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Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake

LAWRENCE E. ARMSTRONG, CARL M. MARESH, CATHERINE V. GABAREE, JAY R. HOFFMAN, STAVROS A. KAVOURAS, ROBERT W. KENEFICK, JOHN W. CASTELLANI, AND LYNN E. AHLQUIST
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Armstrong, Lawrence E., Carl M. Maresh, Catherine V. Gabaree, Jay R. Hoffman, Stavros A. Kavouras, Robert W. Kenefick, John W. Castellani, and Lynn E. Ahlquist. Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. *J. Appl. Physiol.* 82(6): 2028–2035, 1997.—This investigation examined the distinct and interactive effects of initial hydration state, exercise-induced dehydration, and water rehydration in a hot environment. On four occasions, 10 men performed a 90-min heat stress test (treadmill walking at 5.6 km/h, 5% grade, 33°C, 56% relative humidity). These heat stress tests differed in pretest hydration [2 euhydrated (EU) and 2 hypohydrated (HY) trials] and water intake during exercise [2 water ad libitum (W) and 2 no water (NW) trials]. HY + NW indicated greater physiological strain than all other trials ($P < 0.05$–0.001) in heart rate, plasma osmolality (P_{osm}), sweat sensitivity (g/°C·min), and rectal temperature. Unexpectedly, final HY + W and EU + W responses for rectal temperature, heart rate, and P_{osm} were similar, despite the initial 3.9 ± 0.2% hypohydration in HY + W. We concluded that differences in pretest P_{osm} (295 ± 7 and 287 ± 5 mosmol/kg for HY + W and EU + W, respectively) resulted in greater water consumption (1.65 and 0.31 liter for HY + W and EU + W, respectively), no voluntary dehydration (0.9% body mass increase), and attenuated thermal and circulatory strain during HY + W.

temperature regulation; body temperature; plasma; fluid shifts; rehydration

STUDIES SPANNING 50 years have demonstrated that hypohydration effects an increased core temperature (27) subsequent to reduced blood volume, hyperosmolality, skin blood flow, and sweat rate (21, 27); an increased cardiovascular strain associated with body water loss, hypovolemia, peripheral vasodilation, tachycardia, decreased venous return, and decreased stroke volume (6, 25); and a decreased capacity to perform submaximal endurance exercise (25). Similarly, it has been documented that minor dehydration (i.e., −1 to −2% of body weight) augments core temperature and cardiovascular strain (13, 19, 23, 26), that the increase in these variables is directly related to the magnitude of dehydration accrued during prolonged exercise (18, 20, 26), and that the optimal rate of rehydration approximates the rate of sweat production (19). Although numerous studies have replaced sweat losses with carbohydrate-electrolyte formulations, few investigations have examined the effects of pure water replacement during exercise on thermal and circulatory responses (7), despite the fact that water is the most common replacement fluid in athletic, industrial, and military settings. Also we are unaware of any previous study that has isolated the effects of hypohydration, dehydration, and water rehydration as they influence temperature regulation and physiological strain during prolonged upright exercise (15, 23). This is significant because rehydration during exercise maintains sweating and/or skin blood flow (7), thereby preserving the ability to dissipate heat, and reduces cardiovascular strain (19). Therefore, the first purpose of this investigation was to determine the distinct effects of preexercise hypohydration (HY, −3.6 ± 0.2 and −3.9 ± 0.2% body mass) and euhydration (EU), with ad libitum water intake (W) or no water intake (NW) during exercise, on thermoregulatory, fluid balance, and circulatory responses. The interactions of HY, EU, W, and NW also were of interest, because combinations of these factors may differentially affect the nature and magnitude of responses to exercise-heat stress (6, 16). Ten test subjects performed four tests (EU + W, EU + NW, HY + W, and HY + NW) involving 90 min of graded treadmill walking in a hot environment. Because exercise-induced strain is directly related to the level of hypohydration (18, 20, 26), we hypothesized that the magnitude of physiological perturbations in the four experimental conditions would be ranked in the following order: HY + NW > HY + W > EU + NW > EU + W.

Furthermore, it is widely recognized that active humans do not voluntarily replace all the water lost during prolonged exercise in heat (14). Known as voluntary dehydration (14, 28), this behavior is complex, involves psychological (i.e., alliesthesia) and physiological components, and results in increased core temperature and cardiovascular strain even when test subjects begin exercise in the euhydrated state. The exact means by which extracellular tonicity affects voluntary dehydration is not known, but it may be due to the fact that plasma osmolality (normal mean: 287 mosmol/kg) does not rise to the threshold (295 mosmol/kg) for thirst (30) until late in the exercise period (14). This hypothetically suggested that, if humans began exercise in a HY state with an elevated plasma osmolality (P_{osm}) and an activated thirst drive, exercise-heat exposure would involve greater total water intake (vs. EU) and perhaps attenuated physiological strain. Therefore, the second purpose of this study was to determine the differential effects of initial HY and EU states on ad libitum water consumption, fluid balance, and physiological responses.

METHODS

Subjects. Ten healthy male university students participated in the investigation. Each subject signed an informed consent statement that had been approved by the University of Connecticut Institutional Review Board. An activity questionnaire indicated that these subjects were nonsmokers and that they regularly participated in recreational sport activities but were not athletes. Their medical histories included no previous heat illness, thermoregulatory disorder, or endocrine dysfunction. Their age was 21 ± 1 yr, height was 174.5 ± 2.1 cm, body mass was 72.70 ± 2.13 kg, surface area was 1.9 ± 0.1 m^2, surface area-to-mass ratio was 256 ± 3 cm^2/kg, and maximal aerobic power ($\dot{V}O_{2max}$) was 57.1 ± 1.5 $ml \cdot kg^{-1} \cdot min^{-1}$.

Protocol. Before experimental testing, each subject's $\dot{V}O_{2max}$ was determined by a continuous treadmill running test (5) verified by a plateau of oxygen uptake (<150 ml O_2) with an increase in exercise intensity. Subsequently, subjects completed four consecutive days of preliminary exercise-heat exposure. The purposes of these exposures were to enhance cardiovascular stability, reduce the risk of heat illness, reduce between-subject variability in measurements, and determine whether any subject probably would not be able to complete daily 90-min tests (13). These preliminary sessions involved cycle ergometer exercise at 47 ± 2% $\dot{V}O_{2max}$ in an environmental chamber (33 ± 1°C, 64 ± 8% relative humidity, 0.1 ± 0.1 m/s air speed). Ambient conditions were monitored during all tests by two instruments: a thermohygrometer (model 3309–60, Cole-Parmer Instrument, Chicago, IL) and a thermoanemometer (model 9850, Alnor Instrument, Skokie, IL). Water was consumed ad libitum. Subjects wore shorts, T-shirt, socks, and athletic shoes. Exercise lasted 80 ± 2 min, unless terminated by predetermined end points of heart rate (HR) >180 beats/min for 5 min, a rectal temperature (T_{re}) >39.5°C, or clinical signs and symptoms of heat exhaustion.

Within 20 ± 1 days of the conclusion of this preliminary program, each subject had completed four experimental heat stress tests (HST) in a hot environment (33 ± 1°C, 56 ± 5% relative humidity, 0.1 ± 0.1 m/s air speed); for most subjects 3–4 days of rest were allowed between HST. The four HST differed in pretest hydration (2 EU and 2 HY trials) and whether subjects consumed chilled water (~10–15°C) during exercise (2 W and 2 NW trials): EU + W, EU + NW, HY + W, and HY + NW. To reduce the likelihood of an order effect, the sequence of treatments was randomized and HST were separated by ≥3 days. Subjects began each HST at the same time of day and wore the same clothing (i.e., shorts, T-shirt, socks, and athletic shoes) during all trials. Each HST lasted 90 min (treadmill walking, 5.6 km/h, 5% grade, 36 ± 2% $\dot{V}O_{2max}$), unless terminated by the predetermined end points described above.

Preparation for HY and EU sessions. Before all HST, subjects abstained from strenuous exercise for 24 h and consumed no food for 4 h. The preexercise hydration status of each subject was verified within 30 min of the start of all HST by triplicate measurements of urine specific gravity (refractometer, Spartan) and body mass and was later confirmed by P_{osm} (osmometer, model 5004, Precision Instruments, Natick, MA) (2).

A baseline body mass was determined for each subject from the average of three to five preprandial morning measurements taken on the days between preliminary exercise-heat exposures and HST. Before HY + NW and HY + W, the subjects dehydrated to −3.4 ± 0.2 and −3.6 ± 0.2%, respectively, of the baseline body mass. This was accomplished via various forms of mild-to-moderate exercise (i.e., jogging, cycling, weight lifting) while the subjects wore a cotton sweat suit over the clothing items described above. Dehydration was accomplished in 3.0 ± 0.5 (HY + NW) and 3.3 ± 0.4 h (HY + W). After the subjects had achieved the desired body mass, they were instructed to consume no fluids before the HST, which began on the next day 17 ± 1 (HY + NW) and 18 ± 1 h (HY + W) later. This resulted in additional overnight body mass losses of 0.2% for HY + NW and 0.3% for HY + W, increasing the total mass losses (immediately before HST) to −3.6 ± 0.2% for HY + NW and −3.9 ± 0.2% for HY + W. Before EU + NW and EU + W, subjects underwent no programmed dehydration and consumed four large glasses of water (>470 ml total) in excess of their normal dietary fluid intake: two glasses before going to sleep on the night before testing and two on awakening on the morning of testing.

Measurements. HR and T_{re} were recorded at 10-min intervals, during each of the four preliminary exercise-heat exposures and four HST, to monitor physiological strain. Chest surface electrodes (lead I configuration) transmitted HR to an external digital receiver (Computer Instrument, Hempstead, NY). T_{re} was monitored by using a thermistor (series 400, Yellow Springs Instruments, Yellow Springs, OH) inserted 10–12 cm beyond the anal sphincter and connected to a thermistor thermometer (model 08402, Cole-Parmer Instrument). The T_{re} was graphed against time (29), allowing computation of the area under the heating curve (i.e., the integral °C·min). Skin temperature was obtained at the chest, forearm, and calf by placing an infrared temperature scanner (Ototemp 3000, Exergen, Newton, MA) on the skin surface, and mean weighted skin temperature ($\overline{T}_{sk}$) was calculated (4).

Body mass was determined on a precision electronic balance (model 700M, SR Instruments, Tonawanda, NY) to an accuracy of ±45 g. Total body sweat rate was calculated from body mass loss (immediate preexercise to postexercise) and was adjusted for water intake, urine output, and evaporative water loss from the respiratory tract (16). Sweat sensitivity was calculated by dividing the sweat loss (g) by the area under the heating curve (°C·min, see above).

Heat storage (HS) in body tissues (W/m^2) was calculated (16) from the formula

$$HS = \frac{0.97\ BW_{pre}(\overline{T}_{b\,post} - \overline{T}_{b\,pre})}{(SA)(t)} \quad (1)$$

where 0.97 is the specific heat of body tissues ($W \cdot h^{-1} \cdot kg^{-1} \cdot °C^{-1}$), BW_{pre} is the preexercise body mass (kg), ($\overline{T}_{b\,post} - \overline{T}_{b\,pre}$) represents the increase in mean body temperature during the 90-min exercise bout (°C), *SA* is the DuBois surface area (m^2) of the body (10), and *t* is the elapsed time (h). The effect of ingesting cool water on body temperature was calculated by using the specific heat of water and of the human body (see above). Radiant heat exchange (*R*) with the environment (W/m^2) was calculated according to the linear approximation equation published by Mitchell and colleagues (17)

$$R = [\sigma \epsilon_{sk}(A_r/SA)(\overline{T}_{sk}{}^4 - \overline{T}_{sur}{}^4)] \quad (2)$$

where σ is the Stefan-Boltzmann constant (5.67×10^{-8} $W \cdot m^{-2} \cdot °K^{-4}$), ϵ_{sk} is the emissivity of the skin (assumed to be 0.99), A_r is the surface area (m^2) of the body that radiates heat, *SA* is the total surface area (m^2) of the body (10), the quantity (A_r/SA) was measured as 0.88, $\overline{T}_{sk}$ is the mean weighted skin temperature (°K), and $\overline{T}_{sur}$ is the mean temperature of the surrounding surfaces (°K). Evaporative heat loss (W/m^2) was calculated by multiplying sweat production

(liters) by the appropriate factor (580 kcal/l sweat) and was corrected by calculating the percentage of sweat that dripped to the floor. Under these environmental and exercise conditions, an iterative computation indicated that 30% of sweat did not evaporate on the skin surface (R. R. Gonzalez, personal communication). Evaporative heat loss was added to radiant heat exchange to derive the total heat dissipation by evaporation and radiation (W/m^2).

Expired oxygen, carbon dioxide, and ventilatory volume were determined by using open-circuit spirometry. Subjects breathed through a two-way valve (model single J, Collins, Braintree, MA), and expired gases were analyzed with an on-line breath-by-breath system (series 2000, Medical Graphics, St. Paul, MN). This metabolic system was calibrated with standard gases before each test. Oxygen consumption was recorded at 30-s intervals during the $\dot{V}O_{2max}$ test, between 10–15 and 40–45 min of preliminary exercise-heat exposures, and between 45–50 and 85–90 min of each HST. Metabolic heat production was calculated from oxygen consumption data (4.8 kcal/l O_2) (3). We also assumed that 80% of metabolic energy evolved as heat (26).

Blood analyses. Venous blood samples during each treatment were obtained before exercise and immediately postexercise. A 20-gauge Teflon cannula (Critikon, Tampa, FL) was placed in a superficial forearm vein. The cannula was kept patent with a 1.5-ml volume of isotonic saline solution at four to six points during each test (<10 ml total). The preexercise blood samples were drawn after a 15-min equilibration period in the environmental chamber. All postexercise blood samples were drawn within 30 s of the conclusion of exercise. Hematocrit was measured in triplicate via the microcapillary technique. Hemoglobin concentrations were determined in duplicate by reflectance photometry (Boehringer Mannheim Diagnostics, Indianapolis, IN). The percent changes in plasma volume and erythrocyte volume were calculated from the appropriate hemoglobin and hematocrit values obtained at rest and after exercise (9). P_{osm} was measured with the freezing-point depression method (model 5004, Precision Instruments). Selected hormonal responses during HST are reported elsewhere (13).

Statistical analyses. Evaluation of the data was accomplished by a treatment × time analysis of variance (i.e., 4 × 2 for blood, 4 × 11 for T_{re} and HR), with repeated measures across time, using a commercial computer program (BMDP Statistical Software, Los Angeles, CA). In the event of a significant *F* ratio, Tukey's multiple comparison analysis was performed to determine specific differences among the sample means. Regression analyses were utilized across treatments to examine the thermoregulatory effects of hypohydration, dehydration, and rehydration. Significance for all statistical tests was established at $P < 0.05$, and all data are expressed as means ± SE.

RESULTS

Preliminary exposures. During the preliminary exercise-heat exposures, subjects experienced a reduction of final HR (178 ± 11 and 165 ± 7 beats/min on *days 1* and *4*, respectively) and final T_{re} (39.3 ± 0.2 and 38.9 ± 0.4°C on *days 1* and *4*, respectively). These responses suggested that partial heat acclimation had occurred in these subjects, because humans require 7–10 days to develop heat acclimation (31). Also, the number of subjects who completed the entire 90-min exercise session increased from *day 1* ($n = 3$) to *day 4* ($n = 8$).

HST. The four 90-min HST were randomized and separated by ≥3 days. The range of total body mass changes (i.e., *day −1* to the end of HST) in all experimental trials was −0.19 to −6.71%. Figure 1 presents comparisons of HR and T_{re} in all treatments. The HY + NW test exhibited significantly greater HR and T_{re} during all exercise measurements beyond 10 min.

Table 1 presents fluid, cardiovascular, metabolic, and thermal variables. The volume of water consumed ad libitum during the HY + W test was 5.3 times greater than that consumed during the EU + W trial. The mean increases in HR per 1% loss of body mass were 38 ± 4, 40 ± 3, and 12 ± 1 beats/min for EU + W, EU + NW, and HY + NW, respectively, during HST. The mean increases in T_{re} per 1% loss of body mass were 0.6 ± 0.1, 0.8 ± 0.1, and 0.4 ± 0.1°C for EU + W, EU + NW, and HY + NW, respectively, during HST.

Table 2 presents the blood and urine variables that are affiliated with Table 1. Mean plasma glucose values indicated that hypoglycemia was not present at the conclusion of any HST. Seven variables in Tables 1 and 2 showed differences between the HY + W and the EU + W or EU + NW treatments; all these involved fluid or circulatory factors.

Figure 2 depicts the relationship between water intake and T_{re} expressed in terms of ΔT_{re} during exercise. Figure 3 shows intratest differences in the

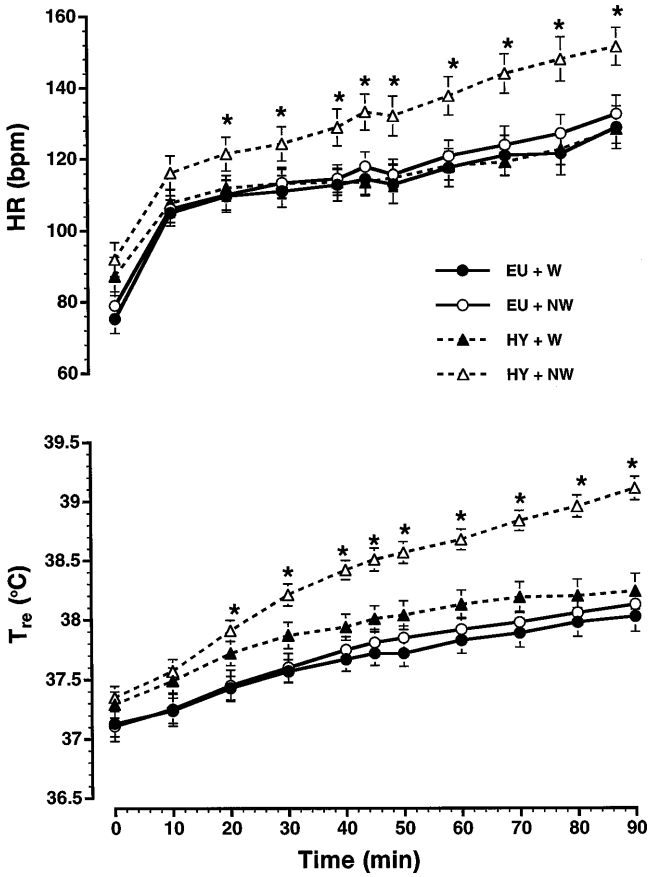

Fig. 1. Heart rate (HR) and rectal temperature (T_{re}) responses during the four 90-min heat stress tests: euhydration + ad libitum water (EW + W), euhydration + no water (EU + NW), hypohydration + ad libitum water (HY + W), and hypohydration + no water (HY + NW). Values are means ± SE; $n = 10$. bpm, Beats/min. *$P <$ 0.05–0.0001 from all other treatments at same time point.

Table 1. *Fluid, cardiovascular, metabolic, and thermal variables*

	Trials			
	EU + W	EU + NW	HY + W	HY + NW
Ad libitum water intake, liters	0.31 ± 0.11	0	1.65 ± 0.18	0
ΔBody mass during HST, %	−1.0 ± 0.2	−1.4 ± 0.1	+0.9 ± 0.2[a,b]	−1.5 ± 0.1[a,c]
Total Δbody mass,[d] %	−1.0 ± 0.2	−1.4 ± 0.1	−3.0 ± 0.2[e]	−5.1 ± 0.2[e]
Final HR, beats/min	129 ± 6	133 ± 6	128 ± 4	152 ± 6[e]
$\dot{V}O_2$, ml·kg^{-1}·min^{-1}	20.3 ± 0.4	19.9 ± 0.6	21.1 ± 0.5	21.2 ± 0.5
Aerobic heat production, W/m^2	210.8 ± 5.7	202.4 ± 8.1	209.2 ± 8.2	210.0 ± 8.0
Total sweat loss, kg	0.96 ± 0.04	0.94 ± 0.04	0.96 ± 0.05	1.01 ± 0.05
Evaporative heat loss, W/m^2	159.9 ± 6.1	155.3 ± 5.4	159.5 ± 8.7	168.7 ± 8.4
Radiative heat loss, W/m^2	8.6 ± 1.0	7.1 ± 1.8	6.5 ± 1.9	9.6 ± 1.6
Sweat rate, g·m^{-2}·h^{-1}	338 ± 13	329 ± 12	338 ± 20	357 ± 19
Heat storage, W/m^2	24.1 ± 3.5	24.1 ± 3.5	17.2 ± 4.1	32.9 ± 3.0[e]
Final T_{re}, °C	38.0 ± 0.1	38.1 ± 0.1	38.2 ± 0.2	39.1 ± 0.1[e]
ΔT_{re}, °C	+0.9 ± 0.1	+1.0 ± 0.1	+1.0 ± 0.1	+1.8 ± 0.1[e]
Final $\bar{T}_{sk}$, °C	35.1 ± 0.2	34.7 ± 0.4	34.6 ± 0.3	35.1 ± 0.3

Values are means ± SE; n = 10. EU, euhydration; HY, hypohydration; W, water ad libitum; NW, no water; $\dot{V}O_2$, O_2 consumption; HR, heart rate; Δ, change during 90-min exercise bout; T_{re}, rectal temperature; $\bar{T}_{sk}$, mean weighted skin temperature; HST, 90-min heat stress test. [a] Significantly different from EU + W ($P < 0.05$–0.001); [b] significantly different from EU + NW ($P < 0.001$); [c] significantly different from HY + W ($P < 0.025$–0.001); [d] during entire protocol; [e] significantly different from all other treatments ($P < 0.05$–0.0001).

relationship between sweat loss and the area under the curve of T_{re} plotted against time (the integral °C·min).

Figure 4 illustrates the effect of water consumption on ΔP_{osm} in EU and HY conditions. Clearly, some subjects consumed ample water to maintain or even decrease P_{osm}. Figure 5 depicts the relationship between sweat sensitivity (g/°C·min) and both total body mass loss (n = 40) and P_{osm} (n = 40) for all HST performed during this investigation. Sweat sensitivity decreased as total body mass loss and P_{osm} increased.

DISCUSSION

Few studies have examined the thermal and cardiovascular responses to pure water replacement during exercise (7) or the concurrent effects of hypohydration, dehydration, and water rehydration on temperature regulation and physiological strain during upright exercise (15, 23). This investigation examined the interactions of preexercise HY and EU with W and NW, because these factors may differentially affect the nature and magnitude of responses to exercise-heat stress (6, 16). Hypohydration was achieved in 3.0–3.3 h by voluntary food and fluid denial combined with physical exercise in a cool environment while subjects wore a cotton sweat suit. A recovery period of 17–18 h was spent in a comfortable environment to provide time for fluid compartments to equilibrate at the achieved hydration level. These dehydration-recovery procedures are consistent with those of previous investigations (26).

We initially hypothesized that the magnitude of physiological perturbations in the four experimental conditions would be ranked in the following order: HY + NW > HY + W > EU + NW > EU + W. In partial support of this hypothesis, the HY + NW trial resulted in greater HR and T_{re} ($P < 0.01$–0.0001) than all other experimental conditions from 20 to 90 min of the HST. HY + NW also resulted in the greatest final HR, heat storage, P_{osm}, ΔP_{osm}, and plasma volume change ($P <$

Table 2. *Blood and urine variables measured in triplicate*

	Trials			
	EU + W	EU + NW	HY + W	HY + NW
P_{osm}, mosmol/kg				
Preexercise	287 ± 6	287 ± 5	295 ± 7*†	296 ± 8*†‡
Postexercise	292 ± 11	294 ± 7	293 ± 8	307 ± 11*†‡§
ΔP_{osm}, mosmol/kg				
Postexercise	+5 ± 2	+7 ± 1	−1 ± 2*	+10 ± 1†§
Δplasma volume, %				
Postexercise	+0.2 ± 1.4	−1.0 ± 0.5	+1.2 ± 2.6	−5.3 ± 1.4†§
Plasma lactate, mmol/l				
Preexercise	1.6 ± 0.2	1.7 ± 0.2	1.2 ± 0.1*†	1.5 ± 0.1
Postexercise	1.3 ± 0.1	1.5 ± 0.1	1.3 ± 0.1	1.4 ± 0.1
Plasma glucose, mmol/l				
Preexercise	5.2 ± 0.5	5.3 ± 0.8	5.2 ± 0.6	5.2 ± 0.6
Postexercise	5.1 ± 0.3	5.2 ± 0.4	4.8 ± 0.3	5.2 ± 0.6
Urine specific gravity				
Preexercise	1.016 ± 0.002	1.015 ± 0.002	1.028 ± 0.001*†	1.029 ± 0.001*†

Values are means ± SE; n = 10. Δ, change during 90-min exercise bout; P_{osm}, osmotic pressure. *Significantly different from EU + NW ($P < 0.025$–0.0001); †significantly different from EU + W ($P < 0.05$–0.0001); ‡significantly different, within treatment ($P < 0.01$); §significantly different from HY + W ($P < 0.01$–0.001).

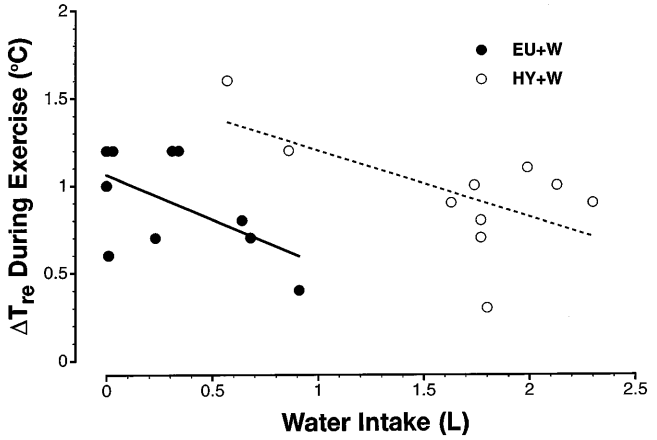

Fig. 2. Relationships between change in T_{re} (ΔT_{re}) and water intake during EU + W and HY + W tests. Slopes of regression lines indicate that this relationship shifted to right when pretest HY was induced.

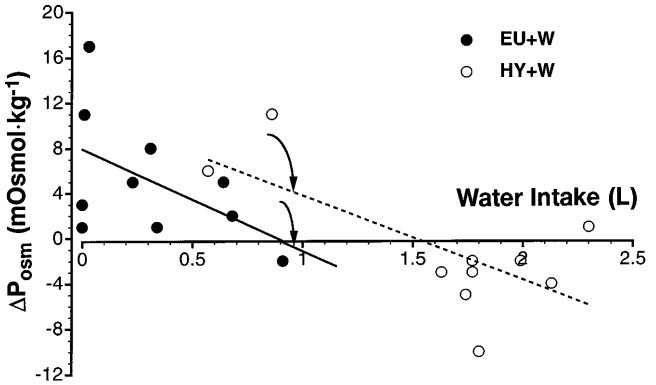

Fig. 4. Relationships between change in plasma osmolality (ΔP_{osm}) and water intake in EU + W and HY + W states. Arrows, water intakes equal to fluid loss (0.96 liter) in both trials.

0.05–0.001). Furthermore, many significant differences ($P < 0.05$–0.001) were observed between the HY + W and HY + NW trials, indicating that the consumption of water had a significant influence on physiological responses when subjects were hypohydrated.

However, the interpretation of responses in the HY + W and EU + W trials was complicated by differences in the volume of water consumed in these trials. For example, when subjects were euhydrated, ad libitum water intake had no significant effect on measured variables (EU + W vs. EU + NW). This outcome was influenced by the fact that four subjects voluntarily drank little or no water during EU + W (Figs. 2 and 4), making their data equivalent to the EU + NW test. As a second example, most HY + W responses were not different from those of EU + W, and some (i.e., Δbody mass and ΔP_{osm}) were significantly smaller. It is likely that this outcome resulted from the great difference in ad libitum water intake between HY + W (1.65 liters) and EU + W (0.31 liters) and not the preexercise hydration status per se, because the responses of HY + NW differed greatly from those of EU + NW. If water intake in the HY + W and EU + W trials had been identical, eliminating the possibility of studying voluntary dehydration and ad libitum drinking, these findings may have been different.

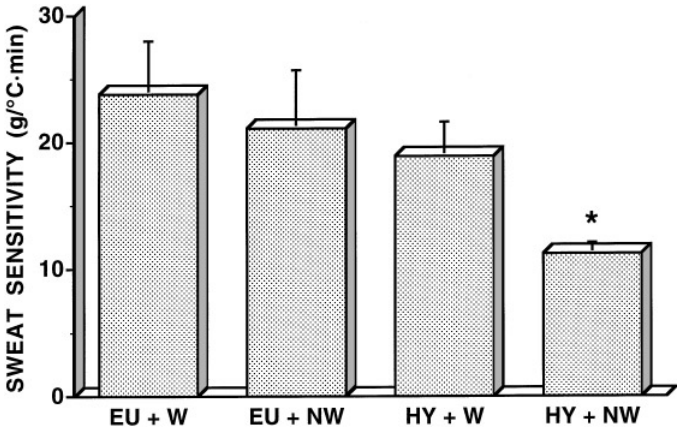

Fig. 3. Sweat sensitivity of each heat stress test normalized for area under heating curve. Values are means ± SE. * Significantly different from all other treatments, $P < 0.05$–0.01.

Effects of water intake during HY + W. The second purpose of this investigation was to determine the differential effects of initial HY and EU on ad libitum water consumption, fluid balance, and physiological responses. We hypothesized that if our subjects began exercise in an HY state with an elevated P_{osm} and an activated thirst drive, water intake would be greater and the anticipated increase in physiological strain would be attenuated during exercise-heat exposure. Clearly, ad libitum water intakes were different, and the similarities of HY + W and EU + W responses during HST (Tables 1 and 2) supported this hypothesis. We believe that the differences in the pretest P_{osm} (295 ± 7 and 287 ± 6 mosmol/kg for HY + W and EU + W, respectively) played an important role in increasing ad libitum water consumption (1.65 and 0.31 liter for HY + W and EU + W, respectively) and attenuating thermal and circulatory strain. Furthermore, the water ingested during the HY + W test resulted in no voluntary dehydration (+0.9% body mass increase). This is unique, because voluntary dehydration is considered to be a universal response during exercise-heat exposures in which subjects drink fluid ad libitum (14, 28). In addition, HY + W was similar or superior to all other treatments in the following responses: Δbody mass during HST, final HR, heat storage, final $\overline{T}_{sk}$, ΔT_{re}, and final T_{re}. It is likely that this resulted from the combined effects of preexercise hydration status and fluid intake during exercise.

Physiologists have advised athletes to consume a volume of fluid that approximates sweat loss (7, 16, 19, 23); this volume (0.96 liter) was identical for EU + W and HY + W (Fig. 4, arrows). If this volume had been consumed by our subjects, it would have resulted in a mean decrease of 1 mosmol/kg during EU + W and a mean increase of 4 mosmol/kg during HY + W. This suggests that the above advice is correct when athletes begin exercise in the euhydrated state. When athletes begin exercise in a hypohydrated state, however, they should consume water in excess of sweat loss to attenuate the detrimental influence of an increased P_{osm} on

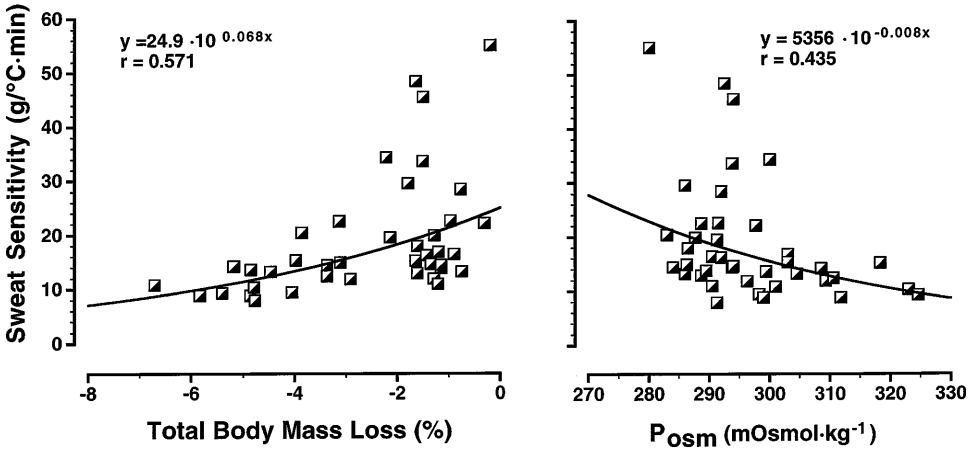

Fig. 5. Relationships between sweat sensitivity and both total body mass loss and final P_{osm}. Both exponential regression equations (n = 39) were significant at $P < 0.01$.

sweat sensitivity during prolonged exercise (6, 18, 20, 22, 29).

The present study also clarifies the influence of preexercise hydration state on the relationship between water intake and ΔT_{re} during exercise. The regression lines for the EU + W and HY + W (Fig. 2) trials have similar negative slopes, but the line representing HY + W is shifted to the right of that representing EU + W. Therefore, for a given water intake (i.e., 1.0 liter), T_{re} was higher (~0.6°C) when subjects were hypohydrated (−3.9% body mass). These findings agree closely with previously published 2-h cycle ergometry tests involving scheduled drinking (19) and indicate that 1.65 liters of additional water were required during HY + W (i.e., 5 times greater than EU + W) to produce a ΔT_{re} that was equivalent to the EU + W test.

Intrasubject drinking differences. Two subjects consumed much less water than other subjects during the HY + W trial; they alone experienced a net loss of body mass, whereas the other eight subjects had a mean body mass gain of 0.9% due to ad libitum drinking. These two men were classified as reluctant drinkers following the method of Szlyk et al. (28) and appear in Figs. 2 and 4 as the subjects with the smallest water intakes (i.e., within the range of EU + W values) and the largest ΔT_{re} and ΔP_{osm}. They also exhibited the lowest sweat sensitivities concurrent with the greatest heat storage rates, radiative heat losses, and final plasma glucose concentrations. Their elevated plasma glucose concentrations are consistent with decreased splanchnic blood flow combined with increased hepatic metabolism (i.e., Q_{10} effect), causing increased hepatic glycogenolysis and glucose release (24). Clearly, these two test subjects exhibited greater strain than others during HY + W, but it is unclear why their internal physiological state did not stimulate greater drinking. Although increased circulating catecholamines also could have increased hepatic glucose release, analyses (13) indicated that the epinephrine and norepinephrine concentrations of these two subjects were similar to the group mean values.

Recent investigations of drinking behavior have attempted to identify why reluctant drinkers respond to internal cues differently from other subjects, despite experiencing similar exercise-heat stress and fluid losses (14, 28). The prevailing theory (11, 14) involves voluntary dehydration, which originates with negative alliesthesia, an unpleasant stimulus engendered by drinking that depends on the internal status of the subject and characteristics of the fluid (i.e., odor, clarity, palatability, temperature). Interestingly, during the EU + W trial, the water intakes and physiological responses of reluctant drinkers were similar to those of the other eight subjects. This demonstrates for the first time that reluctant drinkers may not drink sparingly in all situations and supports the theory of negative alliesthesia.

Heat balance during HY + NW. The mean preexercise T_{re} for all treatments ranged from 37.1 to 37.4°C. At the end of exercise, however, the HY + NW trial resulted in a mean T_{re} that was 0.9–1.1°C greater than that in the three other conditions ($P < 0.001$). Heat production and heat dissipation were analyzed to explain this finding. Oxygen consumption values (range of means 19.9–21.2 $ml \cdot kg^{-1} \cdot min^{-1}$) indicated that there were no between-trial differences in aerobic heat production normalized for surface area (range of means 202.4–210.8 W/m^2). Although radiation was limited by the environmental conditions (i.e., 6.5 W/m^2 in 90 min), because the ambient temperature (33°C) was similar to that of $\overline{T}_{sk}$ (34.6°C), radiative and evaporative heat losses were similar among trials, with the latter accounting for 18–25 times more heat dissipation than the former. Furthermore, considering the environmental conditions during HST (33 ± 1°C, 56 ± 5% relative humidity), it is likely that convective and conductive heat losses (not measured) were similar to the radiant heat losses in Table 1; it is unlikely that they were solely responsible for the increased heat storage in the HY + NW trial (1). Although other investigators have concluded that dehydration increases heat storage during exercise because dry heat loss is diminished (12, 18, 25), that response was not observed in this investigation and may be specific to higher exercise intensities (i.e., >60% $\dot{V}O_{2max}$), where increased systemic and cutaneous vascular resistance have been shown to accompany dehydration and hyperthermia (12).

Although sweat rates were similar for all treatments, the body's potential for evaporative cooling was not

reached during HY + NW because of a reduced sweat sensitivity. Had the sweat sensitivity during HY + NW (11.2 g/°C·min) been equal to the mean of the other three trials (21.3 g/°C·min), the 1.7°C rise in T_{re} during HY + W could have been offset by the evaporation of ~600 g of additional sweat. Tables 1 and 2 suggest the factors that may have influenced this change in sweat sensitivity. For example, the HY + NW trial exhibited a significantly greater plasma volume loss than EU + W and HY + W. Although this suggests that plasma volume influenced sweat sensitivity, it has been documented that increasing or reducing plasma volume does not necessarily result in a systematic improvement or deterioration, respectively, in temperature regulation (6, 7, 18, 27). It is also unlikely that either thermal input from the skin or plasma glucose concentration altered sweat sensitivity during HY + NW, because they were similar during all treatments (Tables 1 and 2). P_{osm}, however, increased significantly during HY + NW only (Table 2) and was inversely related to sweat sensitivity ($P < 0.02$; Fig. 5, *right*). This observation agrees with previous reports (6, 18, 20, 22, 29) that increased extracellular osmolality (i.e., cell dehydration) diminishes thermal sweating in exercising humans.

Summary. Contrary to the paradigm of voluntary dehydration during exercise, the 3.9% body water deficit and elevated pretest P_{osm} resulted in a large water intake during HY + W, a 0.9% body mass increase, and attenuation of the rise in T_{re} via favorable changes in P_{osm} and sweat sensitivity. This suggests that a large osmotic load in fluid or food should be avoided during prolonged exercise-heat exposure and that water consumption guidelines should be designed to minimize the increase in P_{osm}. When subjects begin exercise in the hypohydrated state, this will be accomplished if pure water is consumed at a rate that exceeds both the sweat rate and the amount consumed when exercise is begun in a euhydrated state.

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REFERENCES

1. **Adams, W. C., R. H. Fox, A. J. Fry, and I. C. MacDonald.** Thermoregulation during marathon running in cool, moderate, and hot environments. *J. Appl. Physiol.* 38: 1030–1037, 1975.
2. **Armstrong, L. E., C. M. Maresh, J. W. Castellani, M. F. Bergeron, R. W. Kenefick, K. E. La Gasse, and D. Riebe.** Urinary indices of hydration status. *Int. J. Sport Nutr.* 4: 265–279, 1995.
3. **Astrand, P. O., and K. Rodahl.** *Textbook of Work Physiology.* New York: McGraw-Hill, 1977, p. 99.
4. **Burton, A. C.** Human calorimetry. II. The average temperature of the tissues of the body. *J. Nutr.* 9: 261–280, 1935.
5. **Costill, D. L., and E. L. Fox.** Energetics of marathon running. *Med. Sci. Sports* 1: 81–86, 1969.
6. **Coyle, E., and M. Hamilton.** Fluid replacement during exercise: effects on physiological homeostasis and performance. In: *Perspectives in Exercise Science and Sports Medicine. Fluid Homeostasis During Exercise*, edited by C. V. Gisolfi and D. R. Lamb. Indianapolis, IN: Benchmark, 1990, vol. 3, p. 281–308.
7. **Coyle, E., and S. J. Montain.** Thermal and cardiovascular responses to fluid replacement during exercise. In: *Perspectives in Exercise Science and Sports Medicine. Exercise, Heat, and Thermoregulation*, edited by C. V. Gisolfi, D. R. Lamb, and E. R. Nadel. Dubuque, IA: Brown, 1993, vol. 6, p. 179–224.
8. **Debson, R. L., and K. Sato.** The secretion of salt and water by the eccrine sweat gland. *Arch. Dermatol.* 105: 366–370, 1976.
9. **Dill, D. B., and D. L. Costill.** Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37: 247–248, 1974.
10. **DuBois, D., and E. F. DuBois.** A formula to estimate approximate surface area if height and weight are known. *Arch. Intern. Med.* 17: 863–871, 1916.
11. **Engell, D. B., O. Maller, M. N. Sawka, R. P. Francesconi, L. Drolet, and A. J. Young.** Thirst and fluid intake following graded hypohydration levels in humans. *Physiol. Behav.* 40: 229–236, 1987.
12. **Gonzalez-Alonso, J., R. Mora-Rodriguez, P. R. Below, and E. F. Coyle.** Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. *J. Appl. Physiol.* 79: 1487–1496, 1995.
13. **Hoffman, J. R., C. M. Maresh, L. E. Armstrong, C. L. Gabaree, M. F. Bergeron, R. W. Kenefick, J. W. Castellani, L. E. Ahlquist, and A. Ward.** Effects of hydration state on plasma testosterone, cortisol, and catecholamine concentrations before and during mild exercise at elevated temperature. *Eur. J. Appl. Physiol.* 69: 294–300, 1994.
14. **Hubbard, R. W., P. C. Szlyk, and L. E. Armstrong.** Influence of thirst and fluid palatability on fluid ingestion during exercise. In: *Perspectives in Exercise Science and Sports Medicine. Fluid Homeostasis During Exercise*, edited by C. V. Gisolfi and D. R. Lamb. Carmel, IN: Benchmark, 1990, vol. 3, p. 39–96.
15. **Ladell, W. S. S.** The effects of water and salt intake upon the performance of men working in hot and humid environments. *J. Physiol. (Lond.)* 127: 11–46, 1954.
16. **Mitchell, J. W., E. R. Nadel, and J. A. J. Stolwijk.** Respiratory weight losses during exercise. *J. Appl. Physiol.* 32: 474–476, 1972.
17. **Mitchell, D., C. H. Wyndham, A. J. Vermeulen, T. Hodgson, A. R. Atkins, and H. S. Hofmeyer.** Radiant and convective heat transfer of nude men in dry air. *J. Appl. Physiol.* 26: 111–118, 1969.
18. **Montain, S. J., and E. F. Coyle.** Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. *J. Appl. Physiol.* 73: 903–910, 1992.
19. **Montain, S. J., and E. F. Coyle.** The influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J. Appl. Physiol.* 73: 1340–1350, 1992.
20. **Montain, S. J., and E. F. Coyle.** Influence of timing of fluid ingestion on temperature regulation during exercise. *J. Appl. Physiol.* 75: 688–695, 1993.
21. **Nadel, E. R.** Temperature regulation and prolonged exercise. In: *Perspectives in Exercise Science and Sports Medicine. Prolonged Exercise*, edited by D. R. Lamb and R. Murray. Indianapolis, IN: Benchmark, 1988, vol. 1, p. 125–152.
22. **Nielsen, B.** Effects of changes in plasma volume and osmolality on thermoregulation during exercise. *Acta Physiol. Scand.* 90: 725–730, 1974.
23. **Pitts, G. C., R. C. Johnson, and F. C. Consolazio.** Work in the heat as affected by intake of water, salt, and glucose. *Am. J. Physiol.* 142: 253–259, 1944.
24. **Rowell, L. G., G. L. Brengelmann, J. R. Blackmon, R. D. Twiss, and F. Kusumi.** Splanchnic blood flow and metabolism in heat-stressed man. *J. Appl. Physiol.* 24: 475–484, 1968.
25. **Sawka, M. N.** Body fluid responses and hypohydration during exercise-heat stress. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, edited by K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis, IN: Benchmark, 1988, p. 227–266.
26. **Sawka, M. N., A. J. Young, R. P. Francesconi, S. R. Muza, and K. B. Pandolf.** Thermoregulatory and blood responses

during exercise at graded hypohydration levels. *J. Appl. Physiol.* 59: 1394–1401, 1985.

27. **Sherman, W. M., and D. R. Lamb.** Nutrition and prolonged exercise. In: *Perspectives in Exercise Science and Sports Medicine. Prolonged Exercise*, edited by D. R. Lamb and R. Murray. Indianapolis, IN: Benchmark, 1988, vol. 1, p. 213–280.
28. **Szlyk, P. C., I. V. Sils, R. P. Francesconi, R. W. Hubbard, and L. E. Armstrong.** Effects of water temperature and flavoring on voluntary dehydration in men. *Physiol. Behav.* 45: 639–647, 1989.
29. **Takamata, A., G. W. Mack, C. M. Gillen, A. C. Jozsi, and E. R. Nadel.** Osmoregulatory modulation of thermal sweating in humans: reflex effects of drinking. *Am. J. Physiol.* 268 (*Regulatory Integrative Comp. Physiol.* 37): R414–R422, 1995.
30. **Vokes, T.** Water homeostasis. *Annu. Rev. Nutr.* 7: 383–406, 1987.
31. **Wenger, C. B.** Human heat acclimatization. In: *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*, edited by K. B. Pandolf, M. N. Sawka, and R. R. Gonzalez. Indianapolis, IN: Benchmark, 1988, p. 153–198.