Hyperhydration: thermoregulatory effects during compensable exercise-heat stress

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Latzka, William A., Michael N. Sawka, Scott J. Montain, Gary S. Skrinar, Roger A. Fielding, Ralph P. Matott, and Kent B. Pandolf. Hyperhydration: thermoregulatory effects during compensable exercise-heat stress. J. Appl. Physiol. 83(3): 860–866, 1997.—This study examined the effects of hyperhydration on thermoregulatory responses during compensable exercise-heat stress. The general approach was to determine whether 1-h preexercise hyperhydration [29.1 ml/kg lean body mass; with or without glycerol (1.2 g/kg lean body mass)] would improve sweating responses and reduce core temperature during exercise. During these experiments, the evaporative heat loss required (Erreq) to maintain steady-state core temperature was less than the maximal capacity (Emax) of the climate for evaporative heat loss (Erreq/Emax = 63%). Eight heat-acclimated men completed five trials: euhydration, glycerol hyperhydration, and water hyperhydration both with and without rehydration (replace sweat loss during exercise). During exercise in the heat (35°C, 45% relative humidity), there was no difference between hyperhydration methods for increasing total body water (−1.5 liters). Compared with euhydration, hyperhydration did not alter core temperature, skin temperature, whole body sweating rate, local sweating rate, sweating threshold temperature, sweating sensitivity, or heart rate responses. Similarly, no difference was found between water and glycerol hyperhydration for these physiological responses. These data demonstrate that hyperhydration provides no thermoregulatory advantage over the maintenance of euhydration during compensable exercise-heat stress.

Recent studies have focused on the use of glycerol solutions to achieve hyperhydration (5, 14, 16, 23). They found that subjects drinking glycerol solutions achieved greater hyperhydration compared with subjects drinking water while resting in temperate conditions. Whether glycerol solutions sustain greater hyperhydration than tap water during exercise-heat stress is not known. Freund et al. (5) recently reported that glycerol increases fluid retention by reducing free water clearance. Exercise and heat stress decrease renal blood flow and free water clearance and therefore may reduce the effectiveness of glycerol as a hyperhydrating agent relative to water.

Lyons and colleagues (14) reported that glycerol/water hyperhydration had dramatic effects on improving a person’s ability to thermoregulate during exercise-heat stress. They found that the rectal temperature (Tr) rise was attenuated by 0.7°C and that sweating rate was elevated by ≈ 300 to 400 ml/h above control levels. These thermoregulatory benefits during exercise-heat stress have not been confirmed. Others have reported similar core temperatures and sweating rates between glycerol and water hyperhydration fluids before exercise (16) in a temperate climate or as rehydration solutions during exercise in a warm climate (18). No study has evaluated the effects of hyperhydration on thermoregulation during exercise-heat stress.

The purpose of this study was to determine the efficacy of hyperhydration for improving thermoregulation during compensable exercise-heat stress. Glycerol hyperhydration was compared with water hyperhydration and euhydration under conditions of maintained hydration and progressive dehydration. We hypothesized that hyperhydration would enhance thermoregulatory responses (lower core temperature, improve sweating) above those when subjects are euhydrated and that glycerol hyperhydration would be more effective than water hyperhydration.

MATERIALS AND METHODS

Subjects and preliminary measurements. All subjects received a physical examination, including a medical history, before testing. Subjects were fully informed of all aspects of the study and signed a statement of informed consent approved by the Human Use Review Committee. Nine male subjects participated in this study, and eight completed all trials. The subjects’ average age was 23 ± 6 yr (range 19–36 yr), body mass was 76 ± 15 kg (range 56–100 kg), lean body mass (LBM) was 63 ± 9 kg (range 53–73 kg), maximal oxygen uptake (Vo2max) was 56 ± 8 ml·kg−1·min−1 (range 42–69 ml·kg−1·min−1), and TBW was 46.4 ± 6.4 liters (range 38–54 liters).

HYPERHYDRATION, increased total body water (TBW), has been suggested to improve thermoregulation during exercise in the heat above euhydration levels (26). Studies examining the thermoregulatory effects of hyperhydration during exercise-heat stress have reported disparate results (26). Several studies reported that hyperhydration can reduce thermal strain (11, 14, 17, 21), whereas other studies have reported no thermoregulatory advantage (1, 10, 19). We believe that these conflicting results are because of differences in experimental design and not hyperhydration per se. For example, studies (14, 17, 21) reporting that hyperhydration reduces thermal strain have not had subjects fully replace fluid lost during exercise; therefore, the differences reported may be due to dehydration causing increased thermal strain during “control” conditions. Maintaining euhydration during exercise is essential to determine the efficacy of hyperhydration on thermoregulation during exercise-heat stress.
Preliminary tests included measurements of \( V_\text{O}_2 \text{max} \), submaximal workload determination, body composition, and TBW. In addition, nude body mass was measured for 2 wk in the morning after the subject voided and before breakfast. These body masses were used to establish baseline body weights that represent euhydration. Five exercise-heat stress tests (HSTs) were administered in random order. The subjects wore shorts, athletic shoes, and socks.

Percent body fat, \( V_\text{O}_2 \text{max} \), submaximal exercise intensity, and TBW were measured before the HSTs. Body density was measured by hydrostatic weighing, and residual lung volume was measured while subjects were underwater. Percent body fat and LBM were calculated from body density by using the equation of Siri. Body surface area was calculated by using the DuBois formula. \( V_\text{O}_2 \text{max} \) was determined from a progressive-intensity and continuous-effort treadmill protocol (27). The initial treadmill grade was set at zero and increased 2.5% grade every 1.5 min. Treadmill velocity (2.68 or 3.13 m/s) was determined from the heart rate response at the end of a 10-min warm-up walk (1.56 m/s at a 10% grade). If heart rate was >145 beats/min, the velocity was set at 2.68 m/s; if heart rate was <145 beats/min, velocity was set at 3.13 m/s for the \( V_\text{O}_2 \text{max} \) test. The HST submaximal exercise intensity (\( \sim 45\% \) \( V_\text{O}_2 \text{max} \)) was determined in a temperate climate (22–24°C dry bulb; \( \sim 25–29\% \) relative humidity).

Subjects were heat acclimated by walking at \( \sim 45\% \) \( V_\text{O}_2 \text{max} \) on a treadmill for two 50-min bouts spaced by a 10-min rest period for 6–10 days in a hot dry climate (ambient temperature = 35°C, relative humidity = 45%, air velocity = 1 m/s). Heat acclimation was established when nonsignificant differences were observed in final exercise core temperatures and heart rates on 2 consecutive days of acclimation. During rest and exercise, the subjects were encouraged to drink either cool water or a commercial electrolyte beverage. Subjects discontinued exercise if \( T_\text{es} \) reached 39.5°C or heart rate achieved 90% of maximum for a 5-min period. Body masses (nude and clothed) were obtained before and after exercise. Subjects were asked to participate in additional heat-acclimation sessions on nonrest days if they had greater than 2 consecutive days without an exercise-heat exposure. TBW was measured by using the deuterium-labeled water-dilution technique (5) in the final week of acclimation. This measurement was performed the morning after 8 h of abstinence from food and drink by the subjects and with the subjects seated. The TBW measurement was used to calculate the change in TBW during the HSTs. TBW values were assumed to be the same before HSTs as on TBW-measurement days because hydration procedures and body weights were also similar. Change in TBW was calculated from the change in body mass and adjusted for fluid volume and urine volume during HSTs.

Hydration procedures. Hyperhydration began immediately after venous catheterization and plasma osmolality measurement. The euhydration-condition criteria required an initial plasma osmolality of <286 mosmol/kgH_2O. In the hydration trials, subjects first drank 3.9 ml/kg LBM of the experimental solution (i.e., either glycerol solution or water). The experimental solution administration was double blind; the water and glycerol solutions were of similar sweetness (Aspartame), color, flavor, and temperature (10°C) to mask the taste of glycerol. The glycerol solution contained 1.2 g glycerol/kg LBM and was of a purity for human consumption. After ingestion of the experimental solution, the subject drank a large volume (25.2 ml/kg LBM) of water (36°C). The total volume of fluid consumed in a 30-min period was 29.1 ml/kg LBM. This hyperhydration method is identical to that previously reported (5).

Exercise-HSTs. Exercise-HSTs consisted of subjects attempting 120 min of treadmill exercise (1.56–1.65 m/s at 4–9% grade = 45% of \( V_\text{O}_2 \text{max} \)) in the heat (ambient temperature = 34.9 ± 0.1°C, dew-point temperature = 25.9 ± 0.6°C, air velocity = 1 m/s). For each subject, HSTs were conducted at the same time of day. The required evaporative heat loss (\( E_{\text{req}} \) 253 ± 17 W/m²) was less than the maximal capacity of the climate for evaporative heat loss (\( E_{\text{max}} \) 462 ± 89 W/m²) and therefore was compensable exercise-heat stress (7).

Five exercise-HSTs were attempted: euhydration (Eu), glycerol hyperhydration with no replacement (GD), glycerol hyperhydration/rehydration (GR), water hyperhydration with no replacement (WD), and water hyperhydration/rehydration (WR). The rehydration fluid was water (−36°C) given in equal volumes at ~20, 40, 60, 80, and 100 min of exercise. Rehydration fluid was given to replace fluid lost during HSTs, but the volume was determined during the last day of heat acclimation for each subject. The day before the HSTs, each subject was instructed not to eat for 8 h before the report time; he was weighed and instructed to drink 2 liters of a provided commercial carbohydrate electrolyte beverage by 2200 the evening before the HST. The next morning the subject reported to the laboratory, and a nude body mass measurement was taken after the subject voided.

A flexible Teflon catheter was inserted in a superficial arm vein, and plasma osmolality was measured to ascertain euhydration status. The subject swallowed an esophageal probe and inserted the rectal probe for core temperature measurements. Subjects entered the climatic chamber 30 min before exercise and were instrumented (temperature probes, electrocardiograph leads). Clothed body masses were measured before exercise. Body temperatures and heart rates were continuously monitored, and metabolic rates were measured at ~5, 55, and 100 min of exercise. After the exercise, body masses (clothed and nude) were again measured.

Skin temperatures were measured at five sites (forearm, upper arm, chest, thigh, and calf) by using a thermocouple skin harness, and mean skin temperature (\( T_{\text{es}} \)) was calculated (22). Esophageal temperature (\( T_{\text{es}} \)) was measured from a thermocouple placed at the level of the heart. Because swallowed saliva lowers \( T_{\text{es}} \) measurements, the subject was asked to swallow the mucus saliva that was swallowed during HSTs. The \( T_{\text{es}} \) measurements were recorded immediately before each rehydration period during the HSTs. \( T_{\text{es}} \) was measured from a thermistor inserted 10 cm beyond the anal sphincter. Local sweating rate (\( m_{\text{sw}} \) ) of the upper arm was measured by automated dew-point sensors encased in a ventilated capsule (8), and \( m_{\text{sw}} \) was calculated. Sweating sensitivity was the slope of the regression line when \( m_{\text{sw}} \) was plotted as function of \( T_{\text{es}} \) during the first 20 min of exercise. The threshold for active thermoregulatory sweating was the \( T_{\text{es}} \) when \( m_{\text{sw}} \) exceeded 0.06 mg·cm⁻²·min⁻¹ (23). Total body sweating rate was calculated from pre- and postexercise masses and was corrected for water intake and urine output.

Venous blood samples (10 ml) were taken after subjects were standing for ~25 min in the climatic chamber before exercise, and three samples were taken during exercise (at ~40 and 80 min and a final exercise sample). Venous blood samples were collected from an indwelling Teflon catheter placed within a superficial arm vein. Patency was maintained with heparinized saline; the catheter was flushed with ~2 ml of blood before each 8-ml sample was obtained. Blood samples were measured for hemoglobin, hematocrit, lactate, sodium, potassium, osmolality, glycerol, and protein. Percent change in plasma volume and blood volume was calculated from the appropriate hemoglobin and hematocrit values. Serum was analyzed for sodium and potassium by using a flame photom-
Exercise-heat stress. For Eu, GD, GR, WD, and WR trials, predrink body masses were 76.5 ± 4.6, 76.4 ± 4.7, 76.1 ± 14.6, 76.1 ± 14.6, and 76.1 ± 14.4 kg, respectively (P > 0.05). Predrink plasma osmolalities for Eu, GD, GR, WD, and WR trials were 282 ± 3, 283 ± 5, 284 ± 5, 284 ± 5, and 284 ± 5 mosmol/kgH₂O, respectively (P > 0.05).

Eight subjects completed all trials in this study. A ninth subject was unable to ingest the glycerol solution without becoming nauseated; this subject was removed from the study. Two of the eight subjects on one occasion vomited after drinking the glycerol solution, so the trial was aborted and repeated on another day. One of the eight subjects was unable to complete the 120-min exercise period in two of the five trials. For a given subject, the same amount of fluid (1.84 ± 0.25 liters) was consumed before each hyperhydration trial. The volume of water consumed during exercise was 2.36 ± 0.51, 2.23 ± 0.72, and 2.19 ± 0.52 liters during Eu, GR, and WR trials, respectively. These values were not different (P > 0.05), and the between-trial variability was because of differences in exercise duration or subject drinking tolerance.

Figure 1 presents the change in TBW and the change in plasma volume responses for each trial. For Eu trials, TBW did not change over time. For hyperhydration trials GD, GR, WD, and WR, TBW increased (P < 0.05) by 1.40 ± 0.39, 1.38 ± 0.33, 1.50 ± 0.40, and 1.54 ± 0.31 liters, respectively, in subjects 30 min after drinking, with no difference (P > 0.05) among trials. There were no differences (P > 0.05) in TBW between the GR and WR trials or between the GD and WD trials during exercise. There were no differences in plasma volume among Eu, GD, GR, WD, and WR trials. There were significant (P < 0.05) decreases in plasma volumes from preexercise to the final exercise value in both the GD and GR trials.

Total urinary output values were greater (P < 0.05) in hyperhydration (GD, GR, WD, and WR) than in the Eu trials. The total urinary outputs for the Eu, GD, GR, WD, and WR trials were 0.15 ± 0.18, 0.52 ± 0.38, 0.61 ± 0.19, 0.71 ± 0.34, and 0.70 ± 0.25 liter, respectively. No differences (P > 0.05) in total urinary output were observed between glycerol hyperhydration trials and water hyperhydration trials.

Table 1 presents serum osmolality and glycerol values during each trial. Preexercise serum osmolality was greater (P < 0.05) in GD and GR than Eu trials. The mean total osmolar load from the ingested glycerol was 802 mosmol or 16.6 mosmol/l TBW. Preexercise serum osmolality values were lower (P < 0.05) during WD and WR than Eu trials. During exercise, serum osmolality increased (P < 0.05) in GD and WD trials but did not change (P > 0.05) during exercise in other trials. Serum glycerol levels were greater (P < 0.05) in GD and GR than in Eu, WD, and WR trials. During exercise, serum glycerol did not change (P > 0.05) in Eu, WD, and WR trials; however, serum glycerol decreased (P < 0.05) during exercise in the GD and GR trials by 39 and 31 mg/dl, respectively.

Table 2 presents serum values for sodium, potassium, and lactate during each trial. Preexercise and during exercise, serum lactate and serum potassium
Tsk and mean body temperature

Metabolic rate and heart rate. Metabolic rate increased (P < 0.05) over time during exercise but was not different (P > 0.05) anytime among trials. The average metabolic rates were 337 ± 48, 342 ± 43, 338 ± 48, 341 ± 45, and 346 ± 51 W/m² for Eu, GD, GR, WD, and WR trials, respectively, which corresponded to relative-intensity oxygen uptake levels 44–45% of VO₂max. Figure 2 presents the heart rate responses during exercise-heat stress for each trial. Heart rate was not different (P > 0.05) at rest among trials and increased (P < 0.05) over time during exercise. Final exercise heart rate values were greater (P < 0.05) in GD and WD trials (158 ± 9 and 161 ± 15 beats/min, respectively) than in GR and WR trials (149 ± 13 and 148 ± 17 beats/min, respectively). Final exercise heart rates were similar (P > 0.05) in GR, WR, and Eu trials (149 ± 13, 148 ± 17, and 150 ± 14 beats/min, respectively).

Body temperature. Figure 3 presents Tₑₚ and Tₑₛ responses for each trial. Tₑₚ values were not different (P > 0.05) among trials either preexercise or at anytime during exercise, with values increasing (P < 0.05) over time during exercise. Final exercise Tₑₚ values for Eu, GD, GR, WD, and WR trials were 38.6 ± 0.4, 38.8 ± 0.2, 38.5 ± 0.3, 38.7 ± 0.4, and 38.6 ± 0.4°C, respectively. Preexercise Tₑₛ values were not different (P > 0.05) among trials. Final exercise Tₑₛ values were greater (P < 0.05) during GD and WD (38.3 ± 0.2 and 38.2 ± 0.2°C, respectively) than during Eu (38.0 ± 0.2°C) trials; similar (P > 0.05) final values (38.1 ± 0.2, 38.0 ± 0.1, and 38.0 ± 0.2°C) were found during Eu, GR, and WR trials, respectively.

Figure 4 presents Tₛₖ and mean body temperature (T_b) responses for each trial. Tₛₖ values were similar (P > 0.05) across trials before and during exercise. Final exercise Tₛₖ values for Eu, GD, GR, WD, and WR trials were 35.4 ± 0.9, 35.6 ± 1.0, 35.3 ± 1.3, 35.4 ± 1.0, and 35.5 ± 0.9°C, respectively. The Tₑₚ-Tₛₖ gradients increased (P < 0.05) during exercise, but values were similar (P > 0.05) among trials. Tₛₖ values were not different (P > 0.05) among trials either at preexercise or anytime during exercise, and temperatures increased (P < 0.05) during exercise. Final exercise T_b
values were 38.2 ± 0.1, 38.5 ± 0.1, 38.1 ± 0.1, 38.4 ± 0.1, and 38.3 ± 0.1°C, for Eu, GD, GR, WD, and WR trials, respectively.

Sweating response. Neither whole body sweating rates nor $m_{sw}$ values were different ($P > 0.05$) among trials. The whole body sweating rates were 529 ± 50, 497 ± 48, 520 ± 67, 490 ± 49, and 524 ± 52 g·m⁻²·h⁻¹ for Eu, GD, GR, WD, and WR trials, respectively. Final $m_{sw}$ values were 1.09 ± 0.20, 1.08 ± 0.23, 1.00 ± 0.28, 0.99 ± 0.29, and 1.05 ± 0.28 mg·cm⁻²·min⁻¹ for Eu, GD, GR, WD, and WR trials, respectively. Sweating threshold temperatures were 36.7 ± 0.2, 36.5 ± 0.4, 36.5 ± 0.1, 36.5 ± 0.3, and 36.5 ± 0.4°C, for Eu, GD, GR, WD, and WR trials, respectively ($P > 0.05$). Sweating sensitivity values were 1.01 ± 0.30, 1.03 ± 0.60, 1.03 ± 0.41, 0.98 ± 0.42, and 0.96 ± 0.53 mg·cm⁻²·min⁻¹·°C⁻¹ for Eu, GD, GR, WD, and WR trials, respectively ($P > 0.05$).

DISCUSSION

This study examined the efficacy of two hyperhydration approaches during compensable exercise-heat stress. A time schedule was used that initiated exercise-heat stress when TBW increases were expected to be near their greatest for both glycerol and water hyperhydration approaches (5). In addition, the exercise-heat stress continued through the period (~90 min) when fluid-retention differences between hyperhydration approaches were expected to be maximal (5). Therefore, the design should have been able to discriminate any initial and prolonged hydration advantages between glycerol and water hyperhydration during exercise-heat stress. A design emphasis in this study was to ensure that “baseline” hydration conditions were maintained during exercise-heat stress. Previous studies (6, 11, 14, 21) reporting core temperature advantages from hyperhydration have suffered from confounded baseline conditions, in which subjects may have started exercise hypohydrated, dehydrated during exercise, or started exercise with a lower core temperature caused by the cold drink. For this study, the baseline condition was maintained euhydration during exercise, and fluids were given at body temperature.

This study demonstrates that hyperhydration provides no thermoregulatory advantage compared with euhydration during compensable exercise-heat stress. Compared with euhydration, hyperhydration did not modify $T_{res}$, $T_{es}$, $T_{sk}$, $m_{sw}$, whole body sweating rate, or heart rate responses. In addition, glycerol hyperhydration provided no thermoregulatory advantage com-

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Fig. 3. Rectal (A) and esophageal temperatures (B) during exercise-heat stress trials. Values are means ± SE; $n = 8$ subjects. Symbols are defined as in Fig. 1. *GD temperatures are greater than those for GR and Eu, $P < 0.05$. **GD and WD values greater than those for GR, WR and Eu, $P < 0.05$.

Fig. 4. Mean skin (A) and mean body temperatures (B) during exercise-heat stress trials. Symbols are defined as in Fig. 1. Values are means ± SE; $n = 8$ subjects.
pared with water hyperhydration because responses were essentially identical for both sets of trials. These findings support our notion that previous studies demonstrating thermoregulatory advantages with hyperhydration may have simply shown the adverse effects of hyponhydration or had results systematically confounded from inadequate experimental designs (e.g., treatment-order effect causing heat acclimation; temperature of hyperhydrating fluid).

Our results agree with those of Montner et al. (16), who reported no difference in core temperature between glycerol and water hyperhydration trials. The recent interest in glycerol hyperhydration to improve exercise-heat performance originated from the study of Lyons et al. (14). They reported that glycerol hyperhydration induced remarkable core temperature (~0.7°C) reductions and sweating rate (~0.3 to 0.4 l/h) increases, with no effect on heart rate during compensable heat stress. Lyons et al. also reported that water hyperhydration provided no physiological advantage compared with their control trial. The primary difference among our study and the studies of Montner et al. (16) and Lyons et al. (14) is the subject population. Lyons et al. used unfit unacclimated subjects, whereas the present study and the study of Montner et al. (16) used fit acclimatized subjects. Both training and heat acclimation will expand plasma volume. In untrained men, acute plasma volume expansion has been observed (13) to increase stroke volume during upright exercise but not in endurance-trained athletes, who are naturally plasma volume expanded. Acute expansion of plasma volume does not influence sweating rate (3, 25) and has minimal effects on core temperature (3, 9, 25). Both glycerol and water hyperhydration increase plasma volume by similar amounts while subjects are at rest (5) and do not increase plasma volume during exercise-HSTs. Because there is no difference in plasma volume expansion between glycerol and water hyperhydration, it is unlikely that a difference in plasma volume expansion would account for differences observed among the studies. Montner et al. (16) used a time line and exercise intensity similar to those of Lyons et al. (14) but reported no differences in T_w or whole body sweating rates between glycerol and water hyperhydration trials in temperate conditions. The present study used essentially the same glycerol dosage as those investigators and found no thermoregulatory advantages in the heat.

Our study is the first to examine the effects of hyperhydration on thermoregulatory control of sweating. The sweating threshold and sensitivity values reported in this study are similar to those reported for euhydrated, heat-acclimated subjects with the use of identical methodology (15, 24). Research has demonstrated that changes in blood volume and tonicity can alter the control of sweating (4, 12, 24, 25). In this study, hyperhydration did not alter blood (plasma) volume, but water hyperhydration decreased serum osmolarity and glycerol hyperhydration increased serum osmolarity, almost entirely because of elevated serum glycerol concentration. Because glycerol should penetrate osmosensitive cells, any osmotic increase from glycerol would not be expected to alter thermoregulatory control. Several studies (12, 20, 28) have demonstrated that plasma sodium concentration influences thermoregulatory responses; we observed no difference in serum sodium concentration before exercise. However, in the trials that subjects finished exercise hyponhydrated, serum sodium levels and T_w were greater at the end of the HSTs compared with the trials with rehydration.

One advantage of hyperhydration is that it delays the development of a body water deficit when sweat losses are not replaced. Figure 1 illustrates that preexercise hyperhydration delayed the development of a water deficit until ~60 min of exercise. As expected, when hypohydration was present, physiological strain (T_w, heart rate) increased. Therefore, preexercise hyperhydration can be beneficial when fluid intake is restricted during subsequent exercise. Preexercise hyperhydration can delay the onset of hyponhydration and the physiological strain associated with hypohydration.

We found that both methods of hyperhydration were equally effective for increasing TBW. TBW was increased an average of 1.45 liters 30 min after subjects drank the glycerol solution or water. TBW remained elevated in the rehydration trials throughout exercise but approached euhydration by 60 min of exercise, when fluid replacement was withheld. This is in contrast to studies that demonstrated the beneficial effects of glycerol hyperhydration in resting volunteers (5, 14, 23). From 30 to 150 min after drinking, our subjects exercised in a hot climate, two conditions that decrease renal blood flow (2), glomerular filtration (2), and free water clearance (29). Urine flow decreased ~70% (5.4 ± 3.6 to 1.7 ± 1.2 ml/min) from rest to exercise and was not different among hyperhydration trials. Therefore, the similar fluid retention between glycerol and water trials was likely due to effectiveness of exercise and heat exposure for reducing diuresis.

In summary, we observed that 1) during compensable exercise-heat stress, thermoregulatory responses were identical regardless of whether subjects were euhydrated, water hyperhydrated, or glycerol hyperhydrated; 2) glycerol hyperhydration provided no hydraulic advantage over water hyperhydration during exercise-heat stress because both hyperhydration approaches increased TBW by similar amounts; and 3) hyperhydration delayed the development of body water deficits if fluids were not replaced during exercise-heat stress. We conclude that hyperhydration provides no meaningful advantages over the maintenance of euhydration during compensable exercise-heat stress.

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