Effect of hydration status on thirst, drinking, and related hormonal responses during low-intensity exercise in the heat


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Effect of hydration status on thirst, drinking, and related hormonal responses during low-intensity exercise in the heat. J Appl Physiol 97: 39–44, 2004. First published February 27, 2004; 10.1152/japplphysiol.00956.2003.—During exercise-heat stress, ad libitum drinking frequently fails to match sweat output, resulting in deleterious changes in hormonal, circulatory, thermoregulatory, and psychological status. This condition, known as voluntary dehydration, is largely based on perceived thirst. To examine the role of preexercise dehydration on thirst and drinking during exercise-heat stress, 10 healthy men (21–72.7 yr, 174.5 ± 2.1 cm, mass = 72.7 ± 1.1 kg, percent body fat = 12.8 ± 1.0%, maximal aerobic power (V\textsubscript{O}_\textsubscript{2 max}) = 57 ± 1 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}) that was approved by the University of Connecticut Institutional Review Board and conformed to 45CFR46. Subjects were nonsmokers with no history of endocrine and/or thermoregulatory disorders. Before beginning the study, subjects were briefed on potential risks and benefits, signed a written informed consent, and completed a medical history and allergy questionnaire. Additionally, body mass (model 700M, SR Instruments, Tonawanda, NY), height (GPM, Zurich, Switzerland), skinfold thickness (11), and V\textsubscript{O}_\textsubscript{2 max} were determined. The V\textsubscript{O}_\textsubscript{2 max} protocol was a continuous exercise in the heat challenges the body’s thermoregulatory systems. Hydration status, a potential result of exercise-heat stress, ad libitum drinking frequently fails to match sweat output, resulting in deleterious changes in hormonal, circulatory, thermoregulatory, and psychological status. This condition, known as voluntary dehydration, is largely based on perceived thirst. To examine the role of preexercise dehydration on thirst and drinking during exercise-heat stress, 10 healthy men (21 ± 1 yr, 57 ± 1 ml·kg\textsuperscript{-1}·min\textsuperscript{-1} maximal aerobic power) performed four randomized walking trials (90 min, 5.6 km/h, 5% grade) in the heat (33°C, 56% relative humidity). Trials differed in preexercise hydration status (hydrated (Eu) or hypohydrated to −3.8 ± 0.2% baseline body weight (Hy)) and water intake during exercise [no water (NW) or water ad libitum (W)]. Blood samples taken preexercise and immediately postexercise were analyzed for hematocrit, hemoglobin, serum aldosterone, plasma osmolality (P\textsubscript{osm}), plasma vasopressin (P\textsubscript{AVP}), and plasma renin activity (PRA). Thirst was evaluated at similar times using a subjective nine-point scale. Subjects were thirstier before (6.65 ± 0.65) and drank more during Hy+W (1.65 ± 0.18 liters) than Eu+W (1.59 ± 0.41 and 0.31 ± 0.11 liters, respectively). Postexercise measures of P\textsubscript{osm} and P\textsubscript{AVP} were significantly greater during Hy+NW and plasma volume lower [Hy+NW = −5.5 ± 1.4% vs. Hy+W = +1.0 ± 2.5% (P = 0.059), Eu+NW = −0.7 ± 0.6% (P < 0.05), Eu+W = +0.5 ± 1.6% (P < 0.05)] than all other trials. Except for thirst and drinking, however, no Hy+W values differed from Eu+NW or Eu+W values. In conclusion, dehydration preceding low-intensity exercise in the heat magnifies thirst-driven drinking during exercise-heat stress. Such changes result in similar fluid regulatory hormonal responses and comparable modifications in plasma volume regardless of preexercise hydration state.

CONTINUOUS EXERCISE IN THE heat challenges the body’s thermoregulatory systems. Hyponhydration, a potential result of exercise-heat stress and comparable modifications in plasma volume regardless of preexercise hydration state.

aldosterone; osmolality; renin activity; vasopressin

METHODS

Subjects and prestudy requirements. Ten healthy men participated in this study [age = 21 ± 1 yr, height = 174.5 ± 2.1 cm, mass = 72.7 ± 1.1 kg, percent body fat = 12.8 ± 1.0%, maximal aerobic power (V\textsubscript{O}_\textsubscript{2 max}) = 57 ± 1 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}] that was approved by the University of Connecticut Institutional Review Board and conformed to 45CFR46. Subjects were nonsmokers with no history of endocrine and/or thermoregulatory disorders. Before beginning the study, subjects were briefed on potential risks and benefits, signed a written informed consent, and completed a medical history and allergy questionnaire. Additionally, body mass (model 700M, SR Instruments, Tonawanda, NY), height (GPM, Zurich, Switzerland), skinfold thickness (11), and V\textsubscript{O}_\textsubscript{2 max} were determined. The V\textsubscript{O}_\textsubscript{2 max} protocol was a

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modification of that described by Costill and Fox (9). Briefly, after a 3-min warm-up (5.6 km/h), subjects ran at 12.1 km/h; every 2 min, treadmill grade was increased 2% until the subject reached volitional exhaustion. Specific criteria ensured attainment of \( V_{\text{O}_2\text{max}} \) (38).

To reduce individual variability associated with exercise-heat exposure, subjects exercised on 4 consecutive days in a hot environment (preliminary exercise-heat exposure sessions) before (5.5°C exposure, subjects exercised on 4 consecutive days in a hot environment (costill and fox (9)). Brie...
HYDRATION STATE, WATER INTAKE, AND EXERCISE IN THE HEAT

Table 1. Body mass measurements before and after the dehydration and exercise protocols

<table>
<thead>
<tr>
<th>Trial</th>
<th>Predehydration</th>
<th>Postdehydration</th>
<th>Preexercise</th>
<th>Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eu+NW</td>
<td></td>
<td></td>
<td>72.7±0.2</td>
<td>71.8±0.2*</td>
</tr>
<tr>
<td>Eu+W</td>
<td></td>
<td></td>
<td>72.6±0.2</td>
<td>71.9±0.2*</td>
</tr>
<tr>
<td>Hy+NW</td>
<td>72.8±0.2</td>
<td>-3.4±0.2%</td>
<td>70.1±0.2†</td>
<td>69.0±0.20†</td>
</tr>
<tr>
<td>Hy+W</td>
<td>73.0±2.0</td>
<td>-3.6±0.2%</td>
<td>70.2±0.2→</td>
<td>70.8±0.20+‡‡</td>
</tr>
</tbody>
</table>

Values are means ± SE. Values enclosed by arrows represent percent changes in body mass (in kg) from one time point to another. A time of 17.5 ± 1.1 h separated the dehydration protocol from the exercise protocol. Eu, preexercise euhydration; Hy, preexercise hypohydration to -3.8 ± 0.2% baseline body weight; NW, no water intake during exercise; W, ad libitum water intake during exercise. *Significant difference compared with all other conditions at the same time point, P < 0.05. †Significant difference compared with both Eu trials at similar time points, P < 0.05. ‡Significant difference compared with Hy+NW at a similar time point, P < 0.05.

Fluid-regulating hormones. P_{AVP} responses are presented in Fig. 2. Hy conditions showed a nonsignificant trend toward increased baseline P_{AVP}. P_{AVP} for Eu+W was statistically greater at IP (12.22 ± 5.83 × 10^{-3} fmol/l) than Pre (1.11 ± 0.42 × 10^{-3} fmol/l). A similar increase was seen in Hy+NW, where P_{AVP} at IP was higher than all other conditions. PRA and S_{Ald} responses are shown in Fig. 3. Baseline PRA was similar among conditions at Pre, significantly rising at IP for both NW trials. At IP, Hy+NW PRA was greater than both Eu treatments. S_{Ald} significantly increased from Pre to IP in all trials except Eu+W. Like PRA, Hy+NW S_{Ald} at IP was greater than the corresponding Eu conditions.

DISCUSSION

Considering that subjects completed the same exercise bouts during Hy+W and Eu+W, these results suggest that preexercise dehydration was primarily responsible for the differences in thirst and fluid intake. Although this relationship between dehydration and thirst has been seen at rest (12), a primary finding of this study was that the extended period of hypohydration before low-intensity exercise magnified the drive to drink such that the hormonal and circulatory measures of exercise-heat stress were indistinguishable from results obtained when exercise was initiated in a euhydrated state. Thus significant preexercise dehydration favorably modified the drinking response during this exercise-heat stress.

We recognize that the exercise performed the day before the protocol to induce hypohydration for the Hy+NW and Hy+W trials was a potential weakness of the study design. Given the real-life applicability of low-intensity, prolonged exercise in the heat on consecutive days, however, we believe use of an exercise-dehydration model, as opposed to one offering more control (i.e., passive heating and/or diuretic usage), signifi-

Table 2. Hemoglobin, hematocrit, and PV changes

<table>
<thead>
<tr>
<th>Trial</th>
<th>Hemoglobin, g/dl</th>
<th>Hematocrit, %</th>
<th>PV Shift, % Pre to IP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eu+NW</td>
<td>14.1±0.3</td>
<td>44.6±0.8</td>
<td>-0.7±0.6</td>
</tr>
<tr>
<td>Eu+W</td>
<td>14.1±0.2</td>
<td>44.7±0.7</td>
<td>0.5±1.6</td>
</tr>
<tr>
<td>Hy+NW</td>
<td>14.4±0.3*</td>
<td>45.0±0.8</td>
<td>-5.5±1.4*</td>
</tr>
<tr>
<td>Hy+W</td>
<td>14.0±0.3</td>
<td>44.4±0.6</td>
<td>1.0±2.5</td>
</tr>
</tbody>
</table>

Values are means ± SE. For hemoglobin, significant main effects of trial (Hy+NW > Eu+W and Hy+W) were noted. *Significant difference compared with all other conditions at the same time point (P < 0.05). †Significant differences compared with Hy+W at the same time point and compared with IP within a trial (P < 0.05). For hematocrit, a significant main effect of trial was noted (Hy+W < Eu+NW and Hy+NW) (P < 0.05). For plasma volume (PV) shift: ‡Significant difference compared with Eu+NW and Eu+W (P < 0.05) and nearly significant difference compared with Hy+W (P = 0.059). Pre, preexercise; IP, immediately postexercise.
cantly improved the external validity of the present research. PV, \(P_{osm}\), and fluid regulatory hormones rapidly return to baseline or near-baseline values after low- to moderate-intensity exercise (i.e., <2 h), further supporting the present research design (25, 41). Additionally, the similar Pre hormonal and circulatory responses of Hy+W and Hy+NW support the contention that the exercise bout used to hyphiydrate subjects was not responsible for the observed responses.

\(P_{osm}\), perceived thirst, and PV. Normal values for \(P_{osm}\) after prolonged dehydration and/or low-intensity exercise in the heat (280–295 mosmol/kgH\(_2\)O) were seen at all times except for postexercise Hy+NW. Despite the 3–4% loss in body weight preceding the Hy trials, subjects were able to maintain normal \(P_{osm}\) when allowed to drink water ad libitum (IP, Hy+W = 293 ± 3 mosmol/kgH\(_2\)O) but not when denied fluids (IP, Hy+NW = 307 ± 3 mosmol/kgH\(_2\)O). The further rise in \(P_{osm}\) during Hy+NW (Fig. 1) likely resulted from the absence of fluid to counteract exercise-induced increases in sweat volume and a concomitant decrease in PV. Because a \(P_{osm}\) of 295 mosmol/kgH\(_2\)O is the putative osmolar threshold for thirst (40), the effectiveness of osmolar-driven thirst and drinking is reinforced.

Engell et al. (12) have shown a strong, direct relationship between the intensity of perceived thirst and degree of hypohydration before and after exercise in the heat. Additionally, they reported a direct correlation between hydration before and after exercise in the heat. Additionally, the uniform responses in perceived thirst and \(P_{osm}\) during rehydration and the level of dehydration. The increased drinking frequency and fluid intake during Hy+W, complemented by the uniform responses in perceived thirst and \(P_{osm}\) at Pre and IP, support these conclusions. This study extends the previous findings by showing that the relationship between hypohydration, thirst, and drinking holds true during low-intensity exercise in the heat.

The ability of thirst-driven drinking to prevent hypohydration during Hy+W differs, however, with the classic studies of Adolph (1) and Wolf (43), who proposed that thirst was an inadequate stimulus to maintain appropriate hydration. The dehydration work of Adolph (1), however, documented water intake during exercise initiated in a euhydrated state. Because subjects were required to maintain a hypohydrated state in both the Hy trials for several hours before the exercise trial, fluid intake during the 90-min exercise bouts may be better viewed as a function of rehydration than voluntary dehydration. Although the volume of fluid ingested during rehydration after a dehydration session is only ~50% of the fluid lost (12, 26, 30), studies examining this phenomenon have used rehydration sessions immediately after dehydration, in theroneutral ambient conditions, and at rest. The addition of two strong dipsogenic factors (i.e., increased time without fluids and exercise-heat stress) may have contributed to the significantly increased fluid intake (~60% of prior fluid loss) seen during Hy+W.

\textbf{Fluid-regulating hormones.} That \(P_{osm}\) dictated \(P_{AVP}\) responses during Hy+W and Eu+NW is not surprising, because \(P_{osm}\) is the primary regulator of \(P_{AVP}\) (42). However, two anomalies in our study deserve special note. First, significant differences in baseline \(P_{osm}\) between Hy and Eu were not matched by significant decreases in \(P_{AVP}\). The trend toward an increased \(P_{AVP}\) for preexercise Hy measures did not reach statistical significance likely because \(P_{osm}\) never greatly exceeded 295 mosmol/kgH\(_2\)O (40). Thus, although the increases in resting \(P_{osm}\) may have been statistically significant, it can be argued that it was not physiologically relevant. Second, \(P_{AVP}\) during Eu+W was significantly increased from Pre to IP despite no statistical change in \(P_{osm}\), which is contrary to most studies, which find no change in \(P_{AVP}\) at exercise intensities <40% \(V_O^2\) max (8, 15, 27). In this case, the combination of exercise and heat stress may have induced a sympathetic nervous response, supplementing the nonsignificant stimulation of \(P_{AVP}\) by \(P_{osm}\) (8, 20). It is unclear why this only occurred in the Eu+W trial.

The distinct responses of PRA and \(S_{Ald}\) vs. \(P_{AVP}\) in the present study support a multifactorial drinking regulation system, as proposed by Greenleaf et al. (18). They described two general systems for the regulation of drinking: a sodium-\(P_{osm}\) \(P_{AVP}\) system (2, 39) and a renin-angiotensin II-S\_Ald system (13); the interaction of the two systems controls thirst and fluid replenishment. Greenleaf et al. have suggested that the renin-

![Fig. 2. Group mean (± SE) plasma arginine vasopressin responses. *Significant difference compared with all other values at the same time point, \(P < 0.05\). †Significant difference compared with corresponding Pre value, \(P < 0.05\).](http://jap.physiology.org/)

![Fig. 3. Group mean (± SE) plasma renin activity (A) and aldosterone (B) responses. ANG I, angiotensin I. *Significant difference compared with both Eu values at the same time point, \(P < 0.05\). †Significant difference compared with corresponding Pre value, \(P < 0.05\).](http://jap.physiology.org/)
angiotensin II-S_{AlD} system, influenced by reductions in total body water and PV, is the predominant stimulus to thirst during exercise in the heat. In the present study, however, the combination of 1) the significantly higher Pre P_{osm} in Hy trials (Hy+NW = 296 ± 3 mosmol/kg\(H_2O\), Hy+W = 295 ± 2 mosmol/kg\(H_2O\)) than Eu trials (Eu+NW = 287 ± 2 mosmol/kg\(H_2O\), Eu+W = 287 ± 2 mosmol/kg\(H_2O\}); 2) the comparable responses of P_{osm}, P_{AVP}, and thirst; and 3) the dissimilar patterns of PRA, S_{AlD} and thirst indicate that thirst was more closely associated with P_{osm} and P_{AVP} than with PRA and S_{AlD}. Nose et al. (28) have found similar results, concluding that the sodium-P_{osm}-P_{AVP} system was the dominant influence in dictating thirst during rehydration, after a 2.3% decrease in body mass induced by an exercise-heat stress.

Our results may conflict with those of Greenleaf et al. (18) due to differences in the exercise heat stress (8 continuous days of 2 h of low-intensity exercise in the 39.8°C vs. a single 90-min bout of low-intensity exercise in 33°C) or the importance placed by Greenleaf et al. on reductions in total body water or PV to stimulate the renin-angiotensin II-S_{AlD} system. By allowing fluid balance to equilibrate between dehydration and exercise (17.5 ± 1.1 h), however, the full loss of body mass cannot be attributed to decreases in PV alone, especially considering that several studies have shown PV is selectively restored before intracellular and interstitial fluid compartments after dehydrating exercise in the heat (28, 29, 36). Although it can be assumed that PV remained lower in the Hy conditions compared with the Eu conditions (potentially increasing P_{AVP} and thirst in and of itself), the decrease in PV may have been insufficient to maximally stimulate the renin-angiotensin II-S_{AlD} system. Additionally, during Hy+W, subjects showed no losses in PV during the exercise-heat stress; PV (+1.0%) and body masses (+0.9%) were greater after the trial than before, suggesting that the renin-angiotensin II-S_{AlD} system played a minor role vs. the sodium-P_{osm}-P_{AVP} system.

In conclusion, this investigation suggests that dehydration preceding low-intensity exercise in the heat magnifies thirst-driven drinking, effectively attenuating the negative influences of preexercise hypohydration on PV and fluid-regulatory hormonal responses. Furthermore, the data support the interaction of a minor renin-angiotensin II-S_{AlD} system and a dominant, highly responsive sodium-P_{osm}-P_{AVP} system in regulating thirst in this exercise-heat challenge.

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REFERENCES


