufficient to offset exercise-induced sodium losses. Despite this, body mass, plasma volume and blood sodium concentration were not different between trials, suggesting sodium may have been lost from non-osmotic sodium stores. This suggests sweat sodium losses could be used to reduce sodium balance, although longer studies are required to confirm this thesis.

References

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