

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

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Maresh, C. M., C. L. Gabaree-Boulant, L. E. Armstrong, D. A. Judelson, J. R. Hoffman, J. W. Castellani, R. W. Kenefick, M. F. Bergeron, and D. J. Casa. 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/jappphysiol.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 ± 1 yr, 57 ± 1 ml·kg⁻¹·min⁻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 [euhydrated (Eu) or hypohydrated to $-3.8 \pm 0.2\%$ 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_{osm}), plasma vasopressin (P_{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_{osm} and P_{AVP} were significantly greater during Hy+NW and plasma volume lower [Hy+NW = $-5.5 \pm 1.4\%$ vs. Hy+W = $+1.0 \pm 2.5\%$ ($P = 0.059$), Eu+NW = $-0.7 \pm 0.6\%$ ($P < 0.05$), Eu+W = $+0.5 \pm 1.6\%$ ($P < 0.05$)] 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.

aldosterone; osmolality; renin activity; vasopressin

CONTINUOUS EXERCISE IN THE heat challenges the body's thermoregulatory systems. Hypohydration, a potential result of exercise in the heat, also influences thermoregulation (31, 33), limiting effective temperature maintenance (35). The extent to which thermoregulation is adversely affected dictates the physiological responses, from decrements in physical performance to thermal injury and, in severe cases, even death (21).

Under resting conditions, hypohydration is normally balanced and prevented by increases in thirst-driven drinking, which adequately stimulates fluid intake (17). Physiologically, thirst results from increases in the osmotic pressure of extra-

cellular fluid (37) and is closely tied to several fluid-regulating hormones. During exercise, however, the human thirst mechanism may be insufficient. Despite exercise-induced increases in extracellular osmotic pressure, blood osmolality, and angiotensin II [a powerful dipsogen (32)], enhanced thirst may not sufficiently promote fluid intake to maintain water balance, a phenomenon known as "voluntary dehydration" (42). Despite free access to fluids, exercising subjects voluntarily replace only 66–75% of their net water loss (5, 19, 22, 31).

Repeated days of prolonged low-intensity exercise in a hot, humid environment represent a real-life occupational or recreational scenario for many adults. In examining this situation, our laboratory previously reported that preexercise hypohydration and the corresponding elevations in plasma osmolality (P_{osm}) resulted in a substantial water intake during exercise that increased body mass by 0.9% and attenuated the rise in rectal temperature (T_{re}) via favorable changes in P_{osm} and sweat sensitivity (4). Although research has examined the relationship between hormonal response to exercise-heat stress and hydration status (6, 7, 14, 24), the relationships among thirst, fluid intake, and hydration status (12), and the relationship between plasma volume (PV) and hormonal response to exercise-heat stress (16, 34), none has considered all these factors simultaneously. Therefore, the purpose of this study was to examine the effect of hydration status on perceived thirst, drinking, plasma volume, and hormonal responses during low-intensity exercise in the heat. Measures of circulatory and thermal strain obtained concomitant with this study have been published elsewhere (4). We hypothesized that fluid deprivation would stimulate thirst-driven drinking during exercise, favorably altering hormonal and circulatory measures of exercise-heat stress.

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 = $12.8 \pm 1.0\%$, maximal aerobic power ($\dot{V}O_{2\text{max}}$) = 57 ± 1 ml·kg⁻¹·min⁻¹] that was approved by the University of Connecticut Institutional Review Board and conformed to 45CRF46. 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 $\dot{V}O_{2\text{max}}$ were determined. The $\dot{V}O_{2\text{max}}$ 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 $\dot{V}O_{2\max}$ (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 ± 0.6 days) the experimental trials (4). During these sessions, subjects cycled (model 818E, Monark, Stockholm, Sweden) at $47 \pm 2\%$ $\dot{V}O_{2\max}$ for ~ 90 min in hot conditions ($33 \pm 1^\circ\text{C}$, $64 \pm 8\%$ relative humidity). Heart rate (HR) (UNIQ, Computer Instrument, Hempstead, NY) and T_{re} were continuously measured via a lead I configuration and a thermistor (model 43TF, 400 series probe, Yellow Springs Instruments, Yellow Springs, OH) inserted 10 cm beyond the external anal sphincter, respectively. Drinking was permitted ad libitum and encouraged. During each session, subjects wore shorts, T-shirt, socks, and running shoes. If a subject's HR or T_{re} exceeded 180 beats/min or 39.5°C , respectively, for 5 min, exercise was stopped. Even if exercise ceased, subjects remained in the chamber for the full 90 min.

Experimental design. Subjects completed four experimental trials differing in preexercise hydration status [euhydrated (Eu) or hypohydrated (Hy)] and water intake during exercise [water ad libitum (W) or no water (NW)]. Trial order was randomized; a minimum of 72 h separated each trial. All sessions for a given subject were standardized for time of day. For Eu trials, euhydration was ensured by instructing subjects to drink 474 ml of water the evening before and 474 ml water the morning of those trials (4). For Hy trials, subjects dehydrated ($-3.5 \pm 0.2\%$ body weight) the day before by several hours (3.2 ± 0.4 h) of intermittent exercise (cycling and/or walking) while wearing a cotton sweat suit and running shoes. Body masses were obtained immediately before and routinely throughout the dehydration protocol. Once the desired reduction in body mass (-3%) was attained, subjects refrained from fluid consumption until permitted to drink the next day. Additionally, subjects refrained from eating during the 4 h preceding trials. The 17.5 ± 1.1 -h period between the dehydration protocol and the subsequent experimental trial amply allowed for equilibration of body fluids (4). During one Eu and one Hy trial, subjects drank water ad libitum during exercise (Eu+W and Hy+W, respectively); during the other Eu and Hy trial, drinking was prohibited (Eu+NW and Hy+NW, respectively). All four experimental trials for each subject were completed within 20.0 ± 1.0 days of his last preliminary exercise-heat exposure session.

Experimental protocol. On arrival to the laboratory, subjects provided a urine sample and body mass was measured (wearing shorts and socks). To assess hydration status, urine specific gravity was determined in triplicate via refractometer (model A300CL, Spartan, Tokyo, Japan) and later verified by the preexercise P_{osm} value. A urine specific gravity <1.020 and P_{osm} between 280 and 287 mosmol/kgH₂O indicated euhydration (3). A 20-gauge Teflon cannula (Critikon, Tampa, FL) was then inserted into a superficial forearm vein and a male luer-adaptor (model 5877, Abbott Hospital, Chicago, IL) was attached to the cannula to acquire subsequent blood samples. The cannula was kept patent with isotonic saline. After placement of the cannula, subjects stood quietly for 20 min in the environmental chamber, after which the first experimental blood sample (Pre) was drawn.

Subjects then walked on a motor-driven treadmill for 90 min at 5.6 km/h at 5% grade in hot conditions ($33 \pm 0^\circ\text{C}$ and $55.6 \pm 2.7\%$ relative humidity). Treadmill speed was repeatedly verified by application of a tachometer (model 8204-20, Cole-Parmer Instrument, Chicago, IL) to the moving treadmill belt. Several circulatory and thermoregulatory measures were also obtained during exercise; details of these findings have been reported elsewhere (4).

At the completion of exercise, body mass was measured. Immediately (IP; within 1 min) after exercise completion, blood samples were drawn. All blood samples were obtained without stasis and transferred to plain, chilled EDTA, or chilled heparinized tubes. Hematocrit

(Hct), hemoglobin (Hb), and P_{osm} were evaluated immediately after blood sampling. All other blood samples were centrifuged, aliquoted, and frozen (-90°C) until subsequent analysis.

Thirst scale. Concomitant with blood draws (Pre and IP), subjects were asked to numerically identify their perceived thirst by using a 9-point thirst scale with verbal anchors ranging from 1 ("not thirsty at all") to 9 ("very, very thirsty") (23).

Blood analysis. Hct was assessed in triplicate via microcapillary technique after 4 min of centrifuging at 9,500 g. Values were not corrected for trapped plasma. Hb was ascertained in duplicate via an enzymatic, photometric technique (Reflotron, Boehringer Mannheim Diagnostics, Indianapolis, IN). Percent changes in PV were calculated from Hct and Hb values (10). P_{osm} was evaluated in duplicate via freezing-point depression (model 5004, Precisions Systems, Natick, MA).

After extraction on silica columns (Incstar, Stillwater, MN), plasma arginine vasopressin (P_{AVP}) was determined in duplicate via a commercially available radioimmunoassay kit (Incstar). Extraction recovery was 91.3%. Assay sensitivity was 0.65×10^{-3} fmol/l. The within- and between-assay coefficients of variation were 5.6 and 12.7%, respectively. Plasma renin activity (PRA) was ascertained in duplicate via radioimmunochemical determination of plasma angiotensin I generated during 1 h of incubation at pH 6.0 (Incstar). Assay sensitivity was $0.018 \text{ ng} \cdot 100 \mu\text{l}^{-1} \cdot \text{h}^{-1}$. The within- and between-assay coefficients of variation were 4.1 and 10.9%, respectively. Serum aldosterone (S_{Ald}) was evaluated in duplicate via radioimmunoassay (Diagnostics Products, Los Angeles, CA). Assay sensitivity was 44.4 pmol/l. The within- and between-assay coefficients of variation were 4.2 and 8.3%, respectively.

Statistics. Data were analyzed via a repeated-measures analysis of variance (condition \times time). Significant *F*-ratios were further evaluated with a Fisher's post hoc test. Statistical significance was set at $P < 0.05$. Values reported are means \pm SE.

RESULTS

Body mass, P_{osm} , perceived thirst, water intake, sweat loss, and PV. Body mass data are shown in Table 1. The dehydration protocol successfully decreased body mass, as preexercise body mass measures before the exercise protocol during Hy trials were significantly lower than corresponding Eu trials. During Eu+NW, Eu+W, and Hy+NW, body mass significantly fell from pre- to postexercise. During Hy+W, however, body mass significantly increased during the exercise, causing the postexercise body mass to be statistically greater after Hy+W than after Hy+NW.

P_{osm} and perceived thirst responses are presented in Fig. 1. Pre P_{osm} and perceived thirst were higher in both Hy conditions (Hy+NW: $P_{osm} = 296 \pm 3$ mosmol/kgH₂O, perceived thirst = 5.47 ± 0.76 ; Hy+W: $P_{osm} = 295 \pm 2$ mosmol/kgH₂O, perceived thirst = 6.65 ± 0.65) than Eu trials (Eu+NW: $P_{osm} = 287 \pm 2$ mosmol/kgH₂O, perceived thirst = 2.00 ± 0.35 ; Eu+W: $P_{osm} = 287 \pm 2$ mosmol/kgH₂O, perceived thirst = 1.59 ± 0.41). At IP, P_{osm} and perceived thirst were greater in Hy+NW than all other treatments.

During Hy+W, subjects drank more (1.65 ± 0.18 liters) and more frequently (3.5 ± 0.7 drinks) than during Eu+W (0.31 ± 0.11 liter and 1.5 ± 0.9 drinks, respectively). Sweat loss was stable and similar during all treatments (Hy+NW = 1.01 ± 0.05 kg, Hy+W = 0.96 ± 0.05 kg, Eu+NW = 0.94 ± 0.04 kg, Eu+W = 0.96 ± 0.04 kg). Hb, Hct, and changes in PV are shown in Table 2. The percent change in PV significantly decreased at IP in Hy+NW. For all other conditions, PV was stable and similar throughout.

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

Trial	Predehydration	Postdehydration	Preexercise	Postexercise
Eu+NW			72.7±2.0	71.8±2.0*
			← -1.4±0.1% →	
Eu+W			72.6±2.0	71.9±2.0*
			← -1.0±0.2% →	
Hy+NW	72.8±2.0	70.3±2.0	70.1±2.0†	69.0±2.0*†
	← -3.4±0.2% →		← -1.6±0.1% →	
		← -3.7±0.2% →		
			← -5.2±0.2% →	
Hy+W	73.0±2.0	70.3±1.9	70.2±2.0†	70.8±2.0*†‡
	← -3.6±0.2% →		← +0.9±0.2% →	
		← -3.7±0.2% →		
			← -3.0±0.3% →	

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 $-3.8 \pm 0.2\%$ baseline body weight; NW, no water intake during exercise; W, ad libitum water intake during exercise. *Significant difference compared with corresponding preexercise body mass, $P < 0.05$. †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 ($12.22 \pm 5.83 \times 10^{-3}$ fmol/l) than Pre ($1.11 \pm 0.42 \times 10^{-3}$ 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-

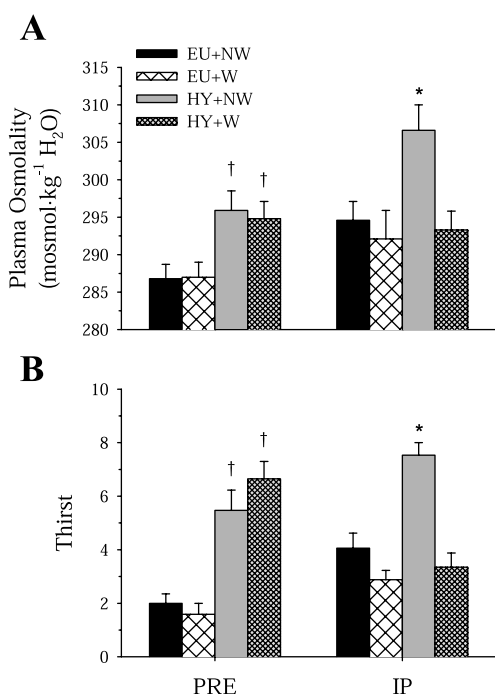


Fig. 1. Group mean (\pm SE) plasma osmolality (A) and perceived thirst (B) responses. Eu, preexercise euhydration; HY, preexercise hypohydration to $-3.8 \pm 0.2\%$ baseline body weight; NW, no water during exercise; W, water ad libitum during exercise; Pre, preexercise; IP, immediately postexercise. *Significant difference compared with all other values at the same time point and compared with corresponding Pre values, $P < 0.05$. †Significant difference compared with both Eu values at the same time point, $P < 0.05$.

Table 2. Hemoglobin, hematocrit, and PV changes

Trial	Hemoglobin, g/dl		Hematocrit, %		PV Shift, % Pre to IP
	Pre	IP	Pre	IP	
Eu+NW	14.1±0.3	14.2±0.3	44.6±0.8	44.7±0.7	-0.7±0.6
Eu+W	14.1±0.2	14.1±0.3	44.7±0.7	44.8±0.8	0.5±1.6
Hy+NW	14.4±0.3†	15.0±0.4*	45.0±0.8	46.0±0.8	-5.5±1.4‡
Hy+W	14.0±0.3	13.9±0.2	44.4±0.7	44.4±0.6	1.0±2.5

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+W 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.

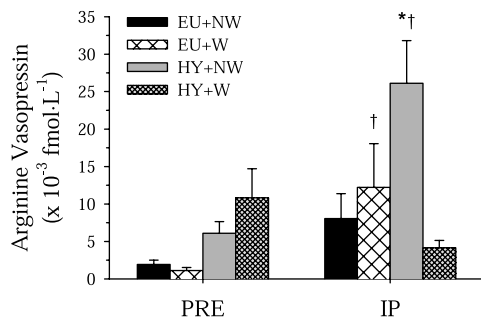


Fig. 2. Group mean (\pm SE) plasma arginine vasopressin responses. *Significant difference compared with all other values at the same time point, $P < 0.05$. †Significant difference compared with corresponding Pre value, $P < 0.05$.

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 hypohydrate 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/kgH₂O) 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/kgH₂O) but not when denied fluids (IP, Hy+NW = 307 ± 3 mosmol/kgH₂O). 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/kgH₂O 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 fluid consumption 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 disagrees, 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 $\sim 50\%$ of the fluid lost (12, 26, 30),

studies examining this phenomenon have used rehydration sessions immediately after dehydration, in thermoneutral 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 ($\sim 60\%$ of prior fluid loss) seen during Hy+W.

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/kgH₂O (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 $< 40\%$ $\dot{V}O_{2\max}$ (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-

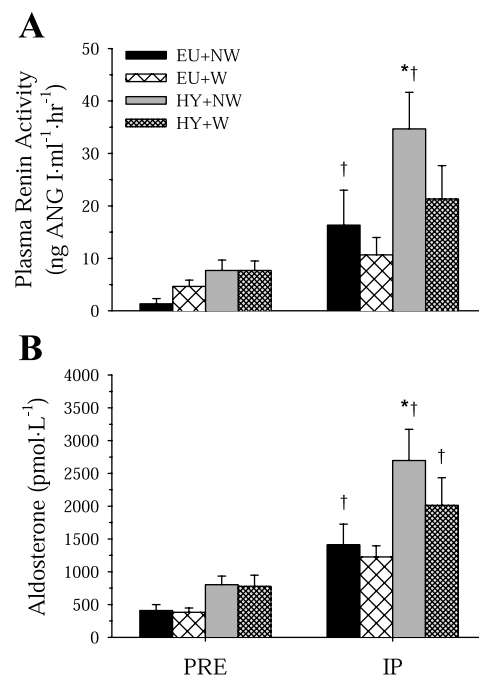


Fig. 3. Group mean (\pm SE) plasma renin activity (A) and aldosterone (B) responses. ANG I, angiotensin I. *Significant difference compared with both Eu values at the same time point, $P < 0.05$. †Significant difference compared with corresponding Pre value, $P < 0.05$.

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 ($Hy+NW = 296 \pm 3$ mosmol/kgH₂O, $Hy+W = 295 \pm 2$ mosmol/kgH₂O) than Eu trials ($Eu+NW = 287 \pm 2$ mosmol/kgH₂O, $Eu+W = 287 \pm 2$ mosmol/kgH₂O); 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 \pm 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 measures. 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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