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# Ingestion of sodium plus water improves cardiovascular function and performance during dehydrating cycling in the heat

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We studied if salt and water ingestion alleviates the physiological strain caused by dehydrating exercise in the heat. Ten trained male cyclists ($\dot{V}O_{2max}$: 60 ± 7 mL/kg/min) completed three randomized trials in a hot-dry environment (33 °C, 30% rh, 2.5 m/s airflow). Ninety minutes before the exercise, participants ingested 10 mL of water/kg body mass either alone (CON trial) or with salt to result in concentrations of 82 or 164 mM $Na^+$ (ModNa$^+$ or HighNa$^+$ trial, respectively). Then, participants cycled at 63% of $\dot{V}O_{2max}$ for 120 min immediately followed by a time-trial. After 120 min of exercise, the reduction in plasma volume was lessened with ModNa$^+$ and HighNa$^+$ trials (−11.9 ± 2.1 and −9.8 ± 4.2%) in comparison with CON (−16.4 ± 3.2%; $P < 0.05$). However, heat accumulation or dissipation (forearm skin blood flow and sweat rate) were not improved by salt ingestion. In contrast, both salt trials maintained cardiac output (~1.3 ± 1.4 L/min; $P < 0.05$) and stroke volume (~10 ± 11 mL/beat; $P < 0.05$) above CON after 120 min of exercise. Furthermore, the salt trials equally improved time-trial performance by 7.4% above CON (~289 ± 42 vs 269 ± 50 W, respectively; $P < 0.05$). Our data suggest that pre-exercise ingestion of salt plus water maintains higher plasma volume during dehydrating exercise in the heat without thermoregulatory effects. However, it maintains cardiovascular function and improves cycling performance.

Pre-exercise increase in plasma volume (PV) (i.e., hypervolemia) has been used to investigate the role of vascular dehydration on the physiological responses to prolonged exercise in the heat. Intravenous infusion of colloid [i.e., dextran, albumin (Sawka et al., 1983; Luetkemeier & Thomas, 1994) ] or crystalloid solutions (Nose et al., 1990; Farquhar et al., 2005) have been used to acutely increase PV before prolonged exercise. It has been found that the increased blood availability with the infusion of these plasma expanders improves cardiac function lowering heart rate (HR) and maintaining cardiac output during dehydrating exercises in the heat (Fortney et al., 1983; Montain & Coyle, 1992). However, to the researchers' surprise, intravenous PV expansion prior to prolonged exercise in the heat did not prevent the elevations in core temperature in endurance trained subjects (Montain & Coyle, 1992; Watt et al., 2000). Furthermore, intravenous PV expansion by 12% did not improve cycling performance (Watt et al., 2000). In contrast to PV expansion, fluid ingestion rehydrates the vascular and other fluid spaces resulting in a lowering of core temperature and maintenance of skin blood flow (Montain & Coyle, 1992). Thus, rehydration beyond the vascular space seems to be required to fully counteract the physiological effects of dehydrating exercise in the heat.

Pre-exercise ingestion of sodium solutions is a noninvasive means of increasing PV while rehydrating fluid spaces beyond the vasculature. Two studies conducted in a thermoneutral ambient environment found that pre-exercise PV expansion induced by sodium-fluid loading (i.e., 164 mM $Na^+$) improved cycling time-trial performance after a prolonged bout of exercise (Greenleaf et al., 1997; Coles & Luetkemeier, 2005). However, those improvements were not accompanied by reduced heart rate or core temperature, which were similar to the control trial (i.e., flavored water or nothing trial). To our knowledge, there are only three studies in the literature addressing the physiological effects of pre-exercise PV expansion with oral saline solutions during exercise-heat stress. Two of these studies reported that core temperature and performance were improved during exercise after ingestion of a highly concentrated sodium solution [i.e., 164–170 mM $Na^+$ (Sims et al., 2007a,b) ]. The third study found no effects on core temperature (Nelson et al., 2008); however, performance was not assessed in this study. To assess performance, Sims and co-workers used a time to exhaustion protocol which has a higher inter-day variability than a time-trial protocol (Jeukendrup et al., 1996). Furthermore, in one of the cited studies (Sims et al., 2007b), 75% of participants did not voluntarily fatigue, but they were stopped when reached the ethically constrained core temperature limit of 39.5 °C. Thus, an absolute temperature threshold delimited performance rather than the individually perceived
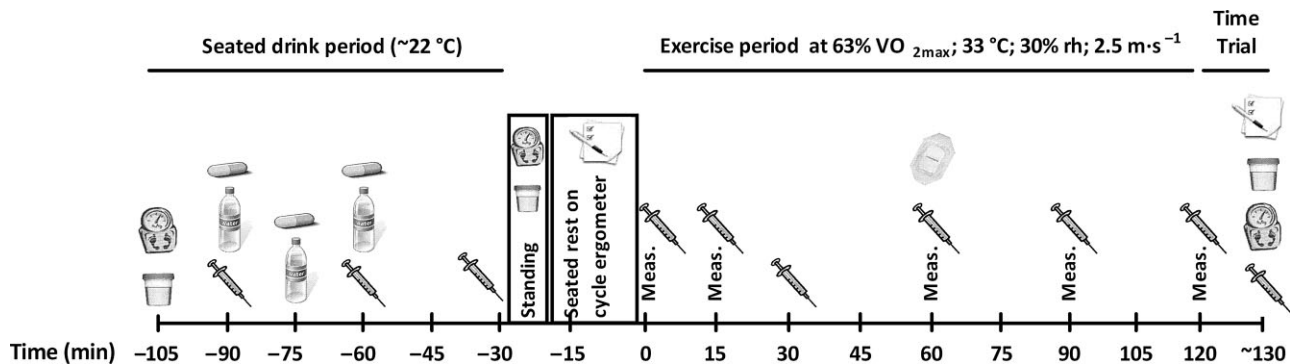
*Fig. 1.* Schematic drawing of experimental protocol. Legend: , nude body mass; , ingestion of salt capsules and water bolus; , gastrointestinal symptoms questionnaire; , sweat sampling (electrolyte concentration in back sweat sample); , urine sampling (specific gravity and electrolytes concentration in urine); , blood sampling ([Hb], hematocrit and glucose, urea, electrolytes and lactate concentration in blood serum); Meas.: intestinal temperature, mean skin temperature, skin blood flow, heart rate, cardiac output, blood pressure, rating of perceived exertion.

fatigue. Therefore, it is unclear if pre-exercise sodium-fluid loading can improve cycling performance during a time-trial in the heat.

The purpose of this study was to determine the effects of pre-exercise ingestion of sodium plus water on the thermoregulatory and cardiovascular responses during dehydrating exercise in the heat. Additionally, we sought to investigate the effects of sodium plus water ingestion on exercise performance. We hypothesized that ingestion of sodium plus water prior to dehydrating exercise in the heat will increase PV reducing the cardiovascular and thermoregulatory strain and improving cycling performance of trained cyclists. We used two concentrations of sodium with water to investigate a possible dose–response effect.

## Methods

### Participants

Ten trained male cyclists volunteered to participate in the study. Participants routinely cycled at least 2 h/day, 4–7 days/week during the last 3 years. Their mean ± standard deviation (SD) for age, height, body mass, percent body fat, maximal oxygen uptake and maximal HR were 33 ± 6 years, 1.81 ± 0.04 m, 77 ± 8 kg, 9 ± 2%, 60 ± 7 mL/kg/min and 184 ± 8 beats/min, respectively. They were non-smokers and were not taking any drugs by medical prescription during the study. All participants were fully informed of any risks and discomforts associated with the experiment before giving their informed written consent to participate. The study was approved by the local Hospital Research Ethics Committee and conducted in accordance with the guidelines of the revised Declaration of Helsinki.

### Preliminary testing and familiarization trial

Before the onset of the experiment, participants performed a continuous incremental cycling test to volitional fatigue on an electronically braked cycle ergometer (Ergoselect 200, Ergoline, Bitz, Germany) to determine their maximal aerobic power ($\dot{V}O_{2max}$). After a 5-min warm-up at 75 W, participants began cycling at 100 W with increments of 25 W/min. Gas exchange data were collected using an automated breath by breath system (Quark b$^2$,

Cosmed, Rome, Italy) and averaged every 15 s. At rest and at the end of each exercise stage, an ECG recording (Quark T12, Cosmed, Rome, Italy) and blood pressure (Gamma GST, Heine, Herrsching, Germany) were measured to discard abnormal cardiovascular responses. Maximal oxygen uptake (i.e., $\dot{V}O_{2max}$) was defined as the highest plateau (two successive maximal readings within 0.15 L/min) reached. Data resulting from this test were used to set the individual workload of each participants during the experimental trials (i.e., 63% $\dot{V}O_{2max}$). After 1 h of rest and rehydration with sports drink, participants underwent a familiarization trial consisting on 1h cycling at the experimental workload in a hot-dry environment immediately followed by a cycling time-trial test. The aims of this trial were to allow the participants to be familiarized with the laboratory testing procedures and with the time-trial protocol (Currell & Jeukendrup, 2008). The preliminary testing and familiarization trial were carried out at least a week before the onset of the experimental trials and participants refrained from hard physical activity the day prior to testing.

### Experimental design

Participants performed three double-blind, randomized exercise trials in a hot-dry environment (33 ± 0.3 °C, 30 ± 4% rh, 2.5 m/s airflow) separated at least by 5 days to avoid heat acclimatization and to ensure a proper restoration of glycogen between trials. Experimental trials were conducted in the morning to avoid an effect of circadian variation on physiological variables (Krauchi & Wirz-Justice, 1994). During the 90 min prior to the exercise, participants ingested 10 mL $H_2O$ per kg of body mass (i.e., 773 ± 77 mL) either alone (control trial; CON trial) or in combination with salt to result in a moderate (82 mM Na$^+$; ModNa$^+$ trial) or a high concentration of sodium (164 mM Na$^+$; HighNa$^+$ trial). The salt dose (i.e., 3640 ± 364 and 7280 ± 727 mg for ModNa$^+$ and HighNa$^+$ trials, respectively) was divided into six opaque gelatine capsules, each containing equal amounts of salt. In the CON trial, empty opaque capsules were provided. The capsules were ingested with water in three equal boluses at −90, −75 and −60 min prior to the onset of exercise (Fig. 1). The content of the capsules was blinded for participants and to most researchers. Then, participants cycled at 63% of their $\dot{V}O_{2max}$ (i.e., 204 ± 13 W) for 120 min with no further fluid ingestion, immediately followed by a cycling time-trial test. Participants were instructed to avoid strenuous exercise for 24 h before each trial and to refrain from all dietary sources of caffeine and alcohol for 48 h before the trials. In addition, food and training diaries were collected to aid in the replication of diet and exercise before each trial.

## Experimental protocol

The evening before each experimental trial, participants were instructed to consume the same meal and to drink 500 mL of water before they went to bed. On the day of testing, participants reported to the laboratory after 8 h of sleep and 2 h after ingesting a standardized high-carbohydrate meal (i.e., 3 g/kg of body mass consisting of orange juice, bread and peach jam) and 500 mL of water to increase the likelihood they would begin the trials in a euhydrated state. On arrival to the laboratory, participants voided before nude body mass was measured using a $\pm$ 0.05 kg sensitive scale (WildCat; Mettler, Toledo, Ohio, USA). A urine sample was collected and immediately analyzed for specific gravity ($U_{SG}$) to confirm euhydration [i.e., $\leq 1.020$; (Sawka et al., 2007) ]. Participants were catheterized (22-G Teflon®; BD Insyte, Becton Dickinson, Madrid, Spain) in an antecubital vein of the arm and seated for 20 min before a baseline blood sample was withdrawn (5 mL). Then, one third of total volume of water (i.e., 258 $\pm$ 26 mL) and two capsules with the corresponding dose of NaCl (or empty in the CON trial) were dropped in the participant's mouth by a researcher to avoid treatment identification. During the drinking period, participants remained seated and a blood sample (5 mL) was withdrawn every 30 min (Fig. 1). The water provided during the drinking period was at room temperature (i.e., ~22 °C).

After the 75 min drinking period, participants voided before nude body mass was measured again. They dressed in shorts and cleated cycling shoes, attached a HR telemetric band around their chest (WearLink®, Polar, Kempele, Finland) and two customized high-storage sweat patches were placed on their back. Then, they entered the climatic chamber, and sat quietly on the cycle ergometer for 20 min while resting body temperatures, HR, skin blood flow and arterial blood pressure were recorded. After a pre-exercise blood sample was drawn, subjects cycled for 120 min at a constant work rate immediately followed by a cycling time-trial (full description later). Rating of perceived exertion (RPE) was measured frequently during exercise [i.e., Borg scale (Borg, 1975)]. Upon completion of the performance test, participants towelled dry and their post-exercise nude body mass was measured again.

## Sweat collection and analysis

Before exercise, participant's back was cleaned with isopropyl alcohol, rinsed with distilled water and dried with a sterile gauze (Montain et al., 2007). Then, two sweat patches composed of sterilized cotton gauze (5 × 5 cm; Indas, Spain) covered with powder-free latex (5 × 7 cm; Aposan, Spain) were attached to the skin of the back using an adhesive wound dress [10 × 12 cm; Tegaderm; 3 M, St. Paul, Minnesota, USA; (Hamouti et al., 2011)]. The latex was used to increase the sweat storage capacity of the patch preventing excessive skin wetness [i.e., hidromeiosis; (Gonzalez et al., 1974)]. After 60 min of exercise, sweat patches were removed using clean tweezers and immediately placed in sealed tubes. The tubes were centrifuged (2000 × g for 10 min at 4 °C) and sweat was transferred into clean tubes (Eppendorf®). Sweat samples were analyzed for electrolytes ($[Na^+]_{sweat}$, $[K^+]_{sweat}$ and $[Cl^-]_{sweat}$) using an ion-selective analyzer (Easylyte Plus, Medica Corporation, Bedford, Massachusetts, USA). Back $[Na^+]_{sweat}$ was extrapoled to whole-body $[Na^+]_{sweat}$ using a specific site equation proposed by Patterson et al. (2000). Sweat $Na^+$ losses were calculated by multiplying the whole-body sweat loss by the whole-body $[Na^+]_{sweat}$ after 60 min of exercise assuming unchanged $[Na^+]_{sweat}$. Recently, we have found that $[Na^+]_{sweat}$ does not differ between 60 min and 90 min of dehydrating exercise in a similarly hot-dry environment (Mora-Rodriguez et al., 2008).

## Urinary analysis

Urine volume collected upon arrival to the laboratory and before and immediately after exercise was measured using a graduated cylinder (Symax, Proton, Madrid, Spain). Urine specimens were analyzed in duplicate for specific gravity ($U_{SG}$) using a handheld refractometer (Master-Sur/Nα, Atago, Tokyo, Japan). The same measurement apparatus and methods used to measure sweat electrolytes were used to measure urine electrolytes ( $[Na^+]_{urine}$, $[K^+]_{urine}$ and $[Cl^-]_{urine}$). Urine $Na^+$ losses were calculated by multiplying the urine volume by $[Na^+]_{urine}$.

## Blood analysis

Blood samples (5 mL) were taken after −90, −60, −30 and 0 min of seated rest during the drinking period and after 10, 30, 60, 90 and 120 min of exercise at a constant work rate. An additional blood sample was taken at the end of the cycling time-trial test. A portion (0.5 mL) of each venous blood sample was immediately analyzed for hemoglobin concentration in duplicate (ABL-520; Radiometer, Brønshøj, Denmark). Hematocrit was measured in triplicate by microcentrifugation (Biocen, Alresa, Madrid, Spain) and corrected for trapped plasma and venous sampling. Relative changes in PV were calculated with the equations outlined by Dill and Costill (1974). The remaining blood sample (4.5 mL) was allowed to clot into serum tubes (Z Serum Sep Clot Activator Vacuette®, Greiner Bio-One GmbH, Kremsmünster, Austria) and then spun at 2000 × g for 10 min in a refrigerated (4 °C) centrifuge (MPW-350R, Med. Instruments, Warsaw, Poland) to separate the serum portion. Blood serum samples were analyzed in duplicate for electrolytes concentration ($[Na^+]_{serum}$, $[K^+]_{serum}$ and $[Cl^-]_{serum}$) using an ion-selective analyzer (EasyLyte Plus, Medica Corporation, Bedford, Massachusetts, USA), glucose concentration using an automated analyzer (EML-105, Radiometer, Brønshøj, Denmark), urea concentration by spectrophotometry (WPA-S2000, Biochrom, Cambridge, UK) using enzymatic assays (BioSystems, Barcelona, Spain) and lactate concentration ( $[Lactate]_{serum}$) using an end-point reaction that involved lactate dehydrogenase and spectrophotometric detection of NADH (Hohorst, 1965). Serum glucose, urea, potassium and sodium concentrations were used to calculate serum osmolality using the following equation (Bhagat et al., 1984):

$$\left[\left(1.89 \times [Na^+]_{serum}\right) + \left(1.38 \times [K^+]_{serum}\right) + \left(1.03 \times [urea]_{serum}\right) \\ + \left(1.08 \times [glucose]_{serum}\right) + 7.45\right].$$

The remaining blood serum samples were stored at −80 °C for further analyses.

## Thermoregulatory measurements

At least 5 h before arriving to the laboratory (Kolka et al., 1997), participants ingested a telemetric body core temperature pill (Cor-Temp™, HQ, Inc., Palmetto, Florida, USA) to measure intestinal temperature ($T_{INT}$) during exercise. The telemetry pill correlates well with other methods of internal temperature measurement such as esophageal and rectal temperatures (O'Brien et al., 1998). Four superficial skin probes (Model 409; Yellow Springs Instruments Inc., Yellow Springs, Ohio, USA) were placed on the leg, thigh, chest, and arm. Mean skin temperature ($T_{SK}$) was calculated using the weighting formula (Ramanathan, 1964):

$$\left[T_{SK} = 0.3 \times (T_{CHEST} + T_{ARM}) + 0.2 \times (T_{THIGH} + T_{LEG})\right].$$

All probes were calibrated before the study using a water bath (Vertex, Velp, Usmate Velate, Italy) and a reference high-resolution (0.1 °C) mercury in-glass thermometer traceable to the German Bureau of Standards (Select, Proton). Forearm skin blood flow ($S_KBF$) was measured using a Laser-Doppler flowmeter (MoorLab, Moor Instruments, Devon, UK). The probe (Model VP12, Moor Instruments), housed in a 1.1-cm diameter skin heater probe (Model VHP1, Moor Instruments), was placed on the dorsum of the left forearm while the arm was relaxed and extended

on a sling at heart level. The $S_KBF$ probe was positioned in place before exercise and remained at the same location for the whole duration of the trial. Maximal $S_KBF$ was measured just before ending each exercise bout (i.e., at 115 min of exercise) by heating the skin at 45 °C (Skin heater SH02, Moor Instruments) during 1–3 min. $S_KBF$ was normalized using each subject's maximal vasodilation value for that trial. Laser-Doppler flowmeter and skin temperature probes were interfaced to a computer via multichannel A/D board (PowerLab 8SP, ADIntruments Ltd., Oxford, UK). Data were displayed and stored every 2 s throughout the trials and analyzed using associated software (LabChart 5.0, ADInstruments Ltd.). Whole-body sweat rate was calculated by subtracting pre-from post-exercise nude body mass correcting for exhalation of metabolic carbon and respiratory water losses (Mitchell et al., 1972). None of the participants urinated or ingested fluids during exercise and thus no correction was needed.

## Cardiovascular measurements

Oxygen uptake ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) were measured for 2 min using a computerized open-circuit spirometry system (Quark b$^2$, Cosmed, Rome, Italy). Cardiac output ($\dot{Q}$) was measured in duplicate using a computerized version of the $CO_2$-rebreathing technique of Collier (1956) adjusting for hemoglobin concentration (McHardy, 1967). $\dot{Q}$ was calculated using the indirect Fick equation:

$$\dot{Q} = \dot{V}CO_2/(CvCO_2 - CaCO_2),$$

where $CvCO_2$ is the concentration of $CO_2$ in mixed venous blood and $CaCO_2$ is the concentration of $CO_2$ in arterial blood. Expired air was analyzed for $O_2$ and $CO_2$ concentration as described earlier. End-tidal $PCO_2$ was determined on a breath-by-breath basis. Mixed venous $PCO_2$ was estimated from the $PCO_2$ equilibrium attained during the rebreathing procedure. The criteria for the $CO_2$ rebreathing equilibrium were: (1) equilibrium was obtained within 15 s of starting the rebreathing procedure and (2) equilibrium $PCO_2$ varied < 1 Torr for a 5-s period. HR was measured using a HR monitor (RS 400, Polar, Kempele, Finland). Stroke volume (SV) was calculated as $SV = \dot{Q} \cdot HR^{-1}$. Systolic (SBP) and diastolic blood pressure (DBP) were measured in triplicate using an automated blood pressure monitor (Tango, Suntech Med. Instruments, Raleigh, North Carolina, USA) while the left arm was relaxed and extended on a sling at heart level. The average coefficient of variation for three blood pressure measurements during cycling was 3%. Mean arterial pressure (MAP) was calculated as [ $(2 \times DBP)$ + SBP]/3. Data for these variables were collected at 0 and after 15, 60, 90 and 120 min of exercise ($\dot{Q}$ only after 15, 60 and 120 min).

## Cycling time-trial performance test

Following the 120 min at a constant work rate, participants began a cycling performance test which required the completion of an individualized set amount of work in the shortest time possible. The amount of work was individually determined for each participant (averaged total work and pedaling cadence were 171 ± 26 kJ and 90–80 rpm, respectively). This amount of work was equivalent to cycling for 10 min at 10% above their individual $\dot{V}O_2$ at the second ventilatory threshold [VT$_2$; (Below et al., 1995) ]. Subjects were instructed to choose the highest possible work rate they could sustain for 10 min and were informed of the estimated completion time if the chosen load was maintained. They could alter the work rate every minute during the test and were given an updated completion time estimate. However, subjects were not informed of total completion time until all three trials were finished. Standardized encouragement was given to the participants by the same researcher who was blind to the treatments.

## Gastrointestinal symptoms questionnaire

Participants completed a questionnaire to assess gastrointestinal symptoms (GI symptoms), which included 17 items on a 10-point scale ranging from 0, *no problem at all*, to 9, *the worst it has ever been* (Pfeiffer et al., 2009). The questionnaire was answered at the end of the drinking and exercise period. The questions were classified and analyzed in three sections as follows: section 1, addressed upper abdominal problems (reflux, heartburn, bloating, cramps, vomiting, nausea); section 2, lower abdominal problems (intestinal cramps, flatulence, urge to defecate, left abdominal pain, right abdominal pain, loose stool, diarrhea); and section 3, systemic problems (dizziness, headache, muscle cramp, urge to urinate). Upper and lower abdominal symptoms above 4 were classified as serious following the criterion used by Pfeiffer et al. (2009).

## Statistical analysis

Sample size was calculated based on a performance mean work rate difference of at least 13.5 W (i.e., 5% improvement from CON) with a standard deviation of 9 W among trials of the same individuals (pilot data). The statistical power was set at 80% ($\alpha = 0.05$) with a result of seven participants needed to be enrolled. A sample size of 10 participants allowed for a dropout rate of up to 30%. Gastrointestinal symptoms were not normally distributed and a nonparametric statistical technique was applied. Differences in gastrointestinal symptoms between treatments were analyzed as differences between mean values using Friedman's two-way rank test. Differences between trials in variables measured at a single time point were analyzed using one-way repeated measures analysis of variance (ANOVA). Data collected repeatedly over time were analyzed using two-way (time × trial) repeated measures ANOVA. After a significant $F$ ratio (Greenhouse–Geisser adjustment for sphericity), pairwise differences were identified using Tukey's (honest significant differences) post-hoc procedure. Pearson's correlation coefficient ($r$) was used to determine the relationship between variables. The significance level was set at $P < 0.05$. Data are presented as means ± standard deviation (SD). All statistics were performed using the Statistical Package for the Social Sciences (SPSS) for Windows (Version 18, SPSS Inc., Chicago, Illinois, USA).

## Results

### Fluid and sodium balance

Participants arrived at the laboratory in a similar hydration state as suggested by similar pre-trial $U_{SG}$ (1.014 ± 0.006, 1.017 ± 0.003 and 1.013 ± 0.006 for CON, ModNa$^+$ and HighNa$^+$ trial, respectively) and body mass (77.5 ± 7.4, 77.7 ± 7.8 and 77.6 ± 7.4 kg for CON, ModNa$^+$ and HighNa$^+$ trial, respectively). During the ModNa$^+$ and HighNa$^+$ trials, urine production was reduced and fluid balance improved at rest and during exercise in comparison with the CON trial (Table 1A, $P < 0.05$). Percent of dehydration was 4.4 ± 0.2, 4.1 ± 0.2 and 4.1 ± 0.2% for CON, ModNa$^+$ and HighNa$^+$ trial, respectively. Fluid balance was not further improved by augmenting the dose of sodium ingested (i.e., ModNa$^+$ vs HighNa$^+$ trials; Table 1A). As expected, both Na$^+$ trials improved total sodium balance in comparison with the CON trial (Table 1B, $P < 0.05$). Of note, pre-exercise ingestion of salt even at the high dose (i.e., HighNa$^+$) was not enough to prevent sodium deficit

Table 1. (A) Fluid and (B) sodium balance during each period and after the entire experimental session.

**A**

| Trial | Drinking period | | | Exercise period | | | | | Total fluid balance (L) |
|---|---|---|---|---|---|---|---|---|---|
| | Fluid intake (L) | Fluid losses | Fluid balance (L) | Fluid intake (L) | Fluid losses | | | Fluid balance (L) | |
| | | Urine loss (L) | | | Sweat loss (L) | Urine loss (L) | Resp/metab (L) | | |
| CON | 0.77 ± 0.08 | 0.43 ± 0.2 | 0.35 ± 0.2 | — | 3.03 ± 0.4 | 0.33 ± 0.1 | 0.42 ± 0.03 | −3.78 ± 0.4 | −3.43 ± 0.5 |
| ModNa+ | 0.77 ± 0.08 | 0.33 ± 0.2† | 0.45 ± 0.1† | — | 3.00 ± 0.4 | 0.21 ± 0.1† | 0.42 ± 0.02 | −3.63 ± 0.4† | −3.18 ± 0.5† |
| HighNa+ | 0.77 ± 0.08 | 0.33 ± 0.2† | 0.44 ± 0.1† | — | 2.96 ± 0.4 | 0.19 ± 0.1† | 0.42 ± 0.03 | −3.56 ± 0.4† | −3.12 ± 0.5† |

**B**

| Trial | Drinking period | | | Exercise period | | | | Total Na+ balance (mmol) |
|---|---|---|---|---|---|---|---|---|
| | Na+ intake (mmol) | Na+ losses | Na+ balance (mmol) | Na+ intake (mmol) | Na+ losses | | Na+ balance (mmol) | |
| | | Urine (mmol) | | | Sweat (mmol) | Urine (mmol) | | |
| CON | 0.2 ± 0.02 | 25.0 ± 8.8 | −24.8 ± 8.8 | — | 109.8 ± 44.0 | 10.2 ± 3.9 | −120.0 ± 43.8 | −144.9 ± 49.3 |
| ModNa+ | 63.4 ± 6.3† | 24.5 ± 9.6 | 38.9 ± 10.4† | — | 116.4 ± 52.4 | 13.4 ± 9.1 | −129.9 ± 52.7 | −91.0 ± 52.6† |
| HighNa+ | 127 ± 12.7†‡ | 22.0 ± 4.3 | 104.8 ± 14.7†‡ | — | 113.2 ± 50.9 | 14.1 ± 8.4 | −127.3 ± 46.1 | −22.5 ± 43.9†‡ |

Data are presented as means ± standard deviation for 10 participants. CON: plain water; ModNa+: water with moderate sodium concentration (82 mM Na+); HighNa+: water with high sodium concentration (164 mM Na+). †Different from CON ($P < 0.05$). ‡Different from ModNa+ ($P < 0.05$).
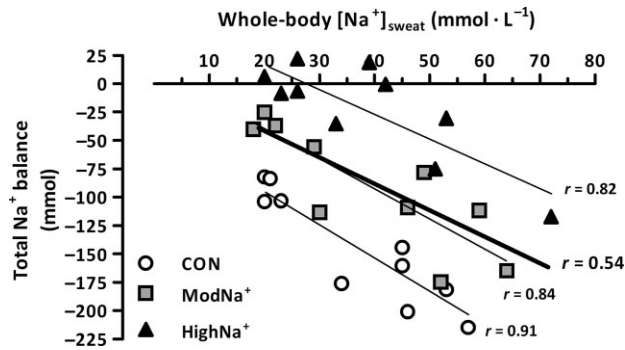
*Fig. 2.* Relationship between whole-body $[Na^+]_{sweat}$ and total $Na^+$ balance during 120 min of exercise at 63% $\dot{V}O_{2max}$ in the heat after ingestion of plain water (CON; 10 mL/kg), water with moderate sodium concentration (82 mM $Na^+$; ModNa$^+$) or water with high sodium concentration (164 mM $Na^+$; HighNa$^+$).

during 2 h of prolonged exercise in a hot-dry environment (Table 1B). However, three participants had a positive sodium balance during the HighNa$^+$ (Fig. 2), although as a group, sodium balance was negative. After 60 min of prolonged exercise, whole-body $[Na^+]_{sweat}$ was not different among trials (36 $\pm$ 15, 39 $\pm$ 17 and 38 $\pm$ 16 mmol/L for the CON, ModNa$^+$ and HighNa$^+$ trial, respectively). The mean whole-body $[Na^+]_{sweat}$ difference between ModNa$^+$ and HighNa$^+$ trials vs CON trial was 3 $\pm$ 5 and 2 $\pm$ 8 mmol/L, respectively. When the individual's whole-body $[Na^+]_{sweat}$ data were correlated with the individual sodium balance data, we obtained significant correlations for all trials ($P < 0.05$ for all trials; Fig. 2).

## Blood responses

Prior to exercise in the ModNa$^+$ trial, PV was significantly expanded above baseline (2.1 $\pm$ 2.1%; Fig. 3, $P < 0.05$) while in the HighNa$^+$ trial increased by 1.0 $\pm$ 2.3% without reaching statistical significance ($P = 0.2$). In contrast, PV decreased from baseline by $-2.5 \pm 3.1\%$ in the CON trial (Fig. 3, $P < 0.05$). As a result, PV was ~4–5% higher in the ModNa$^+$ and HighNa$^+$ trials than the CON trial before exercise (Fig. 3, $P < 0.05$). During exercise, PV was better maintained in the ModNa$^+$ and HighNa$^+$trials than in the CON trial at all time points (Fig. 3, $P < 0.05$) with no difference between the ModNa$^+$ and HighNa$^+$ trials. Pre-drinking $[Na^+]_{serum}$ was similar in all trials (average of 140 $\pm$ 1 mmol/L; Fig. 4A). However, before the onset of exercise, $[Na^+]_{serum}$ was significantly higher in the HighNa$^+$ trial than the CON and ModNa$^+$ trials (141 $\pm$ 1 vs 140 $\pm$ and 140 $\pm$ 1 mmol/L, respectively, $P < 0.05$; Fig. 4A). $[Na^+]_{serum}$ increased over time in all trials ($P < 0.05$) being higher after 120 min of exercise in the HighNa$^+$ trials than the CON trial (148 $\pm$ 2 vs 146 $\pm$ 2 mmol/L, respectively, $P < 0.05$; Fig. 4A). $[Cl^-]_{serum}$ was higher in the ModNa$^+$ and HighNa$^+$ trials than the CON trial before the onset of exercise (104 $\pm$ 2 and 104 $\pm$ 2 vs 102 $\pm$ 2 mmol/L, respectively, $P < 0.05$).
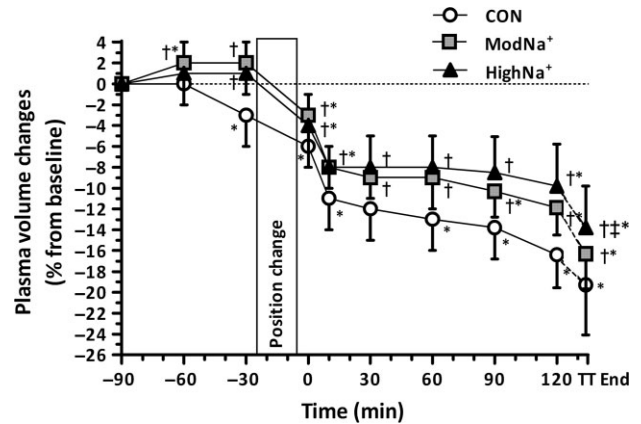


*Fig. 3.* Mean changes in plasma volume before and during 120 min of exercise at 63% $\dot{V}O_{2max}$ in the heat and at the end of the cycling time-trial after ingestion of plain water (CON; 10 mL/kg), water with moderate sodium concentration (82 mM $Na^+$; ModNa$^+$) or water with high sodium concentration (164 mM $Na^+$; HighNa$^+$). Data are presented as means $\pm$ standard deviation. *Different from previous time point within the same trial ($P < 0.05$). †Different from CON at the same time point ($P < 0.05$).

$[Cl^-]_{serum}$ increased over time in all trials ($P < 0.05$) being higher after 120 min of exercise in the HighNa$^+$ and ModNa$^+$ trials than the CON trial (111 $\pm$ 2 and 109 $\pm$ 2 vs 108 $\pm$ 2 mmol/L, respectively, $P < 0.05$). $[K^+]_{serum}$ remained unchanged during the drinking period ($\sim$4.2 $\pm$ 0.2 mmol/L) and increased similarly during exercise in all trials reaching after 120 min of exercise an average value of 5.3 $\pm$ 0.3 mmol/L. Basal serum osmolality was similar in all trials (average of 290 $\pm$ 2 mOsmol/kg H$_2$O; Fig. 4B). However, before the onset of exercise, serum osmolality was significantly higher in the HighNa$^+$ trial than the CON and ModNa$^+$ trials (291 $\pm$ 2 vs 289 $\pm$ 2 and 289 $\pm$ 2 mOsmol/kg H$_2$O, respectively; $P < 0.05$; Fig. 4B). Serum osmolality increased over time in all trials ($P < 0.05$), being higher in the ModNa$^+$ and HighNa$^+$ trials than the CON trial (303 $\pm$ 5 and 305 $\pm$ 4 vs 301 $\pm$ 4 mOsmol/kg H$_2$O, respectively; $P < 0.05$; Fig. 4B) after 120 min of exercise without difference between the ModNa$^+$ and HighNa$^+$ trials.

## Thermoregulatory responses

Prior to exercise, $T_{INT}$ was similar in all trials (averaged 37.0 $\pm$ 0.2 °C; Fig. 5A). During exercise at a constant work rate, $T_{INT}$ increased over time in all trials (Fig. 5A; $P < 0.05$) although no treatment effect was observed. However, it should be noted that, at the end of the cycling time-trial, $T_{INT}$ tended to be lower in the ModNa$^+$ and HighNa$^+$ trials than in the CON trial (0.3 °C difference, Fig. 5A; $P = 0.09$). $T_{SK}$ and forearm $S_KBF$ increased from rest to 15 min of exercise in all trials (Fig. 5B and C; $P < 0.05$) and no treatment effect was observed during exercise. Whole-body sweat rate was
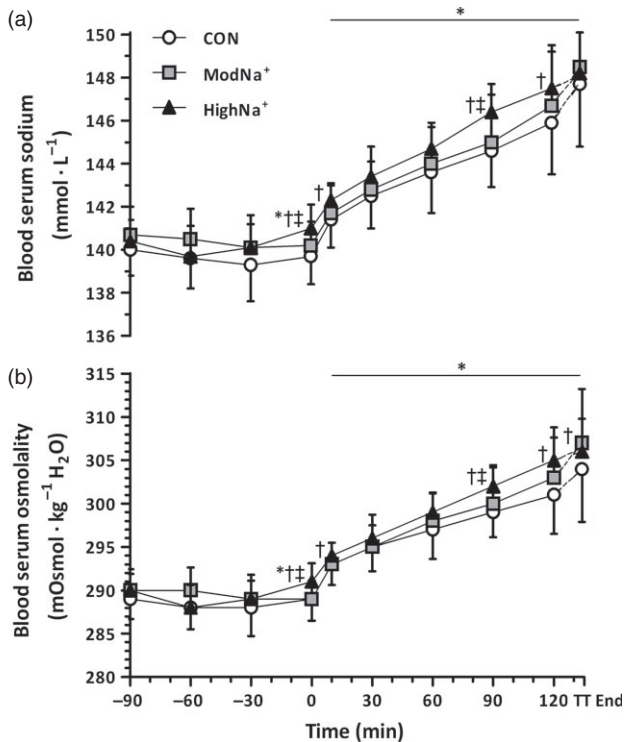
*Fig. 4.* (a) Blood serum sodium concentration and (b) serum osmolality before and during 120 min of exercise at 63% $\dot{V}O_{2max}$ in the heat and at the end of the cycling time-trial after ingestion of plain water (CON; 10 mL/kg), water with moderate sodium concentration (82 mM Na$^+$; ModNa$^+$) or water with high sodium concentration (164 mM Na$^+$; HighNa$^+$). Data are presented as means $\pm$ standard deviation. *Different from previous time point within the same trial ($P < 0.05$). †Different from CON at the same time point ($P < 0.05$). ‡Different from ModNa$^+$ at the same time point ($P < 0.05$).

not different among trials (1.4 $\pm$ 0.2 L/h in all trials). In addition, local sweat rates of the back during the first 60 min of exercise confirmed the lack of differences among trials (1.8 $\pm$ 0.8; 1.9 $\pm$ 0.8 and 1.8 $\pm$ 0.7 mg/cm$^2$/min for CON, ModNa$^+$ and HighNa$^+$ trials, respectively).

## Cardiovascular responses

HR at rest was similar in all trials (averaged 65 $\pm$ 9 beats/min) and increased similarly with exercise in all trials (Fig. 6A; $P < 0.05$). However, at the end of 120 min, HR tended to be lower in the ModNa$^+$ and HighNa$^+$ trials than the CON trial (149 $\pm$ 12 and 149 $\pm$ 14 vs 153 $\pm$ 14 beats/min, respectively, Fig. 6A; $P = 0.06$). At the end of the time-trial, HR was similar in all trials (174 $\pm$ 11, 175 $\pm$ 10 and 175 $\pm$ 10 beats/min for CON, ModNa$^+$ and HighNa$^+$ trials, respectively; Fig. 6A). $\dot{Q}$ was similar in all trials after 15 min of exercise (averaged 19.3 $\pm$ 2.0 L/min; Fig. 6B), but was better maintained in the ModNa$^+$ and HighNa$^+$ trials than the CON trial at 60 and 120 min of exercise
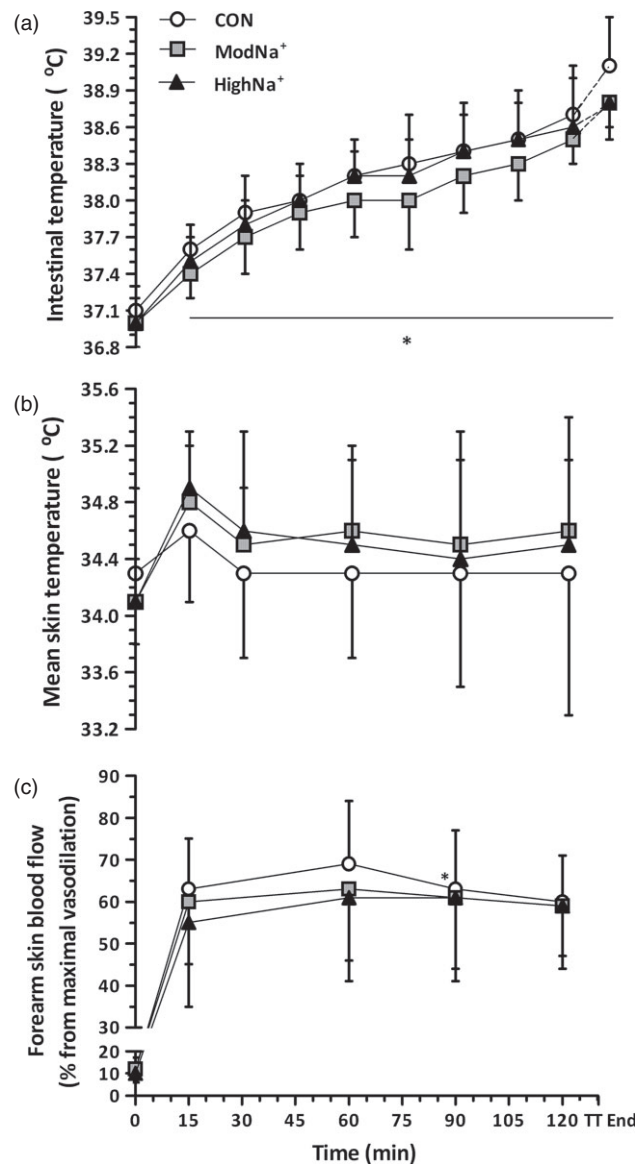


*Fig. 5.* (a) Intestinal temperature, (b) mean skin temperature and (c) forearm skin blood flow response during 120 min of exercise at 63% $\dot{V}O_{2max}$ in the heat and at the end of the cycling time-trial after ingestion of plain water (CON; 10 mL/kg), water with moderate sodium concentration (82 mM Na$^+$; ModNa$^+$) or water with high sodium concentration (164 mM Na$^+$; HighNa$^+$). Data are presented as means $\pm$ standard deviation. *Different from previous time point within the same trial ($P < 0.05$).

(1.3 $\pm$ 1.4 L/min mean difference at 120 min, Fig. 6B, $P < 0.05$). SV was similar at 15 min of exercise in all trials (averaged 154 $\pm$ 20 mL/beat; Fig. 6C), but was better maintained in the ModNa$^+$ and HighNa$^+$ than the CON trial at 60 and 120 min of exercise (10 $\pm$ 11 mL/beat mean difference, Fig. 6C, $P < 0.05$). There was no difference in $\dot{Q}$ or SV between the ModNa$^+$ and HighNa$^+$ trials. MAP was similar in all trials during 120 min of exercise although it declined from 60 to 120 min in the CON trial only (Fig. 6D; $P < 0.05$).
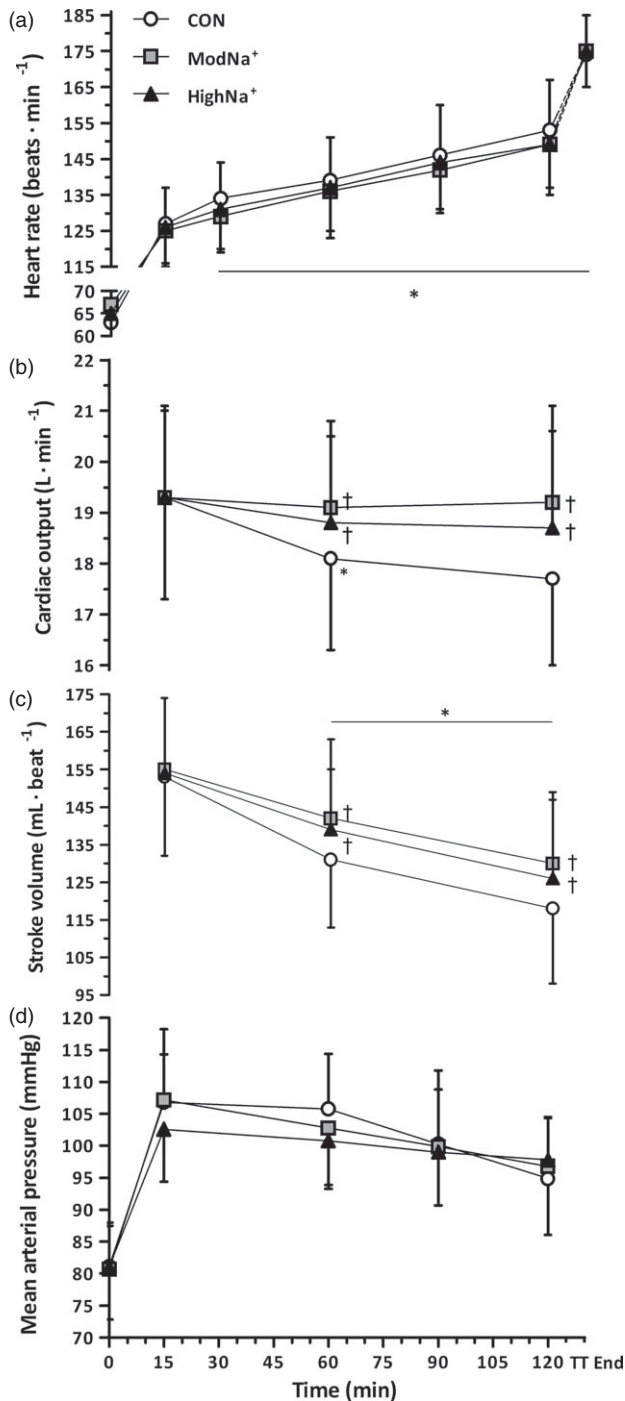
*Fig. 6.* (a) Heart rate, (b) cardiac output, (c) stroke volume and (d) mean arterial pressure response during 120 min of exercise at 63% $\dot{V}O_{2max}$ in the heat and at the end of the cycling time-trial after ingestion of plain water (CON; 10 mL/kg), water with moderate sodium concentration (82 mM $Na^+$; ModNa$^+$) or water with high sodium concentration (164 mM $Na^+$; HighNa$^+$). Data are presented as means $\pm$ standard deviation. *Different from previous time point within the same trial ($P < 0.05$). †Different from CON at the same time point ($P < 0.05$).

## RPE and metabolic responses

During exercise, $\dot{V}O_2$ and RPE increased over time in all trials (Tables 2, $P < 0.05$), although no treatment effect was observed at any time point. Of note, RPE tended to be lower in the ModNa$^+$ and HighNa$^+$ trials than the CON trial at the end of 120 min of exercise (Tables 2, $P = 0.07$). Respiratory exchange ratio (an index of substrate oxidation) was similar in all trials after 15 min of exercise and it decreased over time similarly in all trials (Tables 2, $P < 0.05$). [Lactate]$_{serum}$ remained unchanged during the 120 min of exercise in all trials (Table 2). At the end of the time-trial, [Lactate]$_{serum}$ increased above the 120 min values in all trials (Tables 2, $P < 0.05$), although no treatment effect was observed at this time.

## Cycling time-trial performance

Mean work rate was significantly higher in the ModNa$^+$ and HighNa$^+$ trials than the CON trial (i.e., 7.4% higher; 289 $\pm$ 42 and 289 $\pm$ 42 vs 269 $\pm$ 50 W respectively; Fig. 7, $P < 0.05$). There were no significant differences in performance mean work rate between the ModNa$^+$ and HighNa$^+$ trials. Completion time tended to be lower in the ModNa$^+$ and HighNa$^+$ trials than the CON trial (i.e., 4.5% lower; 597 $\pm$ 71 and 599 $\pm$ 73 s vs 625 $\pm$ 107 s, respectively; $P = 0.07$). There were no significant differences in completion time between the ModNa$^+$ and HighNa$^+$ trials.

## Gastrointestinal tolerance

No treatment effect was observed for upper abdominal, lower abdominal and systemic symptoms during the drinking and exercise period (Table 3). Only 1 of the 10 participants reported serious flatulence problems (i.e., score > 4) before and during exercise in the heat in the CON and ModNa$^+$ trials.

## Discussion

In this study, 90 min before exercise, participants ingested 10 mL of water per kg of body mass either alone (CON trial) or in combination with salt to a dose of 82 and 164 mM $Na^+$ (i.e., ModNa$^+$ and HighNa$^+$ trials, respectively). Both doses of sodium plus water augmented PV ~5% above CON during 120 min of exercise in the heat. This resulted in a better maintenance of $\dot{Q}$ and SV during prolonged dehydrating exercise (Fig. 6B and C). Furthermore, in a time-trial after 120 min of dehydrating exercise, participants increased pedaling power output 7.4% above CON during the ModNa$^+$ and HighNa$^+$ trials. We used time to completion instead of time to exhaustion for the testing of performance because it has been shown to be a more reliable test (Jeukendrup et al., 1996). Our data suggest that the improvements in cardiovascular function with salt ingestion may underlie the improved performance. However, because there was a tendency for lower RPE (Table 2) and thermoregulatory strain (0.3 °C lower $T_{INT}$; Fig. 5A) at the end of the time-trial in the salt trials, it is possible

Table 2. Rating of perceived exertion and metabolic responses during 120 min of cycling at 63% of $\dot{V}O_{2max}$ and at the end of the cycling TT in the heat.

| Variable | Trial | 15 min | 60 min | 120 min | TT End |
|---|---|---|---|---|---|
| $\dot{V}O_2$ (L/min) | CON | 2.90 ± 0.15 | 2.94 ± 0.12* | 3.04 ± 0.15* | – |
| | ModNa$^+$ | 2.90 ± 0.16 | 2.95 ± 0.17* | 3.05 ± 0.16* | – |
| | HighNa$^+$ | 2.91 ± 0.18 | 2.95 ± 0.15* | 3.05 ± 0.19* | – |
| RER | CON | 0.93 ± 0.05 | 0.90 ± 0.04* | 0.89 ± 0.04* | – |
| | ModNa$^+$ | 0.93 ± 0.05 | 0.91 ± 0.04* | 0.89 ± 0.04* | – |
| | HighNa$^+$ | 0.93 ± 0.05 | 0.92 ± 0.04* | 0.89 ± 0.04* | – |
| RPE | CON | 9.8 ± 2.0 | 12.9 ± 1.8* | 16.1 ± 1.6* | – |
| | ModNa$^+$ | 10.1 ± 1.9 | 13.0 ± 1.6* | 15.2 ± 1.6* | – |
| | HighNa$^+$ | 10.0 ± 1.8 | 12.7 ± 1.6* | 15.2 ± 1.9* | – |
| [Lactate]$_{serum}$ (mmol/L) | CON | 2.3 ± 0.9 | – | 2.4 ± 0.9 | 6.6 ± 2.3* |
| | ModNa$^+$ | 1.9 ± 0.8 | – | 2.0 ± 0.5 | 6.9 ± 2.8* |
| | HighNa$^+$ | 2.1 ± 0.8 | – | 2.2 ± 0.7 | 6.4 ± 2.1* |

Data are presented as means ± standard deviation for 10 participants. CON: plain water; ModNa$^+$: water with moderate sodium concentration (82 mM Na$^+$); HighNa$^+$: water with high sodium concentration (164 mM Na$^+$). *Different from previous time point within the same trial ($P < 0.05$). $\dot{V}O_2$, oxygen consumption; RER, respiratory exchange ratio; RPE, rating of perceived exertion; [Lactate]$_{serum}$, blood serum lactate concentration; TT, time-trial.
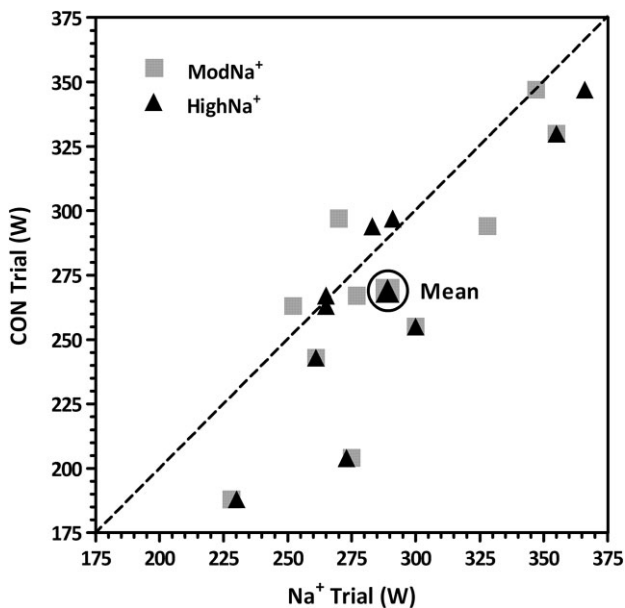


Fig. 7. Individual and mean performance data for each trial plotted against the line of identity and inset histogram of work rate (W).

that cognitive/central factors may also underlie the improved performance. In addition, the performance benefits were achieved with the moderate salt ingestion dose with which most subjects did not report any palatability or gastrointestinal discomfort.

The more applicable finding of this study is that ingestion of a moderate sodium load (82 mM Na$^+$) prior to exercise improves cycling endurance performance during dehydrating exercise in a hot-dry environment (Fig. 7). Sims et al. (2007a, b) recently showed an increase of running and cycling time to exhaustion in the heat after sodium-fluid loading similar to our HighNa$^+$ trial (i.e., 164 mM Na$^+$). They argue that these improvements may be related to a lower core temperature and perceived exertion associated with the increased PV. However, our improved performance was better associ-

ated with improvements in cardiovascular function while the correlations with core temperature and RPE were inconclusive. Our data agree with studies in a thermoneutral environment where sodium-fluid loading (i.e., 164 mM Na$^+$) also improved exercise performance mainly through effects in cardiovascular function (Greenleaf et al., 1997; Coles & Luetkemeier, 2005). Other investigators have linked the reduced cycling performance during prolonged exercise in the heat to the cardiovascular strain mediating increases in perceived exertion (Cheuvront et al., 2010).

During self-paced prolonged cycling in the heat, Periard et al. (2011) found that a thermoregulatory-mediated decrease in SV is associated with the reduction in power output and $\dot{V}O_{2peak}$. In one study, Kenefick et al. (2010) dehydrated subjects to a similar extent than in our study (i.e., 4%) prior to a time-trial performance (15 min). They observed that performance was degraded by 12% when exercising in an environment similar to ours (i.e., 30 °C). We presently find that at least 7% of the reduction in performance could be preserved despite dehydration via the expansion of PV with saline ingestion. Thus, in a situation of progressive dehydration, some of the reductions in performance could be reversed by a pre-exercise manipulation that limits the drift in SV. The link between cardiovascular strain and performance (Cheuvront et al., 2010; Kenefick et al., 2010; Periard et al., 2011) is also present in our data because alleviation of cardiovascular strain results in improved performance without necessarily affecting core temperature.

The mechanism by which improved cardiovascular function affects cycling performance is not clear. Coyle et al. (1990) reported that intravenous PV expansion (~240 mL) improved SV by 15% and also cycling performance. During the time-trial, we did not measure $\dot{V}O_2$ or SV to release subjects from breathing through a mouthpiece, which could have interfered with their performance. It is well established that muscle blood flow to the exercising muscle is reduced when individuals reach

Table 3. Reported scores for each symptom category during each period.

| Trial | Drinking period | | | Exercise period | | |
|---|---|---|---|---|---|---|
| | Upper Abd | Lower Abd | Systemic Symptom | Upper Abd | Lower Abd | Systemic Symptom |
| CON | $0.0 \pm 0.1$ | $0.2 \pm 0.6$ | $0.9 \pm 0.7$ | $0.1 \pm 0.4$ | $0.1 \pm 0.3$ | $1.3 \pm 0.8$ |
| ModNa+ | $0.8 \pm 1.4$ | $0.8 \pm 1.2$ | $1.0 \pm 0.7$ | $0.2 \pm 0.4$ | $0.3 \pm 0.6$ | $0.6 \pm 0.8$ |
| HighNa+ | $0.2 \pm 0.4$ | $0.3 \pm 0.7$ | $0.7 \pm 0.6$ | $0.1 \pm 0.2$ | $0.0 \pm 0.0$ | $0.4 \pm 0.6$ |

Data are presented as means $\pm$ standard deviation for 10 participants. CON, plain water; ModNa+, water with moderate sodium concentration (82 mM Na+); HighNa+, water with high sodium concentration (164 mM Na+).

a dehydration level of approximately 4% and that reduction limits their aerobic capacity (Gonzalez-Alonso et al., 1998, 1999; Gonzalez-Alonso & Calbet, 2003). The lowering of leg blood flow was likely an important contributor of the reduced performance in our highly oxygen-demanding time-trial. If SV had remained higher during the time-trial in the salt trials, this would have enhanced blood flow and oxygen supply to the exercising musculature, which could have allowed them to maintain higher absolute average power output. The enhanced oxygen supply may explain why blood lactate (an indirect index of anaerobic metabolism) during the time-trial was not different to CON (Table 2) despite a 7.4% higher mean work rate (Fig. 7). In summary, an improved muscle perfusion and oxygen supply may be behind the observed performance enhancement.

In contrast to the present results, other authors have reported lowering of core temperature upon pre-exercise sodium-fluid loading (164 mM Na+; Sims et al., 2007a, b). Surprisingly, Sims et al., (2007a, b) reported lowered $[Na^+]_{serum}$ when ingesting the sodium containing beverage. In contrast, we found increased $[Na^+]_{serum}$ in agreement with other authors (Coles & Luetkemeier, 2005; Nelson et al., 2008). We suspect that the difference in core temperature between Sims et al. (2007a, b) and our data may be due to the lowering of $[Na^+]_{serum}$ that they achieved. Several studies have shown an association between saline ingestion and increases in core temperature along with increases in plasma osmolality and $[Na^+]_{serum}$ (Nielsen et al., 1971; Nielsen, 1974; Harrison et al., 1978). Data from Fortney et al. (1984, 1988) using saline infusion maintaining or manipulating osmolality demonstrate the large impact of plasma osmolality in temperature regulation. Hypertonic saline solution in euhydrated humans raises core temperature (Fortney et al., 1988; Ito et al., 2005) via reduced activity of hypothalamic warm-sensitive neurons when they are expose to a hyperosmotic environment (Nakashima et al., 1985). Thus, it is possible that preventing the rise in $[Na^+]_{serum}$ and osmolality during prolonged exercise in the heat may enhance heat dissipation, explaining the lowering of core temperature in the aforementioned Sims's studies.

Although we found performance improvements when comparing the ModNa+ and HighNa+ trials against the CON trial, no differences were detected between the two doses of Na+ ingested. Performance after a bout of pro-

longed exercise in the heat was associated with PV changes, which were not different between the ModNa+ and HighNa+ trials despite participants ingesting twice more sodium (i.e., 164 mM vs 82 mM Na+ trial; Fig. 3). A similar pre-exercise PV expansion despite double Na+ dose ingested may be due to a limitation in gastric emptying and/or intestinal fluid absorption during the HighNa+ trial. However, the higher blood sodium at the onset of exercise in the HighNa+ when compared with the ModNa+ trial argues against a limitation of sodium appearance in blood. On the other hand, we used endurance-trained cyclists who are hypervolemic in essence as a consequence of endurance training adaptations (Green et al., 1991). It is then possible that the lack of PV difference between the ModNa+ and HighNa+ trials could be due to a training effect that reduces the amount of PV available to be expanded because they are close to the ceiling for expansion.

It was unclear if pre-exercise ingestion of sodium plus water could alleviate the cardiovascular drift induced by dehydrating exercise in the heat. Others have reported that HR drift is not ameliorated by pre-exercise ingestion of sodium and water despite a 3–4% expansion in PV (Sims et al., 2007b; Nelson et al., 2008). However, in those studies, cardiovascular drift was assessed based only on HR drift. We presently report that the decline in SV observed during 120 min of dehydrating exercise in the heat (i.e., 22% in the CON trial) is partially attenuated by pre-exercise ingestion of sodium plus water (Fig. 6C). Furthermore, both of our sodium ingestion doses prevented the significant reductions in Q̇ with dehydration (Fig. 6B). Thus, one of the novel findings of this study is that some aspects of cardiovascular drift (i.e., decrease in SV, but not the parallel increase in HR) are delayed by pre-exercise salt and water ingestion already with a dose of 82 mM Na+.

We did not find a statistically lower HR during the ModNa+ and HighNa+ trials despite the higher SV when compared with the CON trial (Fig. 6A). However, HR tended to be lower in the sodium ingestion trials being 4 beats/min lower at 120 min of exercise ($P = 0.06$). Presently, the better maintenance of exercise SV after preexercise ingestion of sodium plus water seems to occur from a better preservation of heart preload (i.e., diastolic filling pressure) via higher availability of blood volume (Fig. 3) in comparison with the CON trial. It is less likely

that Na$^+$ ingestion could have increased heart contractility. In addition, the similar systolic blood pressure suggests that afterload was not modified by Na$^+$ ingestion. It is unclear why the higher preload during the salt trials did not result in a reduced HR in comparison with the CON trial. However, it should be reminded that there was a 3% reduction in HR, which was close to statistically significant.

In an effort to make the treatments as practical as possible, we provided capsules filled with salt that were ingested with water instead of ingesting a saline solution to avoid the strong tasting of saline. Authors using saline have disguised the strong saline flavor by cooling the drinks to 4 °C (Sims et al., 2007a, b) or by adding sweeteners (Coles & Luetkemeier, 2005; Nelson et al., 2008). Our capsule method was successful because PV was increased from baseline in the ModNa$^+$ and HighNa$^+$ trials, respectively (Fig. 3) without reported palatability complains or gastrointestinal disturbances (Table 3). In addition, this method of ingesting salt allowed us to keep treatments blind to the participants increasing the confidence in our performance results. Despite ingestion of 773 mL of water in the CON trial, PV decreased before exercise below baseline (i.e., −2.5%; Fig. 3), effect that has been previously reported (Coles & Luetkemeier, 2005; Nelson et al., 2008). The increased urine production during the CON trial in comparison with the salt trials may partly explain this finding (Table 1A). In addition, PV declined similarly in all three trials just before the beginning of exercise (Fig. 3), which has also been reported in other studies (Greenleaf et al., 1997, 1998).

This rapid decrement in PV was likely the result of the position change that took place 20 min before the start of exercise when participants moved into the exercising hot chamber (Fig. 3).

## Perspectives

We provided capsules filled with salt that were ingested with water prior to exercise, which maintained, during subsequent prolonged exercise in the heat, higher PV than when ingesting water only. Our main finding is that cycling performance was improved despite subjects being dehydrated (i.e., ~4% dehydration) via the expansion of PV with salt ingestion. The PV expansion was associated with a better maintenance of exercise cardiovascular responses (i.e., reducing the SV drift). In addition, all the benefits were obtained with a dose of 82 mM Na$^+$ with no further benefits when doubling the dose (164 mM Na$^+$ trial). In summary, salt and water ingestion (82 mM Na$^+$) prior to exercise improves cardiovascular function, which seems to improve the capacity of endurance-trained cyclists to sustain high workloads during the latter stages of prolonged exercise in the heat.

**Key words:** plasma volume expansion, thermoregulation, cardiovascular drift, fluid-electrolyte balance.

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