

Fluid and electrolyte supplementation for exercise heat stress¹⁻⁴

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ABSTRACT During exercise in the heat, sweat output often exceeds water intake, resulting in a body water deficit (hypohydration) and electrolyte losses. Because daily water losses can be substantial, persons need to emphasize drinking during exercise as well as at meals. For persons consuming a normal diet, electrolyte supplementation is not warranted except perhaps during the first few days of heat exposure. Aerobic exercise is likely to be adversely affected by heat stress and hypohydration; the warmer the climate the greater the potential for performance decrements. Hypohydration increases heat storage and reduces a person's ability to tolerate heat strain. The increased heat storage is mediated by a lower sweating rate (evaporative heat loss) and reduced skin blood flow (dry heat loss) for a given core temperature. Heat-acclimated persons need to pay particular attention to fluid replacement because heat acclimation increases sweat losses, and hypohydration negates the thermoregulatory advantages conferred by acclimation. It has been suggested that hyperhydration (increased total body water) may reduce physiologic strain during exercise heat stress, but data supporting that notion are not robust. Research is recommended for 3 populations with fluid and electrolyte balance problems: older adults, cystic fibrosis patients, and persons with spinal cord injuries. *Am J Clin Nutr* 2000;72(suppl):564S-72S.

KEY WORDS Skin blood flow, cystic fibrosis, dehydration, fluid redistribution, hypohydration, hyperhydration, older adults, spinal cord injury, sweating, thermoregulation

INTRODUCTION

Water and electrolyte balance are critical for the function of all organs and, indeed, for maintaining health in general (1, 2). Water provides the medium for biochemical reactions within cell tissues and is essential for maintaining an adequate blood volume and thus the integrity of the cardiovascular system. The ability of the body to redistribute water within its fluid compartments provides a reservoir to minimize the effects of water deficit. Each body water compartment contains electrolytes, the concentration and composition of which are critical for moving fluid between intracellular and extracellular compartments and for maintaining membrane electrochemical potentials.

Physical exercise and heat stress cause both fluid and electrolyte imbalances that need to be corrected (3-6). Generally, persons dehydrate during exercise in the heat because of the unavailability of fluids or a mismatch between thirst and water requirements (7, 8). In these instances, the person is euhydrated (normally hydrated)

at the beginning of exercise but incurs hypohydration (a body water deficit) over a prolonged period. Hypohydrated persons who exercise in the heat will incur significant adverse effects (9). Hypohydration increases physiologic strain, decreases exercise performance, and negates the thermoregulatory advantages conferred by high aerobic fitness (10, 11) and heat acclimation (10, 12). If strenuous exercise is performed by hypohydrated persons, the medical consequences can be devastating (13, 14).

We review human fluid and electrolyte balance relative to their effects on temperature regulation and exercise performance in the heat. In addition, needs for research on fluids and electrolytes will be discussed for 3 special populations: cystic fibrosis patients, older persons, and persons with spinal cord injuries.

FLUID AND ELECTROLYTE BALANCE

To support the contraction of skeletal muscles, physical exercise routinely increases total body metabolism to 5-15 times the resting rate. Approximately 70-90% of this energy is released as heat, which needs to be dissipated to achieve body heat balance. The relative contributions of evaporative and dry (radiative and conductive) heat exchange to total heat loss vary according to climatic conditions (15). In hot climates, a substantial volume of body water may be lost via sweating to enable evaporative cooling (7).

In addition to climatic conditions, clothing and exercise intensity influence the sweating rate (15, 16). Residents of desert climates often have sweating rates of 0.3-1.2 L/h while performing occupational activities (17). Clothing may be a major concern; persons wearing protective garments often have sweating rates of 1-2 L/h while performing light-intensity exercise (18). For athletes performing high-intensity exercise in the heat, sweating rates of 1.0-2.5 L/h are common. Expected sweating rates from running in different climatic conditions are shown in **Figure 1** (7). The influence of climate and amount of physical activity on

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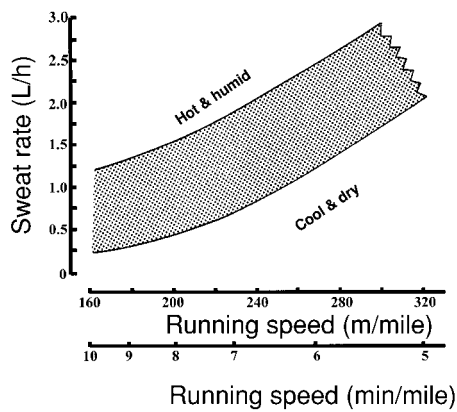


FIGURE 1. An approximation of hourly sweating rates as a function of climate and running speed (7).

daily fluid requirements is shown in **Figure 2** (19, 20). Daily requirements for sedentary to very active persons range from 2–4 L/d in temperate climates and from 4–10 L/d in hot climates.

Sodium chloride is the primary electrolyte in sweat, with potassium, calcium, and magnesium present in smaller amounts. The sodium concentration in sweat averages 35 mmol/L (range: 10–70 mmol/L) and varies by diet, sweating rate, hydration, and degree of heat acclimation (21, 22). Sweat glands reabsorb sodium by active transport, but the ability to reabsorb sweat sodium does not increase with the sweating rate; thus, at high sweating rates the concentration of sodium increases (15). Because heat acclimation improves the ability to reabsorb sodium, acclimated persons have lower sodium concentrations in sweat (>50% reduction) for any specific sweating rate (22). The potassium concentration in sweat averages 5 mmol/L (range: 3–15 mmol/L); that of calcium, 1 mmol/L (range 0.3–2 mmol/L); and that of magnesium, 0.8 mmol/L (range 0.2–1.5 mmol/L) (21). Sex, maturation, and aging do not appear to affect sweat electrolyte concentrations markedly (23, 24). Except for the first several days of heat exposure, electrolyte supplementation is not necessary because normal dietary sodium intake will cover the sweat losses (3, 5). Although sweat contains vitamins, their concentrations are low; thus, vitamin supplementation is not needed (25).

During exercise in the heat, hypohydration must be avoided by matching fluid consumption with sweat loss. This is difficult because thirst is not a good indicator of body water requirements (17, 26, 27). In general, thirst is not perceived until a person has incurred a water deficit of $\approx 2\%$ body weight loss (BWL) (17, 26, 28). Correspondingly, ad libitum water intake during exercise in the heat results in an incomplete replacement of body water losses (**Figure 3**; 17). As a result, unless forced hydration is stressed, some dehydration is likely to occur during exercise in the heat. Humans will usually fully rehydrate at meals, when taking fluids is stimulated by consuming food (4, 17). Thus, active persons need to stress drinking at meals to avoid persistent hypohydration.

Hypohydration reduces the gastric emptying rate of ingested fluids during exercise in the heat (29, 30). Neuffer et al (29), for example, found a reduction of $\approx 20\text{--}25\%$ in the gastric emptying rate when their subjects were hypohydrated (5% body weight) that was related to increased core temperature. Thus, beginning fluid intake during the early stages of exercise heat stress is

important not only to minimize hypohydration but also to maximize the bioavailability of the ingested fluids.

During situations of stress and prolonged high sweat loss, adults will dehydrate by 2–8% BWL. Water constitutes 45–70% of body weight (1); the average male (75 kg) has ≈ 45 L of water (about 60% of body weight). Because adipose tissue is $\approx 10\%$ water but muscle tissue is $\approx 75\%$ water, a person's total body water varies by body composition (1). In addition, muscle water and glycogen content parallel each other, probably because of the osmotic pressure exerted by glycogen granules within the muscle's sarcoplasm (31). As a result, trained athletes have relatively greater total body water than their sedentary counterparts by virtue of a smaller percentage body fat and a higher concentration of skeletal muscle glycogen.

The water contained in body tissues is distributed between the intracellular and extracellular fluid spaces. Because there is free fluid exchange, hypohydration mediated by sweating will influence each fluid space. Nose et al (32) determined the distribution in the rat of body water loss among the fluid spaces as well as among different body organs; they thermally dehydrated rats by 10% BWL and after the animals regained their normal core temperature they measured the animals' body water. Forty-one percent of the water deficit was intracellular and 59% was extracellular. In terms of organs, 40% of the water deficit was from muscle, 30% from skin, 14% from viscera, and 14% from bone. Neither the brain nor liver lost significant water. These researchers concluded that hypohydration redistributes water largely from the intracellular and extracellular spaces of muscle and skin as a way of maintaining blood volume.

Resting plasma volume and osmolality values for heat-acclimated persons hypohydrated to various degrees are shown in **Figure 4** (33). Sweat-induced hypohydration will decrease plasma volume and increase plasma osmotic pressure in proportion to the amount of fluid loss. Plasma volume decreases because it provides the precursor fluid for sweat, and osmolality increases because sweat is ordinarily hypotonic relative to plasma. Sodium and chloride are primarily responsible for the elevated plasma osmolality (34, 35), which mobilizes fluid from the intracellular to the extracellular space to enable defense of plasma volume in hypohydrated persons. This concept is illustrated by heat-acclimated persons, who have a smaller reduction in plasma volume than do unacclimated persons for a given body water deficit (36). By virtue of having more dilute sweat, heat-acclimated persons have additional solutes remaining within the extracellular space to exert an osmotic pressure and redistribute fluid from the intracellular space.

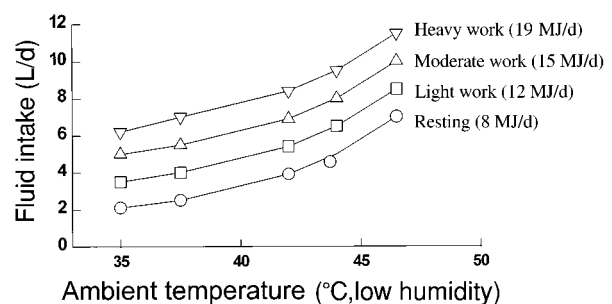


FIGURE 2. Influence of climatic temperature and daily metabolic rate on daily fluid requirements (19, 20).



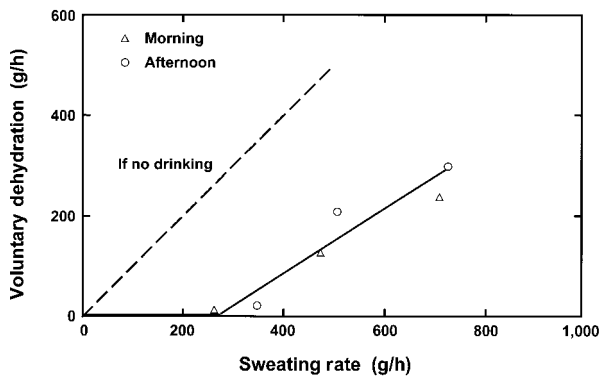


FIGURE 3. Relation between sweating rate and voluntary dehydration (water deficit) during ad libitum drinking by heat-acclimated persons in the desert (17).

Persons who use diuretics for medical purposes or to reduce their body weight are at increased risk of hypohydration because diuretics increase urine formation and generally result in the loss of solutes. Commonly used diuretics include thiazides (eg, Diuril; Merck & Co, Inc, West Point, PA), carbonic anhydrase inhibitors (eg, Diamox Parenteral; Lederle Laboratories, Philadelphia), and furosemide (eg, Lasix; Hoechst-Roussel, Somerville, NJ). Diuretic-induced hypohydration generally results in an isoosmotic hypovolemia, with a much greater ratio of plasma loss to body water loss than in either exercise- or heat-induced hypohydration. Relatively less intracellular fluid is lost after diuretic administration because there is no extracellular solute excess to stimulate redistribution of body water.

EXERCISE PERFORMANCE

Numerous studies have examined the influence of hypohydration on maximal aerobic power and physical work capacity (33). In temperate climates, maximal aerobic power reportedly decreases when hypohydration reaches or exceeds 3% BWL (10, 37, 38). In hot climates, however, water deficits of 2–4% BWL were reported by Craig and Cummings (39) to cause a large reduction in maximal aerobic power. Data from this study indicate a disproportionately larger decrease in maximal aerobic power with an increased magnitude of body water deficit.

The physical work capacity for aerobic exercise of progressive intensity is decreased when a person is hypohydrated (33). Physical work capacity has been shown to be decreased by marginal (1–2% BWL) water deficits that do not alter maximal aerobic power (37, 40), and the reduction is larger with increasing water deficits. Hypohydration resulted in much larger decrements of physical work capacity in hot than in temperate climates. It appears that the thermoregulatory system, perhaps via increased body temperatures, plays an important role in the reduced exercise performance mediated by a body water deficit.

A reduced maximal cardiac output might be the physiologic mechanism by which hypohydration decreases a person's maximal aerobic power and physical work capacity for progressive-intensity exercise. Hypohydration is associated with decreased blood (plasma) volume during both rest and exercise (41, 42); decreased blood volume increases blood viscosity and can reduce venous return. During maximal exercise, viscosity-mediated increased resistance and reduced cardiac filling could decrease both stroke

volume and cardiac output. Several investigators reported a tendency for reduced cardiac output when subjects are hypohydrated during short-term, moderate-intensity exercise (43–45).

It is not surprising that climatic heat stress potentiates the hypohydration-mediated reduction in maximal aerobic power and physical work capacity for progressive-intensity exercise. For euhydrated persons, climatic heat stress alone decreases maximal aerobic power by $\approx 7\%$ (46). In the heat, the superficial skin veins reflexively dilate to increase cutaneous blood flow and volume. The redirection of blood flow to the cutaneous vasculature could decrease maximal aerobic power by 1) reducing the portion of cardiac output that perfuses contracting muscles or 2) decreasing the effective central blood volume and central venous pressure, thereby reducing venous return and cardiac output. Persons who are hypohydrated and encounter environmental heat stress would be hypovolemic and still have to perfuse simultaneously the cutaneous vasculature and contracting skeletal muscles. A substantial volume of blood could be redirected to the skin and thus be removed from the effective central circulation and be unavailable to perfuse the skeletal muscles (47, 48). Thus, both climatic heat stress and hypohydration can act independently to limit cardiac output and therefore oxygen delivery during maximal exercise.

The effects of hypohydration and air temperature on submaximal work output are illustrated in **Figure 5**, which draws on research conducted >50 y ago in the California desert by Adolph et al (17). A metabolic rate of ≈ 650 W (which represents “hard work” for occupational tasks but “moderate work” for athletes) was assumed, with an air temperature of 43°C and low humidity (49). Climatic heat stress reduced submaximal work output at all hydration levels. In addition, the work output decrements from heat stress and hypohydration were additive (17). For example, exposure to 43°C reduced work output by $\approx 25\%$ (compared with temperate conditions), and a 2.5% BWL (compared with euhydration) reduced work output by the same amount; if these events were experienced together, work output was decreased by 50%.

Hypohydration also impairs endurance performance in athletes. Armstrong et al (40) had athletes compete in 1500-, 5000-, and 10000-m races when euhydrated and when hypohydrated.

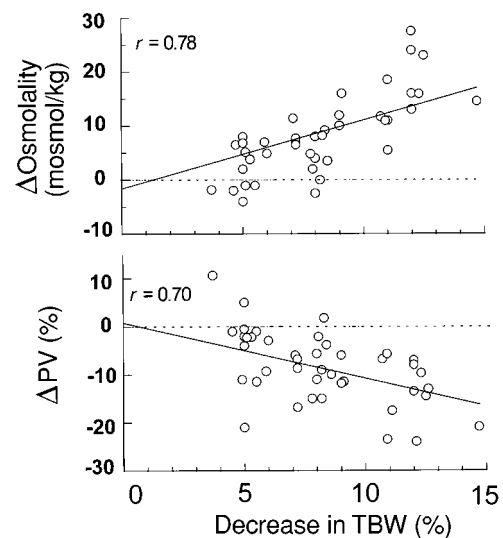


FIGURE 4. Body water loss effects on plasma osmolality and plasma volume (PV) in heat-acclimated persons (33). TBW, total body water.

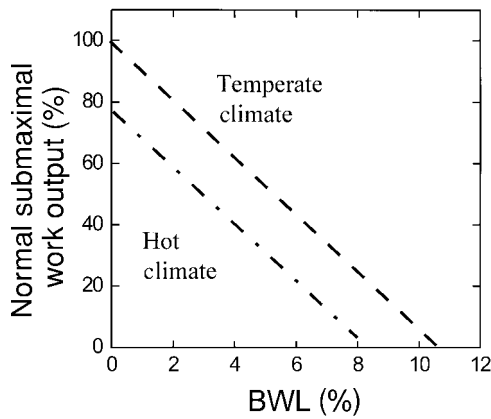


FIGURE 5. Effects of hydration level and climatic temperature on submaximal work output (17). BWL, body weight loss from dehydration.

Hypohydration (2% BWL) was achieved by diuretic administration (furosemide), which decreased plasma volume by 11%. Running performance was impaired at all distances but more in the longer races ($\approx 5\%$ for the 5000 and 10000 m) than in the shortest race (3% for the 1500 m). Burge et al (50), who examined simulated 2000-m rowing performance, found that it took the rowers an average of 22 s longer to complete the task when they were hypohydrated than when they were euhydrated. In addition, hypohydration reduced average power by 5%.

Surprisingly, few investigators have documented the effects of hypohydration on physiologic tolerance to submaximal exercise in the heat. In research conducted by Adolph et al (17), soldiers attempted endurance (2–23 h) walks in the California desert at 4–6.5 km/h [ambient temperature (T_a) $\approx 38^\circ\text{C}$] and either drank water ad libitum or refrained from drinking. These investigators reported that 1 of 59 (2%) soldiers who drank and 11 of 70 (16%) who did not drink suffered exhaustion from heat strain. In subsequent experiments, they reported that 1 of 59 drinking subjects (2%) and 15 of 70 nondrinking subjects (21%) suffered exhaustion from heat strain during an attempted 8-h desert walk. The magnitude of hypohydration was not provided in either set of experiments.

About a decade later, Ladell (51) had subjects attempt a 140-min walk in a hot ($T_a = 38^\circ\text{C}$) environment while ingesting different combinations of salt and water. Exhaustion from heat strain occurred in 9 of 12 (75%) experiments when subjects received neither water nor salt and 3 of 41 (7%) experiments when subjects received only water. More recently, Sawka et al (41) had subjects attempt lengthy treadmill walks ($\approx 25\%$ of maximal oxygen uptake for 140 min) in a hot-dry ($T_a = 49^\circ\text{C}$, relative humidity = 20%) environment when euhydrated and when hypohydrated by 3%, 5%, and 7% BWL. All 8 subjects completed the euhydration and 3% hypohydration experiments, and 7 of 8 completed the 5% hypohydration experiments. For the 7% hypohydration experiments, 6 subjects discontinued after completing only 64 min (mean). Clearly, hypohydration increases the incidence of exhaustion from heat strain.

To address whether hypohydration alters physiologic tolerance to heat strain, Sawka et al (52) had subjects walk to exhaustion in either a euhydrated or hypohydrated (8% of total body water) state. The experiments were designed so that the combined environment ($T_a = 49^\circ\text{C}$, relative humidity = 20%) and

exercise intensity (47% of maximal oxygen uptake) would not allow thermal equilibrium, and heat exhaustion would eventually occur. Hypohydration reduced tolerance time from 121 to 55 min, but more important, it reduced the core temperature a person could tolerate (the core temperature at heat exhaustion was ≈ 0.4 degrees lower in the hypohydrated state). These findings suggest that hypohydration not only impairs exercise performance but also reduces physiologic tolerance to heat strain.

TEMPERATURE REGULATION

Hypohydration increases core temperature responses during exercise in temperate (11, 53) and hot (12, 41) climates. A critical deficit of 1% of body weight elevates core temperature during exercise (54). As the magnitude of water deficit increases, there is a concomitant graded elevation of core temperature during exercise heat stress (41, 55). Relations between body water loss and core temperature elevations reported by studies that examined several water deficits are shown in **Figure 6** (33). The magnitude of core temperature elevation ranges from 0.1 to 0.23°C for every percentage point of body weight lost (17, 41, 55, 56).

Hypohydration not only elevates core temperature responses but also negates the core temperature advantages conferred by high aerobic fitness and heat acclimation (10–12). The effects of hypohydration (5% BWL) on core temperature responses in the same persons when unacclimated and when acclimated are shown in **Figure 7** (12). Acclimation lowered core temperature responses when subjects were euhydrated; when they were hypohydrated, similar core temperature responses were observed for both acclimation states. Thus, the core temperature penalty induced by hypohydration was greater in heat-acclimated than in unacclimated persons.

Hypohydration impairs both dry and evaporative heat loss (or, if the air is warmer than the skin, dehydration aggravates dry heat gain) (6, 7, 9, 57). In **Figure 8**, the local sweating response (58) and skin blood flow responses (59) to hypohydration (5% BWL) during exercise in the heat are illustrated. As shown, hypohydration reduces both effector heat loss responses for a given core temperature (36). In addition, hypohydration is usually associated with either reduced or unchanged whole-body sweating rates at a given metabolic rate in the heat (60). However, even when hypohydration is associated with no change in sweating rate, the core temperature is usually elevated, so that

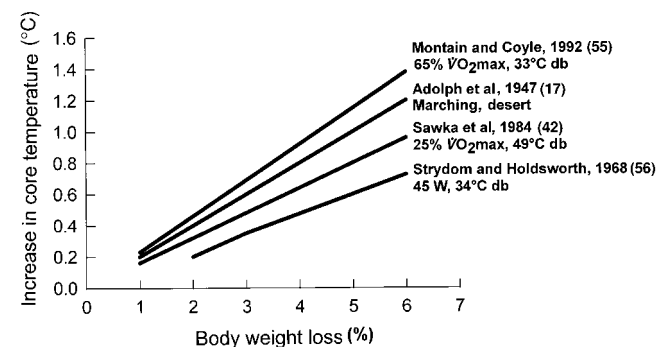


FIGURE 6. Relation between the elevation in core temperature (above euhydration) and hypohydration (measured as percentage body weight loss) during exercise heat stress (33). $\dot{V}O_{2\text{max}}$, maximal aerobic power; db, dry bulb temperature.



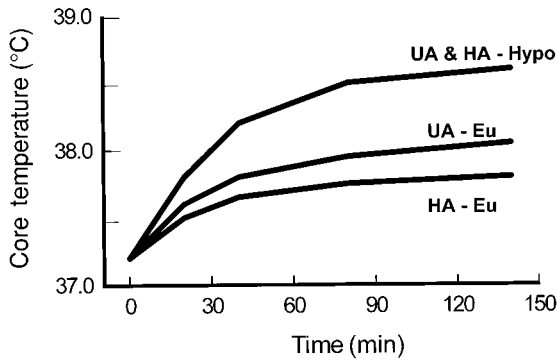


FIGURE 7. Core temperature responses during exercise heat stress in euhydrated (EU) and hypohydrated (hypo; 5% body weight loss) persons both before (UA) and after (HA) being heat acclimated.

the sweating rate for a given core temperature is lower when a person is hypohydrated (60).

Both the singular and combined effects of plasma hyperosmolality and hypovolemia have been suggested as mediating the reduced heat loss response during exercise heat stress (36). Changes in plasma osmolality may relate to tonicity changes in the extracellular fluid that bathes the hypothalamic neurons (61–63). Silva and Boulant (64) showed that in rat brain slices, preoptic-anterior hypothalamic neurons are present that are both thermosensitive and osmosensitive. Such data suggest a central interaction between thermoregulation and body water regulation (65).

Isotonic hypohydration alone can impair heat loss and increase core temperature during exercise heat exposure (66–68). Isotonic hypohydration reduces skin blood flow for a given core temperature and thus the potential for dry heat exchange (67, 69). Fortney et al (69) provided a rationale as to why an isoosmotic hypohydration might reduce skin blood flow and sweating rate, theorizing that hypovolemia might reduce cardiac preload and alter the activity of atrial baroreceptors, which have afferent input to the hypothalamus. Thus, a reduced atrial filling pressure might modify neural information to the hypothalamic thermoregulatory centers, which control skin blood flow and sweating. Gonzalez-Alonso et al (70) showed that hypohydration-mediated hypovolemia increases plasma norepinephrine, which is associated with increased cutaneous vascular resistance and reduced blood flow during exercise heat stress. Other studies reported that acute unloading of atrial baroreceptors during exercise with periods of lower-body negative

pressure (physiologic technique to unload cardiopulmonary baroreceptors) impairs heat loss and increases core temperature (71, 72).

The effects of hypohydration on cardiovascular responses to exercise have been investigated by several researchers (70, 73–75). During submaximal exercise with little heat strain, hypohydration elicits an increase in heart rate and decrease in stroke volume, but cardiac output does not usually change from what is seen in a euhydrated state (43–45). Apparently, during hypohydration a decreased blood volume reduces central venous pressure (73) and cardiac filling, which reduces stroke volume and requires a compensatory increase in heart rate (75). The combination of exercise with heat strain results in competition between the central and peripheral circulations for a limited blood volume (47, 48). As body temperature increases during exercise, cutaneous vasodilation occurs and the superficial veins become more compliant, thus decreasing venous resistance and pressure (15). As a result of decreased blood volume and blood displacement to cutaneous vascular beds, central venous pressure, venous return, and thus cardiac output decrease below euhydration values.

HYPERHYDRATION

Hyperhydration (greater than normal body water) has been suggested to improve thermoregulation and exercise heat performance above that achieved with hypohydration. The idea that hyperhydration might benefit exercise performance arose from observation of the adverse consequences of hypohydration. The theory was that expanding body water might reduce the cardiovascular and heat strain of exercise by expanding blood volume and reducing blood tonicity, thereby improving exercise performance.

Studies in which blood volume was directly expanded (eg, by infusion) have usually reported decreased cardiovascular strain (76–78) during exercise but have yielded disparate results on heat dissipation (77–79) and exercise heat performance (79, 80). Studies that attenuated plasma hyperosmolality during exercise heat stress generally have reported improved heat dissipation (58, 81–83) but have not addressed exercise performance.

Many studies have examined the effects of hyperhydration on thermoregulation in the heat, but most suffer from serious design problems (eg, the control condition represented hypohydration rather than euhydration) (33). Some investigators reported lower core temperatures during exercise after hyperhydration (83–87), but other researchers did not (75, 88, 89). Also, several studies (83, 84, 90) reported higher sweating rates with hyperhydration. In all studies, heart rate was lower during exercise with hyperhydration

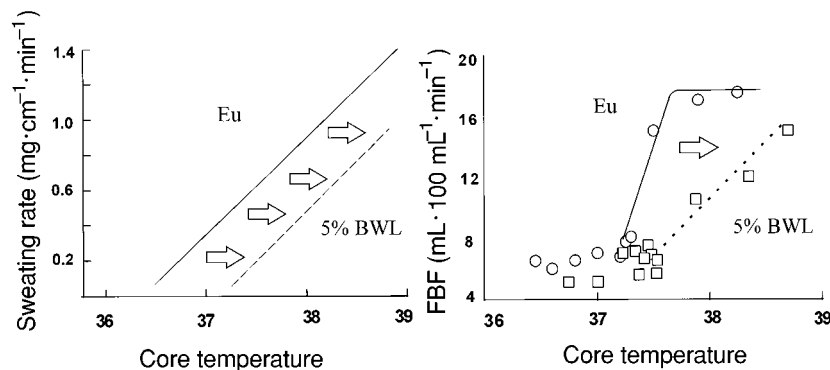


FIGURE 8. Local sweating rate and forearm skin blood flow (FBF) response data for euhydrated (Eu) (○) and hypohydrated (□) [5% body weight loss (BWL)] persons during exercise heat stress.

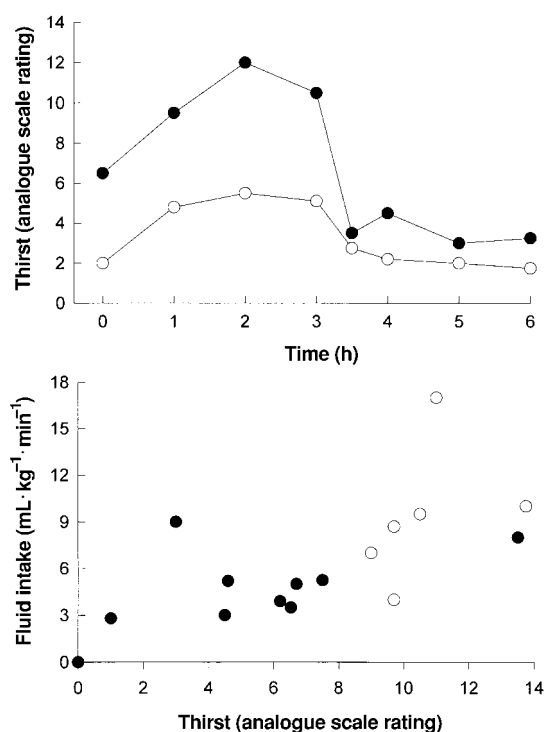


FIGURE 9. Subjective thirst sensation of younger (●) and older (○) men during exercise heat stress and the relation between drinking behavior and thirst sensation.

(33). Together, these findings support the notion that hyperhydration might reduce the thermal and cardiovascular strain of exercise.

The mechanisms responsible for the lower exercise core temperatures in the hyperhydrated state remain unclear. Several studies reported that overdrinking before exercise lowers body core temperature before exercise (84, 85), but this is likely due to the energy cost of warming the ingested fluid. In these studies, exercise per se did not exacerbate the difference that existed before exercise. Hyperhydration, therefore, apparently did not improve heat dissipation during the exercise period. Other studies, however, reported greater exercise sweating rates when subjects were hyperhydrated (84, 85, 90). In addition, Grucza et al (87) found that sweating began earlier when subjects were hyperhydrated. The findings of these last 4 studies suggest that hyperhydration may improve heat dissipation during exercise heat stress.

Although many studies have tried to induce hyperhydration by having subjects overdrink water or take water-electrolyte solutions, these approaches have resulted in only transients expansions of body water. Often, much of the fluid overload is rapidly excreted (91). Evidence has accrued that greater fluid retention can be achieved with an aqueous solution containing glycerol (91, 92). Riedesel et al (92) were the first to report that after hyperhydration with a glycerol solution, subjects excreted significantly less of the water load than did those consuming water alone.

Lyons et al (90) studied whether glycerol-mediated hyperhydration improves thermoregulatory responses to exercise heat stress. Subjects completed 3 trials in which they exercised in a hot (42°C) climate. For one trial, fluid ingestion was restricted to 5.4 mL/kg body wt, and in the other 2 trials subjects ingested

water (21.4 mL/kg) with or without a bolus of glycerol (1 g/kg). Ninety minutes after this hyperhydration period, the subjects began exercise. Compared with drinking water alone, glycerol ingestion increased fluid retention by 30%. In addition, during exercise, glycerol hyperhydration produced a higher sweating rate and substantially lower core temperature (0.7°C) than did control conditions and water hyperhydration. Research from our own laboratory has failed to show any thermoregulatory advantages of either water or glycerol hyperhydration during exercise heat stress (93, 94).

Few studies have examined whether hyperhydration improves exercise performance or heat tolerance. In a study by Blyth and Burt (95), the first to report the effects of hyperhydration on performance during exercise heat stress, subjects ran to exhaustion in a hot climate (49°C) when normally hydrated and when hyperhydrated by drinking 2 L of fluid 30 min before exercise. Thirteen of 18 subjects ran longer when hyperhydrated, but the difference in average time to exhaustion (17.3 compared with 16.9 min) was not statistically significant. More recently, Luetkemeier and Thomas (80), who examined whether hypervolemia improved cycling performance, reported that expansion of blood volume (by 450–500 mL) increased simulated time trial performance by 10% (81 compared with 90 min).

RESEARCH NEEDS

Older adults, persons with cystic fibrosis, and persons who have suffered a spinal cord injury all have unique problems associated with fluid and electrolyte balance during exercise heat stress; additional research is needed on all 3 of these groups. For example, older adults (>55 y) have reduced thirst sensation, less ability to concentrate urine, and reduced potential to dissipate body heat (96–98). The reduced thirst sensation older persons experience when fluid homeostasis is challenged (96) is illustrated in **Figure 9** from the research of Mack et al (98). Those investigators compared younger (18–28 y) and older (65–78 y) persons for thirst sensation and fluid intake during exercise heat stress. After this perturbation, both groups were hypohydrated by $\approx 2.5\%$ BWL, and thirst sensation and fluid intakes were lower in the older group. However, when matched fluid intakes were plotted against thirst sensation, the 2 groups drank the same amounts. Thus, older persons have a reduced thirst sensation, but for a given thirst sensation, they have the same drinking behavior. This observation is important in light of the reduced ability of older persons to conserve water and electrolytes in their kidneys when challenged by body water deficits. Research issues include the testing of strategies [eg, adding solute to stimulate thirst (2)] to optimize fluid and electrolyte replacement in these populations and the effects of hyperhydration on thermoregulation in this group.


Persons with cystic fibrosis, a multisystem disorder that alters sweat gland function, suffer a high incidence of heat injury or illness, probably because of fluid or electrolyte imbalances (99, 100). They have high sweating rates and very high sweat sodium chloride losses that are not abated by heat acclimation (100). In addition, cystic fibrosis patients have suppressed thirst sensations because of the high sweat solute losses, which reduce the osmotic drive for thirst (2, 8, 99). Together, these factors cause large body water deficits during exercise heat stress (99). If cystic fibrosis patients are forced to drink, however, they tolerate the consumed fluid as well as do their healthy counterparts (99). Research

should focus on developing fluid-electrolyte strategies to stimulate thirst and minimize voluntary dehydration in this population.

Persons with spinal cord injury have an impaired ability to thermoregulate during heat stress (101–104). The magnitude of impairment is related to the level and completeness of the lesion; the higher and more complete the cord injury, the greater the thermoregulatory impairment (105). The consequences of spinal cord injury are loss of sympathetic control of heat loss (via vasomotor and sudomotor adjustments) over large areas of skin (106) and the isolation of thermal receptors throughout the body (107). During heat exposure, some sweating can occur over the insensate skin, but it is sparse and not synchronous with sensate skin sweating. Together, these factors lead to a reduced ability to thermoregulate during exercise heat stress (105). Research has not addressed water and electrolyte requirements, thirst sensation, or thermoregulation during hypohydration for this population.

A final research need is the development of rapid, noninvasive technologies to measure total body water and hydration status (108). Valid methods are needed that have enough resolution to measure deficits as small as 5% of total body water.

SUMMARY

During exercise, sweat output often exceeds water intake, producing a body water deficit (hypohydration). The water deficit lowers both intracellular and extracellular volume and also results in plasma hypertonicity and hypovolemia. Aerobic exercise tasks are likely to be adversely affected by hypohydration (109), potentially more so at higher exercise intensities in warm environments (51). Hypohydration increases heat storage and reduces a person's ability to tolerate heat strain. The increased heat storage is mediated by a reduced sweating rate and lower skin blood flow for a given core temperature. Hyperhydration may reduce thermal strain during exercise in the heat, but data supporting that notion are not robust (110). 

REFERENCES

1. Sawka MN. Body fluid responses and hypohydration during exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human performance physiology and environmental medicine at terrestrial extremes*. Indianapolis: Cooper Publishing Group, 1988:227–66.
2. Mack GW, Nadel ER. Body fluid balance during heat stress in humans. In: Fregly MJ, Blatteis CM, eds. *Environmental physiology*. New York: Oxford University Press, 1996:187–214.
3. Marriott BM, ed. *Fluid replacement and heat stress*. Washington, DC: National Academy Press, 1994.
4. Marriott BM, ed. *Nutritional needs in hot environments: application for military personnel in field operations*. Washington, DC: National Academy Press, 1993.
5. Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine position stand: exercise and fluid replacement. *Med Sci Sports Exerc* 1996;28:i–vii.
6. Montain SJ, Maughan RJ, Sawka MN. Fluid replacement strategies for exercise in hot weather. *Athletic Therapy Today* 1996;1:24–7.
7. Sawka MN, Pandolf KB. Effects of body water loss on physiological function and exercise performance. In: Gisolfi CV, Lamb DR, eds. *Perspectives in exercise science and sports medicine*. Vol 3. Carmel, IN: Benchmark Press, 1990:1–38.
8. Greenleaf JE. Problem: thirst, drinking behavior, and involuntary dehydration. *Med Sci Sports Exerc* 1992;24:645–56.
9. Sawka MN. Physiological consequences of hypohydration: exercise performance and thermoregulation. *Med Sci Sports Exerc* 1992;24:657–70.
10. Buskirk ER, Iampietro PF, Bass DE. Work performance after dehydration: effects of physical conditioning and heat acclimatization. *J Appl Physiol* 1958;12:189–94.
11. Cadarette BS, Sawka MN, Toner MM, Pandolf KB. Aerobic fitness and the hypohydration response to exercise-heat stress. *Aviat Space Environ Med* 1984;55:507–12.
12. Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: effects of heat acclimation, gender, and environment. *J Appl Physiol* 1983;55:1147–53.
13. Hubbard RW. Water as a tactical weapon: a doctrine for preventing heat casualties. *Army Sci Conf Proc* 1982;2:125–39.
14. Hales JRS, Hubbard RW, Gaffin SL. Limitations of heat tolerance. In: Fregly MJ, Blatteis CM, eds. *Handbook of physiology*. Section 4. New York: Oxford University Press, 1996:285–355.
15. Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: Fregly MJ, Blatteis CM, eds. *Handbook of physiology*. Section 4, environmental physiology. New York: Oxford University Press, 1996:157–85.
16. Shapiro Y, Pandolf KB, Goldman RF. Predicting sweat loss response to exercise, environment and clothing. *Eur J Appl Physiol* 1982;48:83–96.
17. Adolph EF, Associates. *Physiology of man in the desert*. New York: Intersciences, 1947.
18. Levine L, Quigley MD, Cadarette BS, Sawka MN, Pandolf KB. Physiologic strain associated with wearing toxic-environment protective systems during exercise in the heat. In: Das B, ed. *Advances in industrial ergonomics and safety II*. London: Taylor & Francis, 1990:897–904.
19. Nelson N, Eichna LW, Bean, WB. Determination of water and salt requirements for desert operations. Fort Knox, KY: Armored Force Medical Research Laboratory, 1943. (Report no. 2-6.)
20. Greenleaf JE. Environmental issues that influence intake of replacement beverages. In: Marriott BM, ed. *Fluid replacement and heat stress*. Washington, DC: National Academy Press, 1994:195–214.
21. Brouns F. Heat—sweat—dehydration—rehydration: a praxis oriented approach. *J Sports Sci* 1991;9:143–52.
22. Allan JR, Wilson CG. Influence of acclimatization on sweat sodium concentration. *J Appl Physiol* 1971;30:708–12.
23. Meyer F, Bar-Or O, MacDougall D, Heigenhauser GJF. Sweat electrolyte loss during exercise in the heat: effects of gender and maturation. *Med Sci Sports Exerc* 1992;24:776–81.
24. Morimoto T, Slabochova Z, Naman RK, Sargent F 2nd. Sex differences in physiological reactions to thermal stress. *J Appl Physiol* 1967;22:526–32.
25. Clarkson PM. The effects of exercise and heat on vitamin requirements. In: Marriott BM, ed. *Nutritional needs in hot environments*. Washington, DC: National Academy Press, 1993:137–71.
26. Hubbard RW, Sandick BL, Matthew WT, et al. Voluntary dehydration and alliesthesia for water. *J Appl Physiol* 1984;57:868–73.
27. Engell DB, Maller O, Sawka MN, Francesconi RN, Drolet L, Young AJ. Thirst and fluid intake following graded hypohydration levels in humans. *Physiol Behav* 1987;40:229–36.
28. Armstrong LE, Hubbard RW, Szlyk PC, Matthew WT, Sils IV. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. *Aviat Space Environ Med* 1985;56:765–70.
29. Neuffer PD, Young AJ, Sawka MN. Gastric emptying during exercise: effects of heat stress and hypohydration. *Eur J Appl Physiol* 1989;58:433–9.
30. Owen MD, Kregel KC, Wall PT, Gisolfi CV. Effects of ingesting carbohydrate beverages during exercise in the heat. *Med Sci Sports Exerc* 1986;18:568–75.
31. Neuffer PD, Sawka MN, Young A, Quigley M, Latzka WA, Levine L. Hypohydration does not impair skeletal muscle glycogen resynthesis after exercise. *J Appl Physiol* 1991;70:1490–4.
32. Nose H, Morimoto T, Ogura K. Distribution of water losses among fluid compartments of tissues under thermal dehydration in the rat. *Jpn J Physiol* 1983;33:1019–29.

33. Sawka MN, Montain SJ, Lutzka WA. Body fluid balance during exercise-heat exposure. In: Buskirk ER, Puhl SM, eds. *Body fluid balance: exercise and sport*. Boca Raton, FL: CRC Press, 1996:143–61.
34. Senay LC Jr. Relationship of evaporative rates to serum $[Na^+]$, $[K^+]$, and osmolarity in acute heat stress. *J Appl Physiol* 1968;25:149–52.
35. Kubica R, Nielsen B, Bonnesen A, Rasmussen IB, Stoklosa J, Wilk B. Relationship between plasma volume reduction and plasma electrolyte changes after prolonged bicycle exercise, passive heating and diuretic dehydration. *Acta Physiol Pol* 1983;34:569–79.
36. Sawka MN, Greenleaf JE. Current concepts concerning thirst, dehydration, and fluid replacement: overview. *Med Sci Sports Exerc* 1992;24:643–4.
37. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-, diuretic-, and exercise-induced hypohydration. *J Appl Physiol* 1984;57:1018–23.
38. Webster S, Rutt R, Weltman A. Physiological effects of a weight loss regimen practiced by college wrestlers. *Med Sci Sports Exerc* 1990;22:229–34.
39. Craig EN, Cummings EG. Dehydration and muscular work. *J Appl Physiol* 1966;21:670–4.
40. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc* 1985;17:456–61.
41. Sawka MN, Young AJ, Francesconi RP, Muza SR, Pandolf KB. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol* 1985;59:1394–401.
42. Sawka MN, Francesconi RP, Pimental NA, Pandolf KB. Hydration and vascular fluid shifts during exercise in the heat. *J Appl Physiol* 1984;56:91–6.
43. Allen TE, Smith DP, Miller DK. Hemodynamic response to submaximal exercise after dehydration and rehydration in high school wrestlers. *Med Sci Sports* 1977;9:159–63.
44. Sproles CB, Smith DP, Byrd RJ, Allen TE. Circulatory responses to submaximal exercise after dehydration and rehydration. *J Sports Med Phys Fitness* 1976;16:98–105.
45. Saltin B. Circulatory response to submaximal and maximal exercise after thermal dehydration. *J Appl Physiol* 1964;19:1125–32.
46. Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol* 1985;53:294–8.
47. Rowell LB. *Human circulation: regulation during physical stress*. New York: Oxford University Press, 1986.
48. Johnson JM, Proppe DW. Cardiovascular adjustments to heat stress. In: Fregley MJ, Blatteis CM, eds. *Handbook of physiology*. Section 4. New York: Oxford University Press, 1996:215–43.
49. Pandolf KB, Stroschein LA, Drolet LL, Gonzalez RR, Sawka MN. Prediction modeling of physiological responses and human performance in the heat. *Comput Biol Med* 1986;16:319–29.
50. Burge CM, Carey MF, Payne WR. Rowing performance, fluid balance, and metabolic function following dehydration and rehydration. *Med Sci Sports Exerc* 1993;25:1358–64.
51. Ladell WSS. The effects of water and salt intake upon the performance of men working in hot and humid environments. *J Physiol* 1955;127:11–46.
52. Sawka MN, Young AJ, Lutzka WA, Neuffer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: influence of hydration. *J Appl Physiol* 1992;73:368–75.
53. Grande F, Monagle JE, Buskirk ER, Taylor HL. Body temperature responses to exercise in man on restricted food and water intake. *J Appl Physiol* 1959;14:194–8.
54. Ekblom B, Greenleaf CJ, Greenleaf JE, Hermansen L. Temperature regulation during exercise dehydration in man. *Acta Physiol Scand* 1970;79:475–83.
55. Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992;73:1340–50.
56. Strydom NB, Holdsworth LD. The effects of different levels of water deficit on physiological responses during heat stress. *Gut* 1968;26:95–102.
57. Montain SJ, Lutzka WA, Sawka MN. Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *J Appl Physiol* 1995;79:1434–9.
58. Sawka MN, Gonzalez RR, Young AJ, Dennis RC, Valeri CR, Pandolf KB. Control of thermoregulatory sweating during exercise in the heat. *Am J Physiol* 1989;257:R311–6.
59. Kenney WL, Tankersley CG, Newswanger DL, Hyde DE, Puhl SM, Turner NL. Age and hypohydration independently influence the peripheral vascular response to heat stress. *J Appl Physiol* 1990;68:1902–8.
60. Sawka MN, Francesconi RP, Young AJ, Pandolf KB. Influence of hydration level and body fluids on exercise performance in the heat. *JAMA* 1984;252:1165–9.
61. Kozlowski S, Greenleaf JE, Turlejska E, Nazar K. Extracellular hyperosmolality and body temperature during physical exercise in dogs. *Am J Physiol* 1980;239:R180–3.
62. Senay LC Jr. Temperature regulation and hypohydration: a singular view. *J Appl Physiol* 1979;47:1–7.
63. Boulant JA. Hypothalamic neurons regulating body temperature. In: Fregley MJ, Blatteis CM, eds. *Handbook of physiology*. Section 4. New York: Oxford University Press, 1996:105–25.
64. Silva NL, Boulant JA. Effects of osmotic pressure, glucose, and temperature on neurons in preoptic tissue slices. *Am J Physiol* 1984;247:R335–45.
65. Hori T, Nakashima T, Koga A, Kiyohama T, Inoue T. Convergence of thermal, osmotic and cardiovascular signals on preoptic and anterior hypothalamic neurons in the rat. *Brain Res Bull* 1988;20:879–85.
66. Claremont AD, Costill DL, Fink W, Van Handel P. Heat tolerance following diuretic induced dehydration. *Med Sci Sports* 1976;8:239–43.
67. Nadel ER. Circulatory and thermal regulations during exercise. *Fed Proc* 1980;39:1491–7.
68. Fortney SM, Nadel ER, Wenger CB, Bove JR. Effect of acute alterations of blood volume on circulatory performance in humans. *J Appl Physiol* 1981;50:292–8.
69. Fortney SM, Nadel ER, Wenger CB, Bove JR. Effect of blood volume on sweating rate and body fluids in exercising humans. *J Appl Physiol* 1981;51:1594–600.
70. Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. *J Appl Physiol* 1995;79:1487–96.
71. Gaddis GM, Elizondo RS. Effect of central blood volume decrease upon thermoregulation responses to exercise in the heat. *Fed Proc* 1984;43:627.
72. Mack G, Nose H, Nadel ER. Role of cardiopulmonary baroreflexes during dynamic exercise. *J Appl Physiol* 1988;65:1827–32.
73. Morimoto T. Thermoregulation and body fluids: role of blood volume and central venous pressure. *Jpn J Physiol* 1990;40:165–79.
74. Sawka MN, Knowlton RG, Critz JB. Thermal and circulatory responses to repeated bouts of prolonged running. *Med Sci Sports* 1979;11:177–80.
75. Nadel ER, Fortney SM, Wenger CB. Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol* 1980;49:715–21.
76. Hopper MK, Coggan AR, Coyle EF. Exercise stroke volume relative to plasma-volume expansion. *J Appl Physiol* 1988;64:404–8.
77. Nose H, Mack GW, Shi XR, Morimoto K, Nadel ER. Effect of saline infusion during exercise on thermal and circulatory regulations. *J Appl Physiol* 1990;69:609–16.
78. Sawka MN, Hubbard RW, Francesconi RP, Horstman DH. Effects of acute plasma volume expansion on altering exercise-heat performance. *Eur J Appl Physiol* 1983;51:303–12.
79. Deschamps A, Levy RD, Cosio MG, Marliss EB, Magder S. Effect of saline infusion on body temperature and endurance during heavy exercise. *J Appl Physiol* 1989;66:2799–804.
80. Luetkemeier MJ, Thomas EL. Hypervolemia and cycling time trial performance. *Med Sci Sports Exerc* 1994;26:503–9.

81. Fortney SM, Wenger CB, Bove JR, Nadel ER. Effect of hyperosmolality on control of blood flow and sweating. *J Appl Physiol* 1984; 57:1688-95.
82. Harrison MH, Edwards RJ, Fennessy PA. Intravascular volume and tonicity as factors in the regulation of body temperature. *J Appl Physiol* 1978;44:69-75.
83. Nielsen B. Effects of changes in plasma volume and osmolarity on thermoregulation during exercise. *Acta Physiol Scand* 1974;90: 725-30.
84. Moroff SV, Bass DE. Effects of overhydration on man's physiological responses to work in the heat. *J Appl Physiol* 1965;20: 267-70.
85. Nielsen B, Hansen G, Jorgensen SO, Nielsen E. Thermoregulation in exercising man during dehydration and hyperhydration with water and saline. *Int J Biometeorol* 1971;15:195-200.
86. Gisolfi CV, Copping JR. Thermal effects of prolonged treadmill exercise in the heat. *Med Sci Sports* 1974;6:108-13.
87. Grucza R, Szczypaczewska M, Kozlowski S. Thermoregulation in hyperhydrated men during physical exercise. *Eur J Appl Physiol* 1987;56:603-7.
88. Greenleaf JE, Castle BL. Exercise temperature regulation in man during hypohydration and hyperhydration. *J Appl Physiol* 1971;30: 847-53.
89. Candas V, Libert JP, Brandenberger G, Sagot JC, Kahn JM. Thermal and circulatory responses during prolonged exercise at different levels of hydration. *J Physiol (Paris)* 1988;83:11-8.
90. Lyons TP, Riedesel ML, Meuli LE, Chick TW. Effects of glycerol-induced hyperhydration prior to exercise in the heat on sweating and core temperature. *Med Sci Sports Exerc* 1990;22:477-83.
91. Freund BJ, Montain SJ, Young AJ, et al. Glycerol hyperhydration: hormonal, renal, and vascular fluid responses. *J Appl Physiol* 1995; 79:2069-77.
92. Riedesel ML, Allen DY, Peake GT, Al-Qattan K. Hyperhydration with glycerol solutions. *J Appl Physiol* 1987;63:2262-8.
93. Latzka WA, Sawka MN, Montain SJ, et al. Hyperhydration: thermoregulatory effects during compensable exercise-heat stress. *J Appl Physiol* 1997;83:860-6.
94. Latzka WA, Sawka MN, Montain SJ, et al. Hyperhydration: tolerance and cardiovascular effects during uncompensable exercise-heat stress. *J Appl Physiol* 1998;84:1858-64.
95. Blyth CS, Burt JJ. Effect of water balance on ability to perform in high ambient temperatures. *Res Q Exerc Sport* 1932;3:301-7.
96. Kenney WL. Body fluid and temperature regulation as a function of age. In: Lamb DR, Gisolfi CV, Nadel E, eds. *Exercise in older adults*. Indianapolis: Cooper Publishing, 1995:305-51.
97. Kenney WL, Fowler SR. Methylcholine-activated eccrine sweat gland density and output as a function of age. *J Appl Physiol* 1988; 65:1082-6.
98. Mack GW, Weseman CA, Langhans GW, Scherzer H, Gillen CM, Nadel ER. Body fluid balance in dehydrated healthy older men: thirst and renal osmoregulation. *J Appl Physiol* 1994;76:1615-23.
99. Bar-Or O, Blimkie CJ, Hay JA, MacDougall JD, Ward DS, Wilson WM. Voluntary dehydration and heat intolerance in cystic fibrosis. *Lancet* 1992;339:696-9.
100. Orenstein DM, Henke KG, Green CG. Heat acclimation in cystic fibrosis. *J Appl Physiol* 1984;57:408-12.
101. Fitzgerald PI, Sedlock DA, Knowlton RG. Circulatory and thermal adjustments to prolonged exercise in paraplegic women. *Med Sci Sports Exerc* 1990;22:629-35.
102. Attia M, Engel P. Thermoregulatory set-point in paraplegia. In: Hales JRS, ed. *Thermal physiology*. New York: Raven Press, 1984:79-82.
103. Hopman MT, Oeseburg B, Binkhorst RA. Cardiovascular responses in persons with paraplegia to prolonged arm exercise and thermal stress. *Med Sci Sports Exerc* 1993;25:577-83.
104. Gass GC, Camp EM, Nadel ER, Gwinn TH, Engel P. Rectal and rectal vs. esophageal temperatures in paraplegic men during prolonged exercise. *J Appl Physiol* 1988;64:2265-71.
105. Sawka MN, Latzka WA, Pandolf KB. Temperature regulation during upper body exercise: able-bodied and spinal cord injured. *Med Sci Sports Exerc* 1989;21:S132-40.
106. Normell LA. Distribution of impaired cutaneous vasomotor and sudomotor function in paraplegic man. *Scand J Clin Lab Invest* 1974;138:25-41.
107. Freund PR, Brengelmann GL, Rowell LB, Halar E. Attenuated skin blood flow response to hyperthermia in paraplegic men. *J Appl Physiol* 1984;56:1104-9.
108. O'Brien C, Baker-Fulco CJ, Young AJ, Sawka MN. Bioimpedance assessment of hypohydration. *Med Sci Sports Exerc* 1999;31:1466-71.
109. Montain SJ, Smith SA, Matott RP, Zientara GP, Jolesz FA, Sawka MN. Hypohydration effects on skeletal muscle performance and metabolism. A ³¹P MRS study. *J Appl Physiol* 1998;84:1889-94.
110. Sawka MN, Coyle EF. Influence of body water and blood volume on thermoregulation and exercise performance in the heat. In: Holloszy JO, ed. *Exercise and sport sciences reviews*. Baltimore: Williams & Wilkins, 1999:167-218.

