Intravenous vs. oral rehydration: effects on subsequent exercise-heat stress

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This study compared the influence of intravenous vs. oral rehydration after exercise-induced dehydration during a subsequent 90-min exercise bout. It was hypothesized that cardiovascular, thermoregulatory, and hormonal variables would be the same between intravenous and oral rehydration because of similar restoration of plasma volume (PV) and osmotic pressure (Osmo). Eight non-heat-acclimated men received three experimental treatments (counterbalanced design) immediately after exercise-induced dehydration (33°C) to 4% body weight loss. Treatments were intravenous 0.45% NaCl (iv; 25 ml/kg), no fluid (NF), and oral saline (Oral; 25 ml/kg). After rehydration and rest (2 h total), subjects walked at 50% maximal O2 consumption for up to 90 min at 36°C. The following observations were made: 1) heart rate was higher (P < 0.05) in Oral vs. iv at minutes 45, 60, and 75 of exercise; 2) rectal temperature, sweat rate, percent change in PV, and change in plasma Osmo were similar between iv and Oral; 3) change in plasma norepinephrine decreased less (P < 0.05) in Oral compared with iv at minute 45; 4) changes in plasma adrenocorticototropic hormone and cortisol were different between iv and Oral after exercise was initiated; and 5) exercise time was similar between iv (77.4 ± 5.4 min) and Oral (84.2 ± 23 min). These data suggest that after exercise-induced dehydration, iv and Oral were equally effective as rehydration treatments. Thermoregulation, change in adrenocorticotropic hormone, and change in cortisol were not different between iv and Oral after exercise began; this is likely due to similar percent change in PV and change in Osmo.

Core temperature; sweat; norepinephrine; cortisol

PRESENTLY, FLUID REPLACEMENT after dehydration via intravenous infusion is based on clinical manifestations of symptoms and/or the perception that intravenous fluids restore lost body water more effectively than does oral ingestion. However, whether the direct administration (intravenous) of fluid to the intravascular space at rest, after dehydration, helps offset the deleterious effects of hypohydration better than oral ingestion during a subsequent exercise bout is not known. For individuals who often engage in two or more prolonged exercise-heat stress bouts in 1 day (athletes, soldiers, industrial laborers), it is important to identify the most efficacious means of rehydration between exercise sessions. If exercise is initiated in a hypohydrated state or plasma volume (PV) is not adequately restored, it could lead to higher heart rates (HRs), higher core temperature (Tco) values, and decreases in aerobic exercise time (33).

Only one study (18) has compared intravenous infusion with oral ingestion, with use of fluids replaced during exercise. It was found that HR was lower in intravenous infusion vs. oral ingestion after subjects cycled for 2 h. However, direct comparison between those two treatments was difficult because glucose (18%) was present in the intravenous trial only and there were large differences (1,000 ml) in the volume of fluid given between treatments. Other studies of intravenous saline infusion have compared it with no fluid ingestion, only during exercise (8, 28). Oral studies have demonstrated that 2 h of drinking (replacement of 60–65% lost fluids), after thermal or exercise-induced dehydration, fully restores PV (29), improves PV by 5–6.5% (7, 22), and normalizes exercising HR to predehydration values (7). Data are not available that directly address whether exercise performance, 1–2 h postrehydration, is similar between intravenous and oral fluid replacement strategies.

Therefore, the purpose of this study was to answer the question, After exercise-induced dehydration, does rehydration with intravenous infusion, vs. oral ingestion, differentially affect cardiovascular, thermoregulatory, and stress hormone variables during subsequent exercise? Intravenous 0.45% NaCl was chosen as the rehydration fluid because this is commonly administered to dehydrated individuals. A similar type and volume of fluid was given orally. Because the period of rehydration (replacement of 62% of lost fluids) plus rest between exercise bouts was 2 h, it was hypothesized that PV restoration and change (Δ) in osmolality (Osmo) would be similar between treatments because of adequate gastric emptying and intestinal absorption, leading to no differences in any of the measured physiological variables during subsequent exercise.

METHODS

Subjects. Eight men, unacclimatized to heat, participated in this study. Physical characteristics were age, 22.1 ± 0.8 (SE) yr; height, 179.6 ± 15.3 cm; mass, 73.6 ± 2.4 kg; maximal oxygen uptake (Vo2max), 57.9 ± 1.6 ml·kg⁻¹·min⁻¹; and percent body fat, 7.7 ± 0.9%. Subjects signed informed consent statements after attending a briefing meeting. All subjects completed a medical history questionnaire. All procedures were approved by the Institutional Review Board at the University of Connecticut.

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University of Connecticut. Subjects were paid for their participation.

Preliminary testing. Body density was determined from underwater weighing with percent body fat calculated according to Siri (34). All subjects completed an incremental run to exhaustion (6) on a motorized treadmill for determination of VO2max. Briefly, subjects ran at a constant pace (160–220 m/min) for 4 min at a 0% grade. After 4 min, the grade was increased to 4% for 2 min. The grade was then increased 2% every 2 min until the subject reached exhaustion.

Experimental protocol [dehydration (Dh), rehydration, and subsequent exercise]. Food diaries were kept for 3 days before each experimental treatment and analyzed for sodium and carbohydrate intake (Food Processor II, ESHA Research, Salem, OR). Subjects, 12 h postprandial, arrived at the laboratory between 0700 and 0745 h in a euhydrated state. To ensure euhydration, subjects were required to drink 450 ml water before going to bed the night before and 450 ml water in the morning on waking before reporting to the laboratory for testing.

After reporting to the laboratory and after placement of a rectal thermistor and venous cannula, subjects entered the environmental chamber (33°C). After a 20-min standing equilibration, a blood sample was taken, followed by ingestion of a standardized breakfast consisting of one bagel, one banana, and 240–350 ml of fruit juice. The amount of fruit juice was consistent for each individual subject during all three experimental treatments. A predehydration (Pre-Dh) body weight (±50 g; model 700M, SR Instruments, Tona-wanda, NY) was taken, and then subjects dehydrated to ~4% of their Pre-Dh body weight.

The Dh protocol consisted of alternating cycle ergometry (model 818E, Monark) and treadmill walking (Quinton, Seattle, WA) in intervals of 25 min of exercise and 5 min of rest. Body weight was measured during each rest break. Urine was collected throughout Dh and was included as part of the weight loss. Subjects continued exercising until the desired weight loss was achieved. The last exercise mode before the ~4% weight loss was always walking to ensure an upright posture. The mean percent Vo2max during Dh for the three treatments was 50.8 ± 0.2%. The mean temperature and percent relative humidity were 33.0 ± 0.1°C and 47.6 ± 0.5%, respectively. Airflow was 2.3 m/s. HR and rectal temperature (T rect) were measured during Dh to ensure the subject's safety.

After Dh, subjects were removed from the chamber and reclined in a semirecumbent position at 25.5 ± 0.2°C. After 15 min, subjects received one of three rehydration treatments (described in Experimental treatments) over a 45-min period. Each subject received all three treatments (separated by a minimum of 14 days), and the order of treatments was randomized. After rehydration, subjects stood for 55 min in the laboratory and then reentered the environmental chamber.

After the subject stood in the environmental chamber for 20 min, a blood sample was obtained. The subject then sat down and consumed 1 g carbohydrate/kg Pre-Dh body wt of a commercial snack product (Skittles, M and M Mars, Hackettstown, NJ) and 100 ml of distilled, deionized water. Carbohydrates were given before the second exercise bout to offset the possible loss of muscle glycogen during Dh. Subjects voided their bladders, were weighed (preexercise, minute 0), and then began walking at ~50% Vo2max. Walking speed was verified for each test with a hand-held tachometer (model 8204-20, Cole-Parmer Instrument, Chicago, IL). The duration of exercise was intended to be 90 min. The mean temperature and percent relative humidity were 35.9 ± 0.1°C and 46.6 ± 2.1%, respectively, for the three treatments. The temperature during exercise was set higher than during the morning Dh period to simulate an increase in dry-bulb temperature during the course of a day. Airflow was 2.3 m/s. No fluids were consumed during the exercise period. Exercise was terminated if 1) T rect reached 39.5°C, 2) HR exceeded 180 beats/min for 5 consecutive min, 3) subjects showed signs of heat illness, 4) subjects asked to stop, or 5) subjects completed 90 min of exercise.

Experimental treatments. All treatments were given over a 45 min period. They were 1) no rehydration fluid (NF), 2) intravenous saline (iv; 0.45% NaCl, 25°C) infusion, and 3) oral saline (4°C) drinking (Oral). The iv infusion was administered in the arm opposite to the indwelling venous cannula via 21-gauge butterfly cannula. The rate of iv infusion was 0.56 ml·kg⁻¹·min⁻¹. Oral consisted of 4 g of a commercial sugar-free flavored beverage (Kool-Aid) dissolved in 889 ml of 0.45% NaCl and 111 ml of distilled, deionized water. The composition of Oral was 78.6 ± 0.1 meq/l Na⁺, 0.96 ± 0.02 meq/l K⁺, and 2.54 ± 0.07 meq/l Ca²⁺. The osmolality was 145.6 ± 1.1 mosmol/kg. Oral was chilled (4°C) for palatability. Fluid intake (iv and drinking) approximated 25 ml/kg Pre-Dh body wt. This volume has been shown to be at the upper range for orally ingested fluids after exercise-induced Dh (28). The iv fluid intake was measured by weighing saline bags pre- and postinfusion on a scale (model GT8000, O‘Haus, Florham Park, NJ). Oral was weighed before each drink, which was given every 5 min over a 45-min period. The iv treatment was administered by a nurse experienced in cannula placement and intravenous infusion.

Measurements. HR was obtained at 5-min intervals during exercise by a lead 1 configuration by using a HR monitor (Polar Electro). T rect (°C) was measured every 5 min during exercise by using a thermistor (model 401, Yellow Springs Instruments, Yellow Springs, OH) inserted 10 cm past the anal sphincter. Skin temperature (T sk) values (°C) were measured at 10-min intervals during exercise by using thermocouples (model 409, Yellow Springs Instruments) secured on the chest, arm, thigh, and calf. Mean T sk was calculated from these four sites according to Ramanathan (30). Local chest sweat rate (SR ch; mg·min⁻¹·cm⁻²) was measured, via resistance hygrometry, at minutes 0 and 10 by using a commercial dew-point sensor (model B1-102, Bi-Tronics, Guildford, CT). The capsule (12 cm²) was placed on the chest 5 cm medial to the left nipple. Whole body sweat rate (I·min⁻¹·m⁻²) was determined from pre- and postexercise body weights corrected for respiratory losses and blood draw volume. Oxygen uptake (VO₂; ml·kg⁻¹·min⁻¹) was measured by using open-circuit spirometry (model CPX-D, Medical Graphics, St. Paul, MN) during exercise at minutes 20, 40, 60, and 80.

Blood was taken at Pre-Dh, postdehydration (Post-Dh), preexercise (minute 0), and at 15 and 45 min of exercise from a dwelling Teflon cannula (20 gauge) placed in a superficial forearm vein. The arm was pendant during blood draws. Before each blood sample, subjects were in a upright posture for 20 min.

Hematocrit (Hct) was determined in triplicate from whole blood by the microcapillary technique and corrected for trapped plasma (15). Hemoglobin (Hb) was measured in triplicate by the cyanmethemoglobin method (Kit 525, Sigma Chemical, St. Louis, MO). Percent change in PV (%ΔPV) was calculated by using the equation of Dill and Costill (10) from appropriate Hct and Hb values. %ΔPV values were calculated by using Post-Dh for initial Hb and Hct values. Post-Dh was used as the initial point to reflect the effect of the various rehydration treatments after Dh. Plasma Osmo was mea-
sured in triplicate by freezing-point depression (model 5004 m-osmlette, Precision Systems, Natick, MA). Plasma glucose was determined in triplicate via an enzymatic technique (model 2003 glucose/actate analyzer, Yellow Springs Instruments). Plasma norepinephrine (NE) and epinephrine (Epi) were determined in triplicate via high-performance liquid chromatography with electrochemical detection (model 460, Waters, Milford, MA; Ref. 20). Plasma adrenocorticotrophic hormone (ACTH) and cortisol (Cort) were measured in duplicate by solid-phase radioimmunoassay kits (Coat-a-Count, Diagnostic Products, Los Angeles, CA). Plasma ACTH and Cort samples were frozen at –80°C before analysis. All samples for a given subject were analyzed in the same sample run to eliminate interassay variation. Intra-assay coefficient of variations were 7.0 ± 0.15 liters (urine volume was 0.34 ± 0.15 liters for plasma NE, Epi, Cort, and Tre (Table 1), which were no significant differences were found among treatments defined as euhydrated before each treatment. Also, similar among treatments (Table 1). Thus subjects were defined as euhydrated before each treatment. Also no significant differences were found among treatments for plasma NE, Epi, Cort, and T-re (Table 1), which were within normal resting values. However, Pre-Dh plasma ACTH was different among treatments. Dh. The time to dehydrate was 190.0 ± 11.5, 180.0 ± 10.2, and 181.9 ± 7.9 min (P > 0.05); percent weight loss was 4.1 ± 0.1, 4.1 ± 0.1, and 4.2 ± 0.1% (P > 0.05); urine volume was 0.34 ± 0.12, 0.48 ± 0.09, and 0.45 ± 0.15 liters (P > 0.05); and percent VO2max was 51.0 ± 2.0, 51.1 ± 1.8, and 50.4 ± 1.9% (P > 0.05) for iv, NF, and Oral, respectively. After Dh (Table 1), there were significant (P < 0.05) differences among treatments in plasma NE, Epi, and ACTH. Also, T-re (iv vs. Oral) approached significance (P = 0.05).

Rehydration. The volume of intravenous or ingested fluid given during the rehydration period was 1,890 ± 45, 0.15 ± 0.0, and 1,856 ± 65 ml for iv, NF, and Oral, respectively (P > 0.05, iv vs. Oral). Urine volumes were 60 ± 20 ml, 80 ± 20 ml, and 80 ± 20 ml for iv, NF, and Oral, respectively (P > 0.05).

Table 1. Plasma osmolality, hormone concentrations, and rectal temperature before and after dehydration protocol

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<tr>
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<th>Pre-Dh</th>
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<td>NF</td>
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<tr>
<td></td>
<td>iv</td>
<td>NF</td>
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<tr>
<td>Plasma osmolality, m-osm/kg</td>
<td>282.0 ± 0.9</td>
<td>286.2 ± 0.7</td>
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<tr>
<td>Plasma NE, nmol/l</td>
<td>0.99 ± 0.12</td>
<td>0.77 ± 0.15</td>
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<tr>
<td>Plasma Epi, pmol/l</td>
<td>151.2 ± 29.5</td>
<td>125.5 ± 28.9</td>
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<tr>
<td>Plasma ACTH, pmol/l</td>
<td>19.5 ± 2.7</td>
<td>37.3 ± 2.9*</td>
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<tr>
<td>Plasma Cort, nmol/l</td>
<td>380.7 ± 44.1</td>
<td>444.2 ± 30.3</td>
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<tr>
<td>Rectal temperature, °C</td>
<td>37.10 ± 0.15</td>
<td>37.05 ± 0.08</td>
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Values are means ± SE; n, no. of subjects. Values are not corrected for percent change in plasma volume. Pre-Dh, predehydration; Post-Dh, postdehydration; iv, 0.45% intravenous saline; NF, no fluid; Oral, oral saline; NE, norepinephrine; Epi, epinephrine; ACTH, adrenocorticotropic hormone; Cort, cortisol.*Significantly different from iv and Oral, P < 0.05. †Significantly different from iv, P < 0.05. ‡P = 0.05 vs. iv and NF.
PV and Osmo after rehydration and during exercise. PV significantly increased (P < 0.05) by 7–8% after rehydration for iv and Oral and remained at this increased level throughout exercise (Fig. 3). There were no differences in the %ΔPV between iv and Oral. PV did not change after rehydration or during exercise in NF and was lower (P < 0.05) than in iv and Oral. ΔOsmo was significantly different (P < 0.05) in NF compared with iv and Oral after rehydration and during exercise (Fig. 3) with no differences between iv and Oral.

Plasma NE and Epi after rehydration and during exercise. ΔNE was similar among treatment groups at minute 0 (Fig. 4). ΔNE was different during NF (P < 0.05) at minutes 15 and 45 compared with iv and Oral. Also, at minute 45, ΔNE was significantly (P < 0.05) different in Oral compared with iv. No differences in ΔEpi were found among NF, iv, and Oral (Fig. 4).

Plasma ACTH and Cort after rehydration and during exercise. ΔACTH and ΔCort were different after rehydration (minute 0) in iv compared with Oral; i.e., change in plasma levels were less in iv after rehydration (Fig. 5). Once exercise was initiated (minutes 15 and 45), ΔACTH and ΔCort, from Post-Dh, were similar between iv and Oral. For NF, the change in plasma ACTH paralleled that in Oral at minute 0; however, it did not decrease once exercise began. Unlike Oral, ΔCort for NF increased after rehydration and remained elevated at minutes 15 and 45.

Plasma glucose after rehydration and during exercise. Plasma glucose concentrations were similar among treatments at Post-Dh (5.51 ± 0.23, 5.27 ± 0.11, 5.17 ± 0.11 mmol/l), minute 0 (5.03 ± 0.13, 5.21 ± 0.13, 5.11 ± 0.24 mmol/l), minute 15 (6.63 ± 0.27, 7.08 ± 0.44, 6.73 ± 0.56 mmol/l), and minute 45 (5.96 ± 0.50, 7.18 ± 0.26, 5.66 ± 0.29 mmol/l) for iv, NF, and Oral, respectively.

**DISCUSSION**

This study investigated whether intravenous or oral rehydration during rest, after exercise-induced dehydration, results in similar cardiovascular, thermoregulatory, and stress hormone responses during a subsequent exercise bout in a hot environment (36°C). No previous studies have directly examined this. Several studies (1, 35) have addressed the effect of oral rehydration, after dehydration, on a subsequent bout of exercise. They used long dehydration (days) and variable rehydration (1- to 5-h) periods, with fluid ingestion either ad libitum or fixed. Those studies, however, did not address the challenge of rehydrating between exercise bouts on the same day. Recently, Melin et al. (23)
had individuals exercise (35°C) after thermal dehydration (~2.6% body weight loss over 2–3 h) and 1-h rest. Rehydration was given orally by administration of water just before (913 ml bolus) or during the exercise period. Hamilton et al. (18) infused an 18% glucose in water solution (1,224 ml) for 100 min during exercise (70% VO$_2$max) at 22°C. They found that infusion prevented the increases in HR that were seen with water ingestion alone. However, comparisons of infusion vs. ingestion are confounded by glucose content and volume given. As in the oral rehydration studies cited above, the design of this intravenous study does not allow speculation on the effect of resting intravenous infusion, after dehydration, on a subsequent exercise trial.

The principal finding of this investigation was the higher HR during exercise after oral rehydration compared with rehydrating with a similar type and amount of iv saline. In fact, the HR response was similar between Oral and NF at minute 45. Therefore, the question is, Why does rehydrating orally result in a higher cardiac frequency compared with iv rehydration? Several mechanisms have been suggested to explain differences in HR both in and out of a hot environment. These include reduced muscle glycogen stores (19), lower blood volume (13, 26), and higher T$_{co}$ and T$_{sk}$ (31). Reduced muscle glycogen stores in oral rehydration are unlikely because carbohydrate intake before the study was equal and the time to dehydrate

Fig. 3. Percent change in plasma volume (A) and change in plasma osmolality (B) as a function of time after rehydration and during exercise with iv (▲), NF (○), and Oral (△). Values are means ± SE. Post-Dh is considered reference point. *NF significantly different from iv and Oral, P < 0.05.

Fig. 4. Change in plasma norepinephrine (A) and epinephrine (B) as a function of time after rehydration and during exercise with iv (▲), NF (○), and Oral (△). Values are means ± SE. Post-Dh is considered reference point. *NF significantly different from iv and Oral, P < 0.05. †Oral significantly different from iv, P < 0.05.
was similar between treatments. Hypovolemia is associated with decreased PV and lower stroke volumes, leading to compensatory increases in HR to maintain cardiac output. However, this study found no differences in PV between iv and Oral. Higher $T_{cv}$ and $T_{sk}$ result in higher HR due to increases in cutaneous blood flow and pooling, leading to decreased central venous pressure and stroke volume (31), but no differences were found for $T_{re}$ or $T_{sk}$ between iv and Oral. Thus it is likely that the greater cardiovascular drift in Oral occurred independently of reductions in blood volume (25) or differences in body temperatures.

The $\Delta$ plasma NE concentration in Oral, vs. iv, suggests that greater sympathetic nervous activity (SNA) contributed to the higher HR response in Oral. It is well known that HR is elevated by an increase in SNA (5). Plasma NE is a valid measure of SNA because the rate of NE spillover into the veins is proportional to the rate of sympathetic nerve firing (11). Hoffman et al. (21) have observed an increase in SNA after rats drank. However, this effect is transitory; SNA decreases soon after drinking is stopped. Therefore, the higher SNA during exercise seen in the present investigation with oral rehydration is likely not due to the act of drinking itself, because 75 min elapsed after drinking before the onset of exercise. It has been suggested that gastrointestinal distension (2) elevates SNA. Data from Berne et al. (3) do not support this hypothesis, but in their study only 300 ml of water were given; in the present investigation 1,900 ml were ingested. Therefore, the possibility exists that some fluid remained in the intestine after the oral rehydration treatment and triggered a greater sympathetic nervous response. However, if this hypothesis is correct, then it would be expected that the $\Delta$NE in Oral would have been different at minute 15 also when more fluid was in the gut, compared with minute 45. Berne et al. also demonstrated that higher plasma glucose evoked greater muscle SNA after glucose ingestion. They suggest that higher glucose increases splanchnic vasodilation, which, in turn, activates the sympathetic nervous system. However, glucose was similar between iv and Oral at minutes 15 and 45, which suggests no role for plasma glucose in increasing SNA in Oral.

This investigation found no differences in the changes in $T_{re}$, $T_{sk}$, or sweat rate between iv and Oral. These data suggest, from a thermoregulatory point of view, that it is not important how fluid is replaced between two prolonged exercise-heat stress periods. Recently, Takamata et al. (37) found that oral ingestion transiently increased skin blood flow and $SR_{ch}$ via an oropharyngeal reflex. This was associated with a decrease in esophageal temperature ($T_{es}$) and a threshold shift to the left in the $SR_{ch}$-$T_{es}$ relationship in heated, cell-dehydrated humans. The similar rate of fall in $T_{re}$ after rehydration in iv and Oral suggests that intravenous infusion may also transiently affect the thermoregulatory center. Further studies are warranted to determine whether intravenous infusion influences sweating and the $SR_{ch}$-$T_{es}$ relationship to a similar degree as did oral ingestion in cell-dehydrated, heated humans (37). Similar $SR_{ch}$ and higher $T_{re}$ values in NF, compared with iv and Oral, suggest a change in sweating threshold (27) likely mediated by the combination of higher plasma Osmo and lower PV (14, 25, 27, 32) in NF.

Plasma ACTH and Cort did not fall in Oral as significantly as iv after rehydration (minute 0). One hypothesis for the smaller $\Delta$ACTH and $\Delta$Cort from Post-Dh in Oral at minute 0 is the stress associated with ingesting a large volume of cold (4°C), salty (78 meq/l Na+) fluid. Stress is associated with activation of the hypothalamic-pituitary-adrenal axis (38). However, the large fall in plasma ACTH after the beginning of exercise (from minutes 0–15) may be due to the withdrawal of the stress induced by fluid ingestion. ACTH is now likely controlled by other physiological factors.
such as %ΔPV, ΔOsmo, or glucose. Because no differences existed between iv and Oral for these variables, similar responses were observed in ΔACTH and ΔCort after exercise was initiated. Results from previous exercise studies that compared hormonal responses between NF and fluid replacement treatments (4, 9) suggest that differences in either PV or plasma Osmo may account for the higher plasma Cort concentrations during exercise. The pattern seen in the NF trial (elevated ACTH and Cort) lends further support to the hypothesis that lower PV or higher Osmo mediates plasma Cort. Glucose does not appear to have an influence on ACTH and Cort because glucose concentrations did not fall below 3.3 mmol/l, the threshold needed to activate hypothalamic-pituitary-adrenal axis hormones during exercise (36). Further studies are warranted to determine the independent role of PV and Osmo before and during exercise-heat stress on plasma stress hormones.

In summary, this study demonstrates that Oral, after exercise-induced Dh, results in a higher HR during subsequent exercise in the heat compared with iv. PV was not different between treatments, but it was observed that the plasma ΔNE concentration was different in Oral during exercise. This suggests that an augmented sympathetic nervous system response in Oral was responsible for the higher HR observed in that trial. Similar responses in thermoregulatory variables between iv and Oral suggest that both rehydration treatments were equally effective in restoring lost body fluids for use during subsequent exercise. ΔACTH and ΔCort concentrations in NF suggest that PV and Osmo mediate plasma concentrations of these hormones.

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