Preexercise sodium loading aids fluid balance and endurance for women exercising in the heat

Stacy T. Sims,1 Nancy J. Rehrer,1,2 Melanie L. Bell,3 and James D. Cotter1
1School of Physical Education, 2Department of Human Nutrition, and 3Preventive and Social Medicine, University of Otago, Dunedin, New Zealand

Submitted 24 October 2006; accepted in final form 23 April 2007

Sims ST, Rehrer NJ, Bell ML, Cotter JD. Preexercise sodium loading aids fluid balance and endurance for women exercising in the heat. J Appl Physiol 103: 534–541, 2007. First published April 26, 2007; doi:10.1152/japplphysiol.01203.2006.—This study was conducted during the high-hormone phase of both natural and oral contraceptive pill (OCP)-mediated menstrual cycles to determine whether preexercise ingestion of a concentrated sodium beverage would increase plasma volume (PV), reduce physiological strain, and aid endurance of moderately trained women cycling in warm conditions. Thirteen trained cyclists (peak O2 uptake 52 ml·kg−1·min−1 (SD 2), age 26 yr (SD 6), weight 60.8 kg (SD 5)) who were oral contraceptive users (n = 6) or not (n = 7) completed this double-blind, crossover experiment. Cyclists ingested a concentrated-sodium (High Na+: 164 mmol Na+/l) or low-sodium (Low Na+: 10 mmol Na+/l) beverage (10 ml/kg) before cycling to exhaustion at 70% Peak O2 uptake in warm conditions (32°C, 50% relative humidity, air velocity 4.5 m/s). Beverage (~628 ml) was ingested in seven portions across 60 min beginning 105 min before exercise, with no additional fluid given until the end of the trial. Trials were separated by one to two menstrual cycles. High Na+ increased PV (calculated from hematocrit and hemoglobin concentration) before exercise, whereas Low Na+ did not [−4.4 (SD 1.1) vs. −1.9% (SD 1.3); 95% confidence interval: for the difference 5.20, 6.92; P < 0.0001], and it involved greater time to exhaustion [98.8 (SD 25.6) vs. 78.7 (SD 24.6) min; 95% confidence interval: 13.3, 26.8; P < 0.0001]. Core temperature rose more quickly with Low Na+ [1.6°C/h (SD 0.2)] than High Na+ [1.2°C/h (SD 0.2); P = 0.04]. Plasma [AVP], [Na+] concentration, and osmolality, and urine volume, [Na+], and osmolality decreased with sodium loading (P < 0.05) independent of pill usage. Thus preexercise ingestion of a concentrated-sodium beverage increased PV, reduced thermoregulatory strain, and increased exercise capacity for women in the high-hormone phase of natural and oral contraceptive pill-mediated menstrual cycles, in warm conditions.

Address for reprint requests and other correspondence: S. T. Sims, Sports Medicine-Human Performance Laboratory, Arrilarga Sports Center-Lower Level, Stanford Univ., Stanford, CA 94305-6150 (e-mail: stsims@stanford.edu).

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reduces the viscosity of blood, increases heat storage and transfer capacity, and increases exercising cardiac output and maximal $O_2$ uptake, at least in untrained individuals (15, 16, 25). The ability to offload heat, by virtue of the increased volume of circulating fluid, is also increased as circulation to the periphery is enhanced. However, fluid balance is often not achieved; indeed it is not even achievable when fluid availability or gastric emptying rates prevent the rates of ingestion and absorption from matching sweat rates (23). One method of inducing hyperhydration and hypervolemia, originally developed to help offset effects of PV loss in microgravity (3, 16), is “sodium loading.” A sodium-concentrated beverage composed of sodium citrate and sodium chloride (164 mmol Na+/l), with moderate osmolality (253 mosmol/kgH$_2$O), has been shown to expand PV, reduce cardiovascular strain, and aid exercise capacity of untrained and trained men in temperate and hot environments (13–16). Moreover, earlier studies (3, 12) showed that ingested saline solutions between 0.9 and 1.07% expanded PV over a 4-h postingestion time period. Frey and colleagues (12) determined that the 1.07% saline solution elicited the greatest PV expansion and urine concentration over the 4 h postingestion; however, the addition of 1% glucose did not improve the effectiveness of PV expansion, but it increased diuresis. Thus the authors concluded that a slightly hypertonic sodium-only solution provided the most effective means of PV expansion.

In view of the aforementioned compromised thermoregulatory factors that women experience in the high-hormonal phase of their menstrual cycle, the first aim of this study was to determine whether a similar hypervolemia-inducing protocol would be effective for women exercising in the heat. We hypothesized that preexercise ingestion of a high-sodium solution would expand PV, increase exercise endurance, lower the rate of rise of core temperature, reduce cardiovascular strain, and reduce perceived exertion during exercise in the heat. The second aim was to determine the effects of this hyperhydration strategy as a function of habituated OCP usage. We hypothesized that sodium loading would have a hypervolemic effect and benefit women’s exercise capacity in the heat. Additionally, because of the secondary effects of estrogen fluid balance, specifically that estrogen lowers the osmotic threshold for arginine vasopressin (AVP) release with a concomitant increase in sodium and fluid retention, and the greater concentration of estrogen associated with OCPs, we hypothesized that the hypervolemic effects of sodium loading would be attenuated with chronic OCP usage.

METHODS

Participants. Thirteen healthy, eumenorrheic, endurance-trained female cyclists (peak $O_2$ uptake ($V_{O_2peak}$) 52 ml·kg$^{-1}$·min$^{-1}$ (SD 2); age 26 yr (SD 6), weight 60.8 kg (SD 5)), with no history of cardiovascular or renal disease, no contraindications to OCPs, and not taking other medication, were recruited. Participants were separated into natural menstrual cycle (Nat, $n = 7$) or triphasic OCP-mediated (OCP, $n = 6$) groups according to their usage status. Before participation, all participants were informed of the nature and demands of the investigation, after which they gave written informed consent. The study was approved by the Human Ethics Committee of the University of Otago. Of the 13 participants, 12 were familiar with the experimental protocols and conditions, and all had participated in previous laboratory trials. The naïve participant was familiarized with the experimental testing procedures 2 wk before her first experimental session.

Preliminary testing. Before performance testing, all participants underwent an incremental cycling test to exhaustion to ensure that they met the criteria for being categorized as “well trained” [train $\geq$5 times/wk and have peak aerobic power ($V_{O_2peak}$) $>50$ ml $O_2$·kg body mass$^{-1}$·min$^{-1}$]. The $V_{O2peak}$ test was conducted on a separate day, in temperate conditions (19 ± 2°C), 1–2 wk before experimental testing, and was used to set the intensity for the experimental trials. This comprised a progressive incremental test to exhaustion undertaken on a computer-controlled, precision electromagnetically braked bicycle ergometer (Velotron, Racermate, Seatte, WA) during which $O_2$ consumption ($V_{O2}$), $CO_2$ production ($V_{CO2}$), and heart rate (HR) were measured. After a 5- to 10-min warm-up at a self-selected intensity, the incremental test commenced at a workload of 2.25 W/kg, which increased by 40 W after each 150 s until the participant’s HR reached 85% of the age-predicted maximum, after which the workload increased 25 W each 150 s until interval until the subject could no longer maintain the required power output and/or the pedaling frequency dropped below 75 revolutions/min. During the $V_{O2peak}$ test, as well as during subsequently described experimental trials, HR was recorded using a HR monitor (PE 4000, Polar Electro, Kempele, Finland), and $V_{CO2}$, $V_{O2}$, and ventilation were measured on a breath-by-breath basis using an automated respiratory gas analysis system (Cortex Metalyser 3B, Borsdorf, Leipzig, Germany).

Experimental testing. A double-blind, placebo-controlled, crossover design was employed during the winter months of the southern hemisphere (June to September) to control for heat acclimatization. Experimental trials occurred on days 20 or 21 for an endogenous 28-day cycle and on days 18–20 (end of the third week of active pills/high-hormone phase) for a triphasic OCP cycle. Participants were asked to self-monitor menstrual cycle by recording the brand name and cycle of the pill or by recording basal temperature. Day of testing varied from participant to participant because of normal fluctuations in individual cycle length (30). The order of testing the two sodium loads was counterbalanced and randomized. Participants completed two cycling trials to exhaustion at 70% of their temperate-environment $V_{O2}$ in the heat (32°C, 50% relative humidity, air velocity ~4.5 m/s). The performance trial was conducted following ingestion of 10 ml/kg body mass of a control, low-sodium beverage (Low Na$^+$; 10 mmol Na+/l, 43 mosmol/kgH$_2$O) or a concentrated-sodium beverage (High Na$^+$; 164 mmol Na+/l, 253 mosmol/kgH$_2$O) (14–16), chilled overnight to 4°C. Beverage was blind to the researcher conducting trials and to the participant. To minimize order effects, the allocation of the first beverage assignment was random for the first participant and thereafter was alternated.

Standardization. All women had regular menstrual cycles. For those women on the triphasic OCP, they had been following the same schedule of pills for at least 6 mo before testing. To verify phase of the menstrual cycle, plasma estrogen and progesterone concentrations were assessed from the baseline sample before the sodium-loading protocol was undertaken. To standardize the training effects on PV, each participant maintained a training diary of duration, mode, and intensity of activity, and they replicated this for consistency throughout the weeks preceding each trial. Participants completed a standardized 60-min exercise bout at 50% $V_{O2peak}$ in the laboratory, 24 h before each testing day, with no further exercise until experimental testing. Participants refrained from consuming soy-based products (because of phytoestrogen effects) and nonsteroidal anti-inflammatory drugs throughout the experimental period. The same meal (low-moderate sodium and no alcohol, soy, or caffeine permitted) was consumed the evening before each testing session, and the participant drank 750 ml of water throughout that evening before bed. An additional 500 ml of water was consumed between breakfast and the start of the testing protocol. On the day of testing, the participant reported to the laboratory, fully hydrated, after a standardized breakfast (provided; 13 g protein, 10 g fat, 63 g carbohydrate, 1,680 kJ, 265
mg Na\(^{+}\), which was completely consumed between 2.5 and 2 h before experimentation. Urinary specific gravity (USG) was measured in an initial baseline sample to verify similar hydration status in all tests.

**Experimental session.** On arrival at 0900, each participant voided her bladder before body weight was determined (± 10 g). The participant was then seated for catheter placement (22-gauge Teflon intravenous catheter) in the antecubital or other suitable forearm vein of the left arm. On withdrawal of the baseline samples (without stasis), the participant began 1 h of Low Na\(^{+}\) or High Na\(^{+}\) ingestion of 10 ml/kg body mass, given in seven equal portions, every 10 min with a 2-min walk every 20 min. Low Na\(^{+}\) consisted of only of sodium chloride (0.58 g/l), whereas High Na\(^{+}\) was composed only of sodium citrate (7.72 g/l) and sodium chloride (4.5 g/l), as developed by Greenleaf et al. (14–16) as AstroAde.

On complete ingestion of the beverage, the participant remained seated during which time skin thermistors were attached. Blood sampling was conducted 15 min after drinking, with urine sampling and core thermistor insertion immediately following (Fig. 1). Twenty minutes after drinking, each participant entered a climatically controlled chamber (32°C, 50% relative humidity, air velocity ~ 4.5 m/s) and commenced cycling on a cycling ergometer (Velotron, Racermate), fitted to her usual cycling position, without fluid or feedback cues of time or distance. The power output (watts) was set to elicit 70% of her temperate-environment V\(_{O2}\) peak. Exercise stopped when the participant could no longer maintain exercise at the given intensity or when rectal temperature reached 39.5°C. HR was recorded at 1-min intervals throughout exercise. Rectal temperature was measured with a disposable thermistor (Thermistor 400, Mallinckrodt, St. Louis, MO), which was disinfected and reused within each participant. Skin temperatures were measured with insulated thermistors (Type EU, Grant Instrument, Cambridge, UK) taped below the head of the thermistor at four sites: biceps, calf, chest, and thigh, from which mean skin temperature was calculated (41). Temperatures were recorded at 1-min intervals (model 1200, Grant Instrument). V\(_{CO2}\), V\(_{O2}\), and ventilation were measured for 2 min initially at 5-min intervals until 30 min into exercise, and thereafter they were measured at 15-min intervals. Urine was collected at baseline, 15 min after drinking, immediately before exercise, and at exhaustion. Blood samples were at baseline, 15 min after drinking, 10 min into exercise, and at exhaustion. Urine samples were taken 2 min after blood sampling, except during exercise (Fig. 1).

**Blood and urine analysis.** All blood samples (17 ml each) were separated into aliquots for analysis of hematocrit (Hct), hemoglobin concentration ([Hb]), plasma sodium concentration ([Na\(^{+}\)], plasma osmolality, plasma creatinine concentration ([Cr]), and plasma AVP concentration [AVP]. A 2-ml aliquot was analyzed immediately for [Hb] (Hemoximeter, OSM3 Radiometer, Copenhagen) and Hct in quadruplicate. Blood for Hct was drawn into capillary tubes and centrifuged for 6 min at 3,000 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%. One of the remaining three additional aliquots of blood (5 ml each) was transferred into one tube containing heparin for plasma osmolality determination. The last two aliquots were transferred into tubes containing EDTA for determination of plasma [AVP], plasma [Cr], plasma estrogen concentration ([E\(_2\)], and progesterone and progestins concentration ([P\(_4\)]. All three tubes were then centrifuged for 10 min at 6°C and 3,000 revolutions/min (model GS-15R Centrifuge, Beckman-Coulter, Fullerton, CA).

Plasma osmolality was measured using vapor point depression (Osmometer, model Vapro5520, Wescor, Logan, UT). Urine [Na\(^{+}\)] and plasma [Na\(^{+}\)] were measured using an indirect ion-specific electrode analyzer (Aeroset/c8000, Otago, Southern Community Laboratories, Dunedin, NZ) in triplicate. One-third of urine aliquots were frozen at −80°C until analysis for [Na\(^{+}\)] was conducted. USG was measured in triplicate with a hand refractometer (ATAGO, Tokyo, Japan). Because analyses for hormones and electrolytes were not performed immediately, plasma and urine were put on ice and stored at −80°C until analyses.

Plasma [AVP], [E\(_2\)], and [P\(_4\)] were analyzed at a local medical laboratory (Southern Community Laboratories) utilizing the radioimmunoassay technique. The plasma [AVP] was measured using an “in-house” antiserum with synthetic AVP (Ferring or Sigma) used for standards and preparation of \(^{125}\)I-labeled AVP using chloramine T and purification by HPLC. Samples were extracted with acetonitrile (1:2 acetonitrile-plasma), and the supernatant was dried down before the assay. A 3-day preincubation step at 4°C is followed by a further 3 days at 4°C after the addition of the \(^{125}\)I-AVP. Separation of bound and free hormone is by polyethylene glycol plus-globulin precipitation. The plasma [E\(_2\)] was measured via Sorin (sensitive) RIA kit (Sorin Biomedica Diagnostics, Saluggia, Italy), whereas plasma [P\(_4\)] was determined using the commercial conjugate-antibody method (ELISA kit, Assay Designs, Ann Arbor, MI) with sample concentrations determined against a standard curve. Intra- and interassay coefficients of variation, respectively, for the midrange standards were the following: for plasma [AVP], (23.2 pg/ml) 5.1 and 9.8%; for plasma [E\(_2\)] (115 pg/ml) 2.8 and 6.0%; and for plasma [P\(_4\)] (1.5 ng/ml) 3.6% and 4.8%.

**Calculations.** Changes in PV from baseline were estimated from changes in Hct and [Hb] using the following equation (9):

\[
\%\Delta PV = 100 \left( \frac{[Hb_0]/[Hb]}{[(1 - Hct)/(1 - Hct_0)]} - 1 \right)
\]

in which subscripts \(t\) and \(0\) denote measurements at time \(t\) and at baseline (−90 min), respectively. Hb is in grams per 100 milliliters, and Hct is a fraction. Hct was multiplied by 0.96 and then 0.91 to

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Fig. 1. Time line of experimental sessions. D, drinking; B, blood sampling; BW/U, body mass (kg) and urine sampling (volume, osmolality); S, O\(_2\) consumption and CO\(_2\) production for rating of perceived exertion sampling; EXH, exhaustion; 10Ex, 10 min of exercise.
correct for trapped plasma and the venous-to-whole blood Hct excess, respectively.

Statistical analysis. Mixed models were used to model each of the outcomes as a function of time and beverage. The repeated-measures nature of the study design was taken into account by using the covariance structures available in the SAS (version 9.1) procedure MIXED. Measurements were assumed to be more highly correlated within, as opposed to between, participants and also within, vs. between, treatments within a participant. Treatment differences in least squares means for each of the outcomes at each time point were calculated from the model. Because each time point was tested, a means model (entering time as a categorical variable in the model) was used, except when hypotheses were specifically about response slopes with respect to time. End time (exhaustion), E2, and P4 were measured only once and were tested using paired t-tests. Sample size was determined from a post hoc power calculation based on pilot work and the outcomes of our women's rest study; with an alpha level of 0.05 and a sample size of six participants per group, the beta level (power) was ≥0.80 for detecting effect sizes. Differences in least squares means for each of the outcomes and 95% confidence intervals (95% CI) were calculated from the model and were considered statistically significant at \( P < 0.05 \). Values are given as means (SD).

RESULTS

Participant compliance. To ensure participant compliance, training, diet, temperature (Nat), and pill usage (OCP) logs were collected and reviewed on the morning of the second experimental session. Additionally, hydration compliance was checked via USG. A participant with USG ≤ 1.020 was considered euhydrated. All participants completed all documentation and standardization procedures requested. Participant baseline characteristics are reported in Table 1.

Acute PV changes, plasma [Na\(^+\)], plasma osmolality, and plasma [AVP]. There was significantly greater PV expansion with the High Na\(^+\) than with the Low Na\(^+\) \([-4.4 \text{ (SD 1.1)} \times -1.9\% \text{ (SD 1.3); } P = 0.0001; 95\% \text{ CI for the difference: 5.2, 6.9}; \text{ Fig. 2}\]. This occurred 15 min postdrinking. There was no evidence of a difference between OCP and natural (Nat) in the high-hormone phase in PV change (\( \Delta \)) with either High Na\(^+\) \( [\Delta 4.0 \text{ (SD 1.2)} \times [\Delta -2.03 \text{ (SD 1.5)} \times \Delta -1.84 \text{ (SD 1.3)} \times 0.047] \). Plasma [Na\(^+\)] rose to less extent in High Na\(^+\) than in Low Na\(^+\) \( [\Delta 1.8 \text{ (SD 0.6)} \times \Delta 3.1 \text{ (SD 0.4)} \text{ mmol/l}; P = 0.03; 95\% \text{ CI for the difference: 0.16, 2.44}] \), and it rose less rapidly (slope 0.70 mmol/h; 95% CI for the difference: 1.26, 0.15; \( P = 0.01 \)). However, no significant differences were observed between OCP and Nat groups \([\Delta 1.9 \text{ (0.7)} \times \Delta 1.6 \text{ (0.8)} \text{ mmol/l for High Na\(^+\)} \times \text{ OCP vs. Nat, respectively (} P = 0.683); \Delta 3.2 \text{ (0.3)} \times \Delta 2.8 \text{ (0.6)} \text{ mmol/l for Low Na\(^+\)} \times \text{ OCP vs. Nat, respectively (} P = 0.562)]. Consistent with the plasma Na\(^+\) responses, plasma osmolality rose to less extent in High Na\(^+\) than in Low Na\(^+\) \( [\Delta 10.7 \text{ vs. } \Delta 15.5 \text{ mosmol/kgH}_2\text{O for High Na\(^+\)} \times \text{ vs. Low Na\(^+\)}, \text{ respectively; } P < 0.0001; 95\% \text{ CI for the difference: 2.9, 6.7}\text{ mosmol/kgH}_2\text{O}, \) and they responded similarly in OCP and Nat (Fig. 3A). Additionally, a positive correlation was observed between the change (from baseline) of [AVP] and plasma osmolality rise (OCP High Na\(^+\) \( r = 0.94, \) Low Na\(^+\) \( r = 0.93; \) Nat High Na\(^+\) \( r = 0.95, \) Low Na\(^+\) \( r = 0.94 \text{ Fig. 3B}\).

Plasma [AVP] was similar between beverages over the drinking period \([1.4 \text{ (SD 0.4)} \times 1.3 \text{ (SD 3.1)} \text{ pg/ml; 95\% CI for the difference: } -1.0, 0.5 \text{ pg/ml: } P = 0.575], \) but the rise across exercise was smaller in High Na\(^+\) than in Low Na\(^+\) \( [\Delta 5.2 \text{ (SD 6.2)} \text{ vs. } 7.1 \text{ (SD 5.0)} \text{ pg/ml; 95\% CI for the difference: } -3.0, -1.5 \text{ pg/ml; } P < 0.0001] \) and occurred more slowly (slope: 2.4 vs. 3.1 \text{ pg·ml}^{-1}·\text{h}^{-1}; 95\% CI: 1.2, 3.7). The OCP and Nat groups were similar with regard to plasma [AVP] responses (Fig. 4); plasma [AVP] rose more rapidly during exercise with Low Na\(^+\) than with High Na\(^+\), independently of OCP usage.

Exercise tolerance and thermoregulatory responses. Individual performance and temperature data are presented in Tables 2 and 3, respectively. Time to exhaustion was longer

Table 1. Baseline subject characteristics

<table>
<thead>
<tr>
<th>Beverage</th>
<th>Triphasic OCP (High Hormone)</th>
<th>Natural 28-Day Cycle (Late Luteal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>24.0 (5.0)</td>
<td>29.0 (6.0)</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>61.9 (5.2)</td>
<td>62.7 (4.8)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>160.6 (6.0)</td>
<td>165.2 (5.0)</td>
</tr>
<tr>
<td>V\text{O}_2 peak, ml·kg\text{}^{-1}·min\text{}^{-1}</td>
<td>53.1 (2.5)</td>
<td>51.2 (1.8)</td>
</tr>
</tbody>
</table>

Table values are means (SD). OCP, oral contraceptive pill; V\text{O}_2 peak, peak O\text{2} uptake; E2, estradiol concentration; P4, progesterone and progestins concentration; AVP, arginine vasopressin concentration; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; High Na\(^+\), high sodium; Low Na\(^+\), low Na\(^+\). *P < 0.05 between OCP and Natural.

J Appl Physiol • VOL 103 • AUGUST 2007 • www.jap.org
with the High Na⁺ beverage for all participants [mean: 98.8 (SD 25.6) vs. 78.7 (SD 24.6) min; 95% CI for the difference: 13.3, 26.8; \(P = 0.0001\)]. Time to exhaustion was similar between OCP and Nat with both High Na⁺ [100.3 (SD 24.2) vs. 96.3 (SD 25.4) min; \(P = 0.482\)] and Low Na⁺ [82.1 (SD 26.3) vs. 77.6 (SD 25.4) min; \(P = 0.587\)]. Independent of pill usage, all participants ceased exercise because of volitional exhaustion, not core temperature restrictions, in the High Na⁺ with the longer time to exhaustion. Core temperature was higher at rest in OCP than in Nat for both beverage conditions (High Na⁺: 95% CI for the difference: 0.05, 0.33, \(P = 0.05\); Low Na⁺: 95% CI for the difference: 0.10, 0.36, \(P = 0.05\)), but it rose similarly during exercise in both groups (Fig. 5). Rate of core temperature rise was greater during exercise with the Low Na⁺ [1.6°C/h (SD 0.2)] than with the High Na⁺ [1.2°C/h (SD 0.2); \(P = 0.04\)] with no significant difference between OCP and Nat. Mean skin temperature increased over time in both trials (\(P < 0.00\)), and it was similar between both beverages (\(P = 0.85\)).

Average HR was lower during exercise with High Na⁺ than with Low Na⁺ [156 (SD 18) vs. 165 (SD 16) beats/min, respectively; \(P = 0.04\), with a slower rate of rise observed during exercise with High Na⁺ than with Low Na⁺ (slope 0.39 vs. 0.45 beats·min⁻¹·h⁻¹, respectively; \(P < 0.005\)). Mean rating of perceived exertion was lower in High Na⁺ than in Low Na⁺ [16 (SD 2) vs. 18 (SD 1), respectively; \(P = 0.05\)]. There were no significant differences between OCP and Nat groups for either variable.

**Fluid balance.** Following ingestion of \(\sim 628\) ml (SD 40) of fluid before exercise in each beverage condition, \(\sim 330\) ml (SD 70) more urine was produced during the Low Na⁺ trial than during the High Na⁺ trial (\(P = 0.03\)) independent of OCP usage. Mean sweat rate as calculated from rate of body mass loss was also greater with Low Na⁺ (Table 4). Urine osmolality was higher at the end of exercise with Low Na⁺ than with High Na⁺ (95% CI for the difference \(-233.06, -15.23\) mosmol/kgH₂O; \(P = 0.03\)) at the end of exercise between trials, despite exercise being 20% longer in High Na⁺ (Fig. 6).

**DISCUSSION**

This study is novel in that it is the first to investigate the effects of an acute sodium load on exercise tolerance and physiological strain of endurance-trained female cyclists. Moreover, the high hormone phase of both oral contraceptive pill and natural menstrual cycles was examined because this is the phase of the menstrual cycle in which women are most compromised physiologically with regard to fluid balance and thermoregulatory capacity and exercise performance in the heat (29, 30, 38). In line with our laboratory’s previous findings for men running in the heat (40), sodium loading led to a modest fluid (water and sodium) retention and hypervolemia and to reduced thermoregulatory strain and greater endurance during subsequent exercise. These effects were independent of OCP usage, although women using OCPs had a slightly higher resting core temperature.

As the core body temperature rises, and increased demand for heat dissipation ensues; the PV decrease that accompanies sweat-related hypohydration may compromise work capacity...
Blood flow to the muscles must be maintained at a high level during exercise to support metabolism, but augmented blood flow to the skin is also needed to transfer heat to the body surface where it can be dissipated. Hyperthermia appears to be the critical determinant of exercise capacity in the heat with emphasis on implementing strategies to minimize the rise in core temperature during exercise (25–27). In women, reproductive hormones affect fluid balance and can affect thermoregulatory control during exercise (6, 37–39). Core temperature is elevated during the high-hormone phase of both the OCP and Nat menstrual cycles, with difference of up to 0.6°C, both at rest and during exercise in temperate environments, and this is maintained in hot environments (21, 29, 33, 37). Our data demonstrate a higher baseline core temperature in those on OCPs; however, the rate of core temperature rise was similar in both groups. This observation supports the observation of Charkoudian and colleagues (4–6, 18) that the thermogenic effect of progestins and their inhibition of cutaneous vasodilation may reset baseline core temperature upward with chronic use of oral contraceptives.

In the present study, as in the study on men (40), sodium loading was evaluated as a means of prehydration to reduce cardiac and thermoregulatory strain associated with exercise in the heat. One benefit of the sodium loading and increased fluid for redistribution is the slower rise in rectal temperature, as determined in both studies. In the men’s study, the slope of rise in the High Na \(^+\)/H11001 trial was 0.32°C/h compared with 0.37°C/h in the Low Na \(^+\)/H11001 trial; with the women, it was 0.21°C/h in the High Na \(^+\)/H11001 compared with 0.29°C/h in the Low Na \(^+\)/H11001 trials.

Increased body temperature increases sinoatrial node depolarization rate directly and indirectly (via catecholamines) and thus increases HR. In the men’s study, HR averaged 157 beats/min during exercise in High Na \(^+\)/H11001 and 161 beats/min in Low Na \(^+\)/H11001, with the average cardiovascular drift in Low Na \(^+\)/H11001 being twice that in High Na \(^+\)/H11001 (0.44 vs. 0.22 beats/min), and the time-matched final HR higher. The women demonstrated similar cardiovascular responses. The average HR during exercise in the High Na \(^+\) for the women was significantly lower (156 vs. 165 beats/min) in the Low Na \(^+\)/H11001 and they also experienced.

### Table 2. Individual performance times

<table>
<thead>
<tr>
<th>Participant</th>
<th>Watts</th>
<th>High Na (^+) End, min</th>
<th>Low Na (^+) End, min</th>
<th>(\Delta)Time, min</th>
<th>Difference, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>A*</td>
<td>150</td>
<td>117.5</td>
<td>80.3</td>
<td>+ 37.2</td>
<td>31.6</td>
</tr>
<tr>
<td>B*</td>
<td>160</td>
<td>116.7</td>
<td>110.3</td>
<td>+ 6.3</td>
<td>5.4</td>
</tr>
<tr>
<td>C*</td>
<td>155</td>
<td>97.2</td>
<td>80.0</td>
<td>+ 17.2</td>
<td>17.7</td>
</tr>
<tr>
<td>D*</td>
<td>170</td>
<td>127.1</td>
<td>100.3</td>
<td>+ 27.2</td>
<td>21.4</td>
</tr>
<tr>
<td>E*</td>
<td>145</td>
<td>58.5</td>
<td>40.2</td>
<td>+ 18.3</td>
<td>31.2</td>
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<td>F*</td>
<td>155</td>
<td>100.2</td>
<td>82.5</td>
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<td>H</td>
<td>145</td>
<td>55.5</td>
<td>46.5</td>
<td>+ 9.0</td>
<td>16.2</td>
</tr>
<tr>
<td>I</td>
<td>140</td>
<td>102.1</td>
<td>88.1</td>
<td>+ 14.0</td>
<td>13.7</td>
</tr>
<tr>
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<td>145</td>
<td>93.2</td>
<td>65.3</td>
<td>+ 27.8</td>
<td>29.9</td>
</tr>
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<td>+ 48.0</td>
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</tr>
<tr>
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<td>93.3</td>
<td>+ 15.7</td>
<td>14.4</td>
</tr>
<tr>
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<td>50.1</td>
<td>+ 17.1</td>
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<tr>
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<td>120.5</td>
<td>+ 6.4</td>
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</tr>
<tr>
<td>Mean</td>
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<td>98.8</td>
<td>78.7</td>
<td>+ 30.7</td>
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</tr>
<tr>
<td>SD</td>
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<td>5.6</td>
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</tbody>
</table>

End, time to exhaustion; Watts, watts sustained during exercise for both trials; \(\Delta\), change. *OCP participant.

### Table 3. Individual reasons for exercise termination and core temperature at Exh

<table>
<thead>
<tr>
<th>Participant</th>
<th>Watts</th>
<th>High Na (^+) End, °C</th>
<th>Low Na (^+) End, °C</th>
<th>Reason</th>
<th>High Na (^+) End, °C</th>
<th>Low Na (^+) End, °C</th>
<th>Reason</th>
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<tr>
<td>A*</td>
<td>150</td>
<td>39.10</td>
<td>38.70</td>
<td>Exh</td>
<td>39.10</td>
<td>38.70</td>
<td>Exh</td>
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<tr>
<td>B*</td>
<td>160</td>
<td>39.05</td>
<td>39.50</td>
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<td>Exh</td>
</tr>
<tr>
<td>D*</td>
<td>170</td>
<td>39.10</td>
<td>38.25</td>
<td>Exh</td>
<td>39.10</td>
<td>39.40</td>
<td>Exh</td>
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<tr>
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<td>Exh</td>
<td>39.10</td>
<td>39.40</td>
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<td>Exh</td>
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<td>39.95</td>
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</tr>
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<td>Exh</td>
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<tr>
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<td>0.40</td>
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</table>

Exh, volitional exhaustion; Rec, ethical restriction end point of 39.5°C. *OCP participant.

In the present study, as in the study on men (40), sodium loading was evaluated as a means of prehydration to reduce cardiac and thermoregulatory strain associated with exercise in the heat. One benefit of the sodium loading and increased fluid for redistribution is the slower rise in rectal temperature, as determined in both studies. In the men’s study, the slope of rise in the High Na \(^+\) trials was 0.32°C/h compared with 0.37°C/h in the Low Na \(^+\) trial; with the women, it was 0.21°C/h in the High Na \(^+\) compared with 0.29°C/h in the Low Na \(^+\) trials. Increased body temperature increases sinoatrial node depolarization rate directly and indirectly (via catecholamines) and thus increases HR. In the men’s study, HR averaged 157 beats/min during exercise in High Na \(^+\) and 161 beats/min in Low Na \(^+\), with the average cardiovascular drift in Low Na \(^+\) being twice that in High Na \(^+\)(0.44 vs. 0.22 beats/min), and the time-matched final HR higher. The women demonstrated similar cardiovascular responses. The average HR during exercise in the High Na \(^+\) for the women was significantly lower (156 vs. 165 beats/min) in the Low Na \(^+\) and they also experienced.
a higher time-matched final HR. With the Low Na+, an earlier onset of performance-inhibiting hypohydration may have contributed to the cardiovascular responses. This is likely because the increased cutaneous dilation and compliance that occur with displacement of blood to the skin for cooling results in an inability to sustain adequate cardiac output, blood pressure, and perfusion to the brain (7, 13, 19, 25, 27).

Montain and Coyle (25) demonstrated that the ingestion of fluid during exercise increases skin blood flow, and therefore thermoregulatory capacity, independent of increases in the circulating blood volume. In the present investigation, no additional fluid was consumed during the exercise trials; thus the physiological strain of exercise in the heat was not attenuated by fluid intake during the exercise trials. Subsequently, the attenuation of physiological strain cannot be attributed to a PV/blood volume expansion. The interaction between thermoregulation and osmoregulation becomes an important issue here. In the present investigation and in the study on men (40), attenuation of plasma osmolality and plasma sodium was observed. Because an increase in osmolality occurs in heat-stress conditions, osmotic inhibitory input to the thermoregulatory center decreases thermoregulatory responses as well as increases osmotic AVP secretion and thirst (7). Moreover, a rise in plasma osmolality increases inhibitory input to the thermoregulatory center, thus reducing the body’s ability to thermoregulate (10, 11, 19). In both the men’s and women’s exercise studies, a slower rate of change of plasma osmolality was observed with the High Na+ compared with the Low Na+ trials.

In the present investigation, a slower AVP response to dehydrating exercise occurred in the High Na+ trials. This attenuated response of AVP may be due to the modification of AVP sensitivity that occurs in the luteal phase, or more likely it is due to a reduced plasma osmolality across the exercise duration of High Na+. The results of the present study agree with those of previous researchers (7, 10, 11, 19, 27) in that hyperosmolality appears to be an important factor in the regulation of AVP release, observed here with the lower rate of change of plasma sodium and osmolality concomitant with the attenuation of AVP response in High Na+. A greater time to exercise termination and a reduced perceived exertion indicate an increased exercise capacity with sodium loading in women during the high-hormone phase of the menstrual cycle. It appears that the increased fluid retention decreased cardiac strain and attenuated plasma osmolality and the rate of core temperature rise when no fluid was given during exhaustive cycling exercise in warm conditions. This can be attributed to the reduction of osmotic inhibitory input to the thermoregulatory system. The high-hormone phase of the menstrual cycle, whether natural or OCP regulated, is the phase in which women are most compromised for thermoregulation and fluid balance. Although exogenous female sex hormones alter cutaneous vasodilation, increase resting core temperature, and adversely affect fluid regulatory hormones, the results of the present investigation do not indicate that chronic use of OCPs further compromises women. No other differences were observed between the Nat and the OCP groups, aside from an increased resting core temperature in the OCP group, with regard to the responses to preexercise sodium loading and the subsequent increased exercise capacity. Further research is needed to determine whether these performance enhancing effects of preexercise sodium loading still occur when additional fluid is consumed during exercise.

Fig. 6. Changes in urine osmolality between beverages. Data are means (SD) of OCP and Nat for both beverages.
to try to adjust their competition schedule to their menstrual cycle as well as use a preexercise sodium-loading protocol before competition in the heat. However, sporting events and competition occurs regardless of menstrual cycle phase. The results of the present studies indicate that cardiac strain decreases and exercise capacity increases in the luteal phase using sodium loading. It is, however, unclear whether cardiovascular and/or thermoregulatory mechanisms, or resultant mechanisms, are responsible for this. The performance enhancement following sodium loading found in this study was achieved with no further fluid ingestion during exercise. Rehydration behaviors in some real-life exercise situations are such that considerable hyponatremia still develops, even if fluid is available during exercise.

ACKNOWLEDGMENTS

The authors thank Dr. John Greenleaf and Nina Stachenfeld for essential ideas and collegiality, the participants, and Robyn Bell for technical assistance.

REFERENCES

This project was funded by the Caversham Foundation.

REFERENCES


