Sodium Loading Aids Fluid Balance and Reduces Physiological Strain of Trained Men Exercising in the Heat

STACY T. SIMS1,2, LINDA van VLIET1, JAMES D. COTTER1, and NANCY J. REHRER1,2

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ABSTRACT

SIMS, S. T., L. van VLIET, J. D. COTTER, and N. J. REHRER. Sodium Loading Aids Fluid Balance and Reduces Physiological Strain of Trained Men Exercising in the Heat. Med. Sci. Sports Exerc., Vol. 39, No. 1, pp. 123–130, 2007. Purpose: This study was conducted to determine whether preexercise ingestion of a highly concentrated sodium beverage would increase plasma volume (PV) and reduce the physiological strain of moderately trained males running in the heat. Methods: Eight endurance-trained (VO2max: 58 mL·kg−1·min−1 (SD 5); 36 yr (SD 11)) runners completed this double-blind, crossover experiment. Runners ingested a high-sodium (High Na+: 164 mmol Na+·L−1) or low-sodium (Low Na+: 10 mmol Na+·L−1) beverage before running to exhaustion at 70% VO2max in warm conditions (32°C; 50% RH, Uv = 1.5 m·s−1). Beverages (~757 mL) were ingested in seven portions across 60 min beginning 105 min before exercise. Results: Trials were separated by 1–3 wk. Heart rate and core and skin temperatures were measured throughout exercise. Urine and venous blood were sampled before and after drinking and exercise. Results: High Na+ increased PV before exercise (4.5% (SD 3.7)), calculated from Hct and [Hb]), whereas Low Na+ did not (0.0% (SD 0.5); P = 0.04), and involved greater time to exercise termination in the six who stopped because of an ethical end point (core temperature 39.5°C; 57.9 min (SD 6) vs 46.4 min (SD 4); P = 0.04) and those who were exhausted (96.1 min (SD 22) vs 75.3 min (SD 21); P = 0.03; High Na+ vs Low Na+, respectively). At equivalent times before exercise termination, High Na+ also resulted in lower core temperature (38.9°C vs 39.3°C; P = 0.00) and perceived exertion (P = 0.01) and a tendency for lower heart rate (164 vs 174 bpm; P = 0.08). Conclusions: Preexercise ingestion of a high-sodium beverage increased plasma volume before exercise and involved less thermoregulatory and perceived strain during exercise and increased exercise capacity in warm conditions. Key Words: CITRATE, RUNNING, HYPERVOLUMIA, HYPERHYDRATION

Plasma volume (PV) is fundamental to cardiovascular function and may impact on work capacity and endurance exercise performance (23,32). Expansion of PV (hypervolemia) has been suggested to improve thermoregulation and exercise performance in the heat; however, this remains equivocal. The expansion of blood volume from expanded body water (hyperhydration) may reduce the cardiovascular and heat strain observed with exercise, thereby improving exercise performance. Many studies have examined methods for inducing hypervolemia, including infusion of crystalloid or colloid solutions or pre-exercise overdrinking of plain water or water–electrolyte solutions (1,25). Infusion is unrealistic for most circumstances, whereas overdrinking has caused only transient expansions of body water, with much of the fluid overload rapidly excreted, at least partly because of its hypotonicity. The use of solutions containing glycerol has induced modest hyperhydration and has resulted in limited and mixed results in terms of thermoregulatory and performance advantages, and has sometimes shown side effects (20,28). Studies in which plasma hyperosmolality was attenuated during exercise-induced heat stress have shown improved heat dissipation (6,7,24), but many have not addressed exercise performance (12,24).

Recently, “sodium loading” with a sodium concentrated beverage composed of sodium citrate and sodium chloride (164 mmol Na+·L−1) with moderate osmolality (253 mOsm·kg−1) has been shown to be effective in inducing hyperhydration and hypervolemia at rest (14,25,30). Compared with no fluid and with salt tablets with water (136.8 mmol Na+·L−1) this sodium-loading protocol elicited the greatest plasma volume expansion at rest (4,8). Greenleaf et al. (14,15) also observed PV expansion and enhanced cycling performance in men, in temperate conditions after sodium loading. It remains unknown, however, whether the same effect would be evident in trained males, who might be more inclined to use such an approach but who are already hypervolemic (11) and who show less cardiovascular benefit in exercise from artificially induced hypervolemia (9). Also, one would expect

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that a situation in which sodium loading would be particularly beneficial would be for orthostatically stressful exercise performed in the heat, because this involves both a very high demand for cutaneous perfusion and a pooling of blood. Therefore, the aims of this project were to determine whether ingestion of a highly concentrated sodium-citrate beverage would induce hypervolemia in trained individuals and enhance running capacity in the heat.

**METHODS**

**Subjects.** Eight healthy, nonsmoking, male, moderately trained runners, maximal oxygen consumption (VO_{2max}) 57.5 mL·min^{-1}·kg^{-1} (SD 5.4), age 36 yr (SD 11), body mass 75.2 kg (SD 6.7), height 179.5 cm (SD 5.5), completed this institutionally approved study, which was conducted in the University of Otago Environmental Chamber Laboratory for Human Performance. Each subject gave written informed consent.

**Experimental design.** A double-blind, placebo-controlled, crossover design was employed during the winter months of the southern hemisphere (June to September; average daily maximum outside temperature of 7–10°C, respectively) to control for heat acclimatization. Participants completed two treadmill trials to exhaustion in the heat (32°C, 50% RH, V_a ≈ 1.5 m·s^{-1}) at 70% of their temperate-environment VO_{2max}. An incremental treadmill VO_{2max} test was conducted on a separate day, 1–2 wk before experimental testing, to assess fitness and to set the intensity for the experimental trials. Participants then underwent a familiarization session to mimic the actual trial(s). Experimental trials were randomized, separated by 1–3 wk, and involved a run-to-exhaustion performance test after ingestion of 10 mL·kg^{-1} body mass (~759 mL) of a control, low-sodium beverage (Low Na^+, 10 mmol Na^+·L^{-1}, 0.58 g NaCl, 42 mOsm·kg^{-1}), or a high-sodium beverage (High Na^+, 164 mmol Na^+·L^{-1}, 7.72 g of sodium-citrate with 4.5 g NaCl, 253 mOsm·kg^{-1}) (15,16), chilled overnight at 4°C. Beverage was blind to the researcher conducting trials and to the participant. The allocation of the first beverage assignment was random for the first participant and alternated thereafter.

**Standardization.** To standardize training effects on PV, each participant maintained a training diary of duration, mode, and intensity of activity, which was replicated for consistency preceding each trial. Further, each participant completed a 40-min treadmill run at 50% VO_{2max} 48 h before each testing day and then refrained from training until the experimental trial. The same meal, of each participant’s choice (no alcohol or caffeine permitted) was consumed the evening before each testing session, and participants were required to drink 750 mL of water throughout that evening. They were asked to refrain from smoking or drinking alcohol and to avoid tea, coffee, cola (or other caffeinated beverages) on the day before and the day of the test. On the day of testing, participants reported to the lab, fully hydrated, after a standardized breakfast (1680 kJ, 13 g protein, 10 g fat, 63 g CHO, 265 mg Na^+), which was consumed between 2.5 and 2 h before experimentation. An additional 500 mL of water was given to participants between breakfast and the start of the testing protocol. Urine specific gravity was measured in an initial baseline sample to verify adequate hydration status before each test (U_{SG} ≤ 1.020).

**Protocol.** On arrival at 0900 h, participants voided before nude body mass was recorded (± 10 g). Each participant was then seated for catheter placement (22-gauge Teflon intravenous catheter) in a suitable vein of the left arm. To ensure steady-state plasma volume and constituents, participants stood for 20 min before baseline blood samples were taken.

**Sodium loading.** On withdrawal of the baseline blood sample, the participant began ingesting the beverage, which was measured (659–870 mL) in seven equal portions, one every 10 min (Fig. 1). During this drinking period, they were required to walk approximately 1 min every 20 min to limit venous pooling; however, they were required to stand in one place for 20 min before blood sampling (15). To avoid any perceptive taste variations in the beverages, the participants were instructed that the study was designed to investigate varying concentrations of sodium in a preexercise beverage; additionally, each beverage was chilled overnight at 4°C and remained refrigerated until being partitioned into individual boluses.

FIGURE 1—Experimental protocol. S, sampling (VO_{2}, VCO_{2}, perceived exertion, heart rate, and skin and rectal temperatures); B, blood sampling; BB/WU, measurement of body mass (kg), urine osmolality, plasma and urine [Na^+], and urine specific gravity; D, drink bolus; EXH, volitional exhaustion or T_{rec} = 39.5°C.
Each bolus was left at room temperature (20 ± 2°C) for 5 min before consumption. There were no comments from participants on flavor being salty regardless of High Na⁺ or Low Na⁺ beverage.

**Exercise testing.** Participants entered the climatically controlled chamber (32°C, 50% RH, \( V_{\text{a}} \approx 1.5 \text{ m s}^{-1} \)) 45 min after consuming the beverage and commenced running on a treadmill, without feedback cues of time or distance. The speed of the treadmill was set to eliciting 70% of the temperate-environment \( \dot{V}O_{2\text{max}} \). Exercise stopped when the participant could no longer maintain exercise at the given intensity or when the participant reached the ethically constrained rectal temperature limit of 39.5°C. Heart rate was recorded at 1-min intervals throughout exercise (Polar s120, Polar Electro Oy). Rectal temperature was measured with a disposable thermistor (Thermister 400, Mallinckrodt, St. Louis, MO), which was disinfected and reused within participants. Skin temperatures were measured with insulated thermistors (Type EU, Grant Instruments Ltd, Cambridge, UK) at four sites: biceps, calf, chest, and thigh, from which mean skin temperature was calculated (5). Temperatures were recorded at 30-s intervals (Model 1200, Grant Instruments Ltd, Cambridge, UK). Carbon dioxide production, \( \dot{V}O_2 \), and ventilation were measured for 2-min periods at 5-min intervals until 30 min into exercise, then at 15-min intervals using a gas-analysis system (Cortex Metalyser 3B, Borsdorf/Leipzig Germany). Urine was collected at baseline (−105 min), 20 min after drinking (−45 min), immediately before exercise (0 min), and at exhaustion. Blood samples were taken immediately before urine sampling (Fig. 1).

**Blood and urine analysis.** All blood samples (12 mL each) were separated into aliquots for analysis of hematocrit (Hct), hemoglobin concentration (Hb), plasma sodium concentration ([Na⁺]), and osmolality. A 2-mL aliquot was analyzed immediately for [Hb] (Hemoximeter, OSM3 Radiometer, Copenhagen, Denmark) and Hct, in quadruplicate. Blood for Hct was drawn into capillary tubes and centrifuged for 6 min at 3000 rpm (Hawksley Microcentrifuge, Sussex, UK) and read using a modified microcapillary tube reader (Damon/IEC division, Needham Heights, MA); the measurement error was ±0.25%.

The remaining two additional aliquots of blood (5 mL each) were transferred into tubes containing lithium heparin and were centrifuged for 10 min at 6°C and 3000 rpm (Model GS-15R Centrifuge, Beckman-Coulter, Fullerton, CA). Plasma osmolality was measured using vapor-point depression (Osmometer, Model Vapro5520, Wescor Inc, Logan, UT). Urine [Na⁺] and plasma [Na⁺] and [K⁺] were measured using the Cobas Mira Plus analyzer, ion-selective electrode (Roche). Urine specific gravity was measured in triplicate with a handheld refractometer (ATAGO Co Ltd, Tokyo, Japan). Because electrolyte analyses were not performed immediately, plasma and urine samples were put on ice and stored at −80°C until analyses.

**Calculations.** Changes in PV from baseline were estimated from changes in Hct and [Hb] using the following equation (2):

\[
\%\Delta PV = 100[(\text{Hb}t/\text{Hb}0)((1 - \text{Hct}t)/(1 - \text{Hct}0))] - 100\%
\]

in which subscripts \( t \) and 0 denote measurements at time \( t \) and at baseline (−105 min), respectively. Hb is in grams

### TABLE 1. Individual body temperatures, performance, heart rate, and ratings of perceived exertion.

<table>
<thead>
<tr>
<th>Time comparison</th>
<th>Rectal</th>
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<th>Rectal</th>
<th>Exh</th>
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<td>125 min</td>
<td>48 min</td>
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<tr>
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**HR (bpm)**

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<th>59 min</th>
<th>125 min</th>
<th>48 min</th>
<th>38 min</th>
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<th>53 min</th>
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**RHR (bpm)**

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Reasons for ending trial: exh, exhaustion; rectal, rectal temperature (\( T_{\text{rec}} \)) > 39.5 °C; time comparison = the means of the collected data every 30 s for 2.5 min before the end point of the shortest exercise trial. \( P \) values are for paired \( t \)-test comparisons between High Na⁺ and Low Na⁺ means. RHR, resting heart rate.
per 100 mL and Hct is a fraction. Hct was multiplied by 0.96 and then 0.91 to correct for trapped plasma and the venous-to-whole-blood Hct excess, respectively (2). Sweat loss was estimated by change in body mass corrected for urinary and blood losses and fluid intakes. Rates of sweat loss were approximated by dividing by exercise time. Individuals’ final temperatures were calculated as the mean of the five measurements recorded during the 2.5 min before the end point of the shortest exercise trial.

Statistical analysis. Significance of effects of beverage and time on plasma volume, plasma osmolality, respiratory exchange ratio (RER), and plasma sodium concentration were established by two-way repeated-measures ANOVA. Bonferroni-corrected post hoc tests were used to determine differences between significant means. Paired t-tests were used to determine differences in fluid loss, urinary loss, mass loss, rate of change in urine [Na\(^+\)], urine and plasma osmolality, RER, time to exercise termination, and slope of heart rate and rectal temperature. Relationships between selected dependent measures were conducted using Pearson product correlations. Differences were considered statistically significant when \( P < 0.05 \). Values are reported as means (SD).

RESULTS

Participant compliance. Training and diet logs were collected and reviewed on the morning of the second experimental session. Hydration compliance was additionally checked via urine specific gravity (\( U_{\text{SG}} \leq 1.020 \)). All participants apparently complied with standardization procedures requested.

Exercise tolerance. Core temperature increased during exercise in both conditions, reaching the 39.5°C limit in six of eight subjects in the Low Na\(^+\) trial and in five subjects in the High Na\(^+\) trial (Table 1). A greater time to exercise termination occurred in those who were stopped because of the ethically constrained end point of 39.5°C and in those who reached volitional exhaustion (39.5°C; 57.9 min (SD 6) vs 46.4 min (SD 4); \( P = 0.04 \); exhaustion: 96.1 min (SD 22) vs 75.3 min (SD 21); \( P = 0.03 \); High Na\(^+\) vs Low Na\(^+\), respectively, Fig. 2). Individual results are recorded in Table 2. A ninth participant, E, completed only one of two trials, withdrawing from the study, and thus none of his data were included in the results. Individual performance and temperature data are presented in Tables 1 and 2. The time-matched final rating of perceived exertion (RPE) was higher in Low Na\(^+\) than in High Na\(^+\) trial (\( P = 0.04 \), Table 2). The respiratory exchange ratio remained equivalent between beverage conditions (Fig. 2), although this analysis was restricted to seven participants because of failure of online gas analyses during one of the trials.

Cardiovascular and thermal responses. After drinking, plasma volume was expanded before exercise in High Na\(^+\) (4.5% (SD 3.7)) but not in Low Na\(^+\) (0.0% (SD 0.5), \( P = 0.04 \)). Plasma volume decreased immediately before exercise in both trials (Fig. 3). The small declines in PV during exercise were similar for both High Na\(^+\) and Low Na\(^+\) (−2.5% (SD 2.6) and −3.1% (SD 3.4), \( P = 0.90 \)). Heart rate averaged 157 bpm (SD 11) during exercise in High Na\(^+\) and 161 bpm (SD 16) in Low Na\(^+\) (\( P = 0.88 \)). Whereas the average cardiovascular drift (i.e., the rate of rise after the initial 5 min of exercise) in Low Na\(^+\) was twice that in High Na\(^+\) (0.44 vs 0.22 min\(^{-1}\)), and the time-matched final heart rate was higher (Table 1), these

![FIGURE 2—Respiratory exchange ratio (RER) during exercise to exhaustion at approximately 70% \( \dot{V}O_{2\text{max}} \) in warm conditions (32°C, 50% RH, \( V_{\text{se}} = 1.5 \text{ m·s}^{-1} \)) after ingestion of a high-sodium beverage (High Na\(^+\), 164 mmol Na\(^+\)·L\(^{-1}\)) or a low-sodium beverage (Low Na\(^+\), 10 mmol Na\(^+\)·L\(^{-1}\); \( P = 0.045 \)). N = 7 because of failure of online gas analyses during one trial.](http://www.acsm-msse.org)

![FIGURE 3—Mean changes in plasma volume before exercising to exhaustion at approximately 70% \( \dot{V}O_{2\text{max}} \) in warm conditions (32°C, 50% RH, \( V_{\text{se}} = 1.5 \text{ m·s}^{-1} \)) after ingestion of a high-sodium beverage (High Na\(^+\), 164 mmol Na\(^+\)·L\(^{-1}\)) or a low-sodium beverage (Low Na\(^+\), 10 mmol Na\(^+\)·L\(^{-1}\)). Values are presented as means (SD). Repeated-measures ANOVA indicated a significant beverage effect (*\( P = 0.038 \)).](http://www.acsm-msse.org)

<p>| TABLE 2. Individual performance times. |
| High Na(^+) | Low Na(^+) | Δ Time |</p>
<table>
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<tr>
<th>(min)</th>
<th>(min)</th>
<th>(min)</th>
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<tr>
<td>A</td>
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<td>47.0</td>
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<tr>
<td>B</td>
<td>82.2</td>
<td>47.0</td>
<td>35.3</td>
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<tr>
<td>C</td>
<td>85.2</td>
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<tr>
<td>D</td>
<td>138.3</td>
<td>125.9</td>
<td>10.4</td>
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<tr>
<td>F</td>
<td>59.0</td>
<td>48.8</td>
<td>10.2</td>
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<tr>
<td>G</td>
<td>59.3</td>
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<tr>
<td>H</td>
<td>70.0</td>
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<td>16.8</td>
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<tr>
<td>I</td>
<td>37.4</td>
<td>38.4</td>
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differences were not statistically significant (both $P = 0.08$). Mean $T_{rec}$ and mean $T_{skin}$ increased over time in both trials ($P < 0.001$), although no treatment effect was observed. Yet, the time-matched final $T_{rec}$ was lower in High Na$^+$ than in Low Na$^+$ ($P = 0.00$; Table 1).

**Discussion**

The current study demonstrated for the first time in trained men that an acute sodium-fluid load increases plasma volume at rest and reduces physiological strain in warm conditions (under our ethical and environmental conditions). The fluid balance effects observed here support the plasma volume expansion observed in the few previous studies that have used a highly concentrated sodium drink but that were conducted using less trained, and presumably less hypervolemic, individuals (14–16). As far as we know, the reduced physiological strain during exercise is novel with regard to the sodium-loading strategy and heat stress.

**Plasma osmolality and [Na$^+$].** The overall increase in plasma osmolality during exercise was not significantly different between High Na$^+$ and Low Na$^+$; however, the rate of change in osmolality with High Na$^+$ was slower than that of Low Na$^+$ ($P = 0.00$, Fig. 4). Plasma [Na$^+$] was stable at rest after drinking for both High Na$^+$ and Low Na$^+$, with no significant differences between conditions ($P = 0.66$). Plasma [Na$^+$] increased slightly during exercise, by 0.8 mmol Na$^+$L$^{-1}$ for High Na$^+$ and by 2.8 mmol Na$^+$L$^{-1}$ for Low Na$^+$. The increase over the whole trial tended to be larger in Low Na$^+$ than in High Na$^+$ ($P = 0.06$; Fig. 5).

**Fluid balance.** After ingestion of approximately 757 mL of fluid in each condition, sweat rates and volumes were equivalent (Table 3a), whereas more urine was produced throughout the Low Na$^+$ trial than during the High Na$^+$ trial (Table 3b).

**Discussion**

The current study demonstrated for the first time in trained men that an acute sodium-fluid load increases plasma volume at rest and reduces physiological strain in warm conditions (under our ethical and environmental conditions). The fluid balance effects observed here support the plasma volume expansion observed in the few previous studies that have used a highly concentrated sodium drink but that were conducted using less trained, and presumably less hypervolemic, individuals (14–16). As far as we know, the reduced physiological strain during exercise is novel with regard to the sodium-loading strategy and heat stress.

**Plasma volume changes.** Previous studies involving acute expansion of PV and attenuation of plasma hyperosmolality during exercise heat stress have shown improved heat dissipation but have not addressed exercise performance (6,12,24). Greenleaf et al. (13) studied PV
The increases in PV after sodium loading in the present study (4.7%) were not as large as in previous studies. Apart from the obvious effect of drink volume, this might be attributable to the posture in which PV was measured. Participants were standing (with intermittent walking) in this study because that was their exercising posture. Standing increases filtration at dependent capillaries and, thus, decreases PV (2,18,19). Although the ingestion of High Na⁺ resulted in only a transient PV expansion, there was still fluid retention, as evidenced by reduced urinary output before commencing exercise. It is probable that most of the extra fluid was shifted to the interstitial compartment after its absorption. Thus, although the hypervolemic effect of sodium-fluid loading may have been limited by transcapillary fluid shifts, a sodium-plus-water expansion of the ECF would still have potential benefit during prolonged exercise via its availability for sweat and plasma. Moreover, the ingestion of water with the sodium load attenuates the rise in plasma [Na⁺] concentration during exercise. The modest increase in PV may also be attributable to our participants being more trained than in previous sodium-fluid-loading studies (1,4,12–15,24,25). That is, the reliable existence of a training-induced hypervolemia (18) would mean that a given volume of ingested fluid represents a smaller proportion of the PV and, thus, less relative expansion. Although only an estimation, it seems appropriate that the approximately 4% expansion of PV corresponds with the drink volume of 750 mL being 4% of 15 L, which would be approximately the ECF volume of these participants (13,16,30).

It is noteworthy that PV decreases reported in the literature were only the extra fluid was shifted to the interstitial compartment after its absorption. Thus, although the hypervolemic effect of sodium-fluid loading may have been limited by transcapillary fluid shifts, a sodium-plus-water expansion of the ECF would still have potential benefit during prolonged exercise via its availability for sweat and plasma. Moreover, the ingestion of water with the sodium load attenuates the rise in plasma [Na⁺] concentration during exercise. The modest increase in PV may also be attributable to our participants being more trained than in previous sodium-fluid-loading studies (1,4,12–15,24,25). That is, the reliable existence of a training-induced hypervolemia (18) would mean that a given volume of ingested fluid represents a smaller proportion of the PV and, thus, less relative expansion. Although only an estimation, it seems appropriate that the approximately 4% expansion of PV corresponds with the drink volume of 750 mL being 4% of 15 L, which would be approximately the ECF volume of these participants (13,16,30).

Exercise capacity. In addition to the PV expansion and fluid retention at rest with sodium loading, the other principal finding of this study was the significant improvement in exercise capacity in the heat after sodium loading. Although these findings are consistent with previous research using the same application of sodium loading (16) or other sodium-containing beverages (27), the novel finding is that the enhanced fluid retention was associated with reduced physiological strain in trained individuals, despite higher training status being associated with a relative insensitivity for cardiovascular effects to acutely induced hypervolemia (4,17). Moreover, increased tolerance to exercise after sodium loading has not always been supported (1,34), but those studies have used either a low sodium content (27) or temperate conditions (1). Greenleaf et al. (16) reported a significantly greater mean exercise time to exhaustion using this beverage compared with a multicomponent carbohydrate drink or control. They concluded that the higher expansion of PV just before exercise as well as a greater acid buffering and possible...
increased energy substrate from citrate might have contributed to the greater endurance. We also found an expansion of PV, the extent of which was reasonably closely related to the additional endurance duration, but we have no data to verify the possible roles of acid buffering or substrate delivery with this beverage.

Ethical restrictions (39.5°C Trec limit) prevented a definitive determination of exercise capacity in the current conditions, but Trec was, on average, 0.4°C higher in Low Na⁺ at end exercise, which was at the 39.5°C limit for six participants and was close to the levels associated with the fatigue coinciding with critically high core temperature (~40°C) reported previously for trained individuals (10,17,31). Moreover, linear modeling of the core temperature responses of these six participants predicted that if they had been allowed to continue exercising, their mean time to 40°C would have been an additional 28 min in High Na⁺ and approximately 9 min in Low Na⁺. At the 39.5°C termination point, High Na⁺ was also associated with a lower perceived exertion and a tendency for lower HR, and the exercise duration was modestly related to the extent of attenuation in both Trec (r = 0.919) and heart rate (r = 0.852). Which factor(s) caused exercise intolerance is unknown, but could potentially have included factors secondary to the higher cardiovascular and thermal strain, and perhaps helped mediate the higher exertion; factors such as local tissue ischemia, lactate accumulation, or higher glycogen use associated with higher catecholamines (8,18,26).

This study used a design in which participants were euhydrated at the outset but rehydration was not undertaken during the exercise bout. Hence, the effect of drinking during exercise was not examined. It is possible that effects of a high-sodium drink would be less evident or absent if drinking had been permitted during exercise, but it is also noteworthy that runners generally rehydrate only a minor proportion of their fluid losses during a competitive bout, even when running longer durations than used here (e.g., a marathon (22)). The optimal timing of intake was also not determined in this study and must be considered when applying sodium loading for hyperhydration purposes. Third-space movement of fluid is one issue here. Greenleaf et al. (13,15) and Mack and Nadel (21) have suggested that the integrated change over time in PV may be similar with beverages differing in osmolality, but the peak PV may differ because of different fluid shifts between the interstitial fluid and the gut.

CONCLUSIONS

Drinking fluids with a higher sodium concentration (with sodium citrate) than in regular sports drinks, before exercise, can elicit a transient hypervolemic response that is partly preserved (relative to a low-sodium drink) in exercise, and is associated with improved physiological tolerance to exercise in warm conditions in trained males. The ergogenic benefit is related to the degree of preexercise hyponatremia, but it is unclear whether cardiovascular and/or thermoregulatory mechanisms, or resultant mechanisms, are responsible for this. The performance enhancement after sodium loading found in this study was achieved with no further fluid ingestion during exercise. Because it is unlikely that athletes would attempt to hyperhydrate with sodium loading in the absence of any rehydration during exercise, in particular in hot and/or humid conditions, the benefit of sodium loading with additional drinking during exercise is, as yet, unknown.

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REFERENCES


29. Deleted in proof.


