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Physiological consequences of hypohydration: exercise performance and thermoregulation

MICHAEL N. SAWKA

U.S. Army Research Institute of Environmental Medicine, Natick, MA 01760–5007

ABSTRACT

SAWKA, M. N. Physiological consequences of hypohydration: exercise performance and thermoregulation. *Med. Sci. Sports Exerc.*, Vol. 24, No. 6, pp. 657–670, 1992. During exercise in the heat, sweat output often exceeds water intake, which results in a body water deficit or hypohydration. This water deficit occurs from both the intracellular and extracellular fluid compartments, and causes a hypertonic-hypovolemia of the blood. Aerobic exercise tasks are likely to be adversely affected by hypohydration; and the warmer the environment the greater the potential for performance decrements. Hypohydration causes greater heat storage and reduces one's ability to tolerate heat strain. The greater heat storage is mediated by reduced sweating rate (evaporative heat loss) and reduced skin blood flow (dry heat loss) for a given core temperature. Reductions of sweating rate and skin blood flow are most tightly coupled to blood hypertonicity and hypovolemia, respectively. In addition, hypovolemia and the displacement of blood to the skin make it difficult to maintain central venous pressure and thus an adequate cardiac output to simultaneously support metabolism and thermoregulation during exercise-heat stress.

BLOOD VOLUME, CORE TEMPERATURE, DEHYDRATION, HEAT STRESS, HYPEROSMOLALITY, HYPOVOLEMIA, SKIN BLOOD FLOW, SWEATING, TONICITY

Adolph and associates (1) were the first to effectively communicate the devastating effects that body water loss can have on physiological strain and exercise performance in the heat. During exercise-heat exposure, body water is primarily lost through sweat output, which results in increased plasma tonicity (osmolality) with decreased blood volume (hyperosmotic-hypovolemia). The fact that either hypertonicity or hypovolemia can adversely affect temperature regulation and exercise performance was established during the 1930s through the 1950s (see Table 1; 1,25,49,64,86) and has been confirmed by subsequent investigators. Exercise/environmental physiologists, however, have generally emphasized the role of hypovolemia and have often ignored the role of hypertonicity on degrading thermoregulation and exercise performance (63).

This paper describes the genesis of current concepts concerning the effects of body water loss on fluid redistribution, exercise performance, and temperature regulation. Particular attention is directed to delineating the relative importance of hypertonicity and hypovolemia on human thermoregulation and exercise performance. Throughout this paper, the term "euhydration" refers to "normal" body water content, whereas "hypohydration" refers to body water deficit. The more common term "dehydration" denotes the dynamic loss of body water or the transition from euhydration to hypohydration.

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BODY WATER LOSS AND REDISTRIBUTION

Physical exercise increases total body metabolic rate to provide energy for skeletal muscle contraction. Depending on the type of exercise, between 70% and 100% of metabolism is released as heat and needs to be dissipated in order to achieve body heat balance. Depending on the environmental temperature, the relative contributions of evaporative and dry heat exchange to the total heat loss vary, and the hotter the environment the greater the dependence on evaporative heat loss, and thus on sweating (80). Therefore, in hot environments, a considerable amount of body water is lost via eccrine sweat gland secretion to enable evaporative cooling of the body (128).

For athletes, the highest sweating rates occur during prolonged high intensity exercise in the heat. Figure 1 provides approximate hourly sweating rates, and therefore water requirements, for runners based upon metabolic rate data from several laboratories (105). The amount of body fluid lost as sweat can vary greatly, and sweating rates of 1 $l \cdot h^{-1}$ are common. The highest sweating rate reported in the literature was 3.7 $l \cdot h^{-1}$, which was measured for Alberto Salazar during the 1984 Olympic Marathon (5). The upper limits for fluid replacement during exercise-heat stress are set by the maximal gastric emptying rates, which approximate 1.0–1.5 $l \cdot h^{-1}$ for an average adult male (67,70). The

TABLE 1. Early observations on the role of blood volume and tonicity on temperature regulation in dehydrated humans.

	Blood Volume		Plasma Tonicity
1935	Lee and Mulder Protein content increased suggesting plasma volume decreased	*1939*	Dontas Sardine eating delayed sweating
1937	Harvard Fatigue Laboratory Boulder City, NV Blood volume decreased	*1942*	Desert Laboratory Unit Colorado Desert, CA Serum chloride increased
1942	Desert Laboratory Unit Colorado Desert, CA Blood volume decreased and lead to circulatory failure	*1956*	Pearcy et al. Serum chloride alters sweat rate during exercise

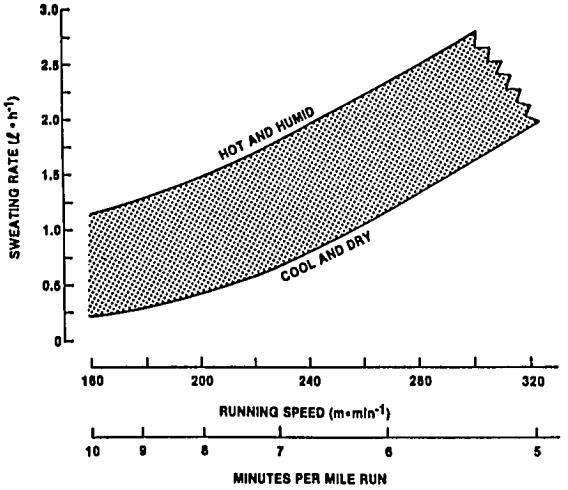

Figure 1—An approximation of hourly sweating rates for runners (105).

gastric emptying rates are reported to decreased during high intensity (>75% O_2max) exercise (22,75), hypohydration (76,93) and heat strain (76,85).

It is difficult to balance the volume of fluid consumed to the volume of sweat output during exercise-heat exposure. Investigators report (1,29) that thirst provides a poor index of body water requirements and that *ad libitum* drinking results in incomplete fluid replacement or "involuntary dehydration" (8,52,87). Humans will commonly dehydrate by 2–8% of their body weight during exercise-heat stress (1,5,10,92). In one study (29) of hypohydrated humans, the multiple regression analyses indicated that the consumed volume of rehydration fluid was related to plasma hyperosmolality ($r^2 = 0.58$), with only small amounts of additional variance accounted for by hypovolemia ($r^2 = 0.07$) and subjective thirst indices ($r^2 = 0.13$) after exercise-heat exposure.

Water is the largest component of the human body and represents 45–70% of body weight. The average male (75 kg) contains about 45 l of water, which corresponds to about 60% of body weight. Since adipose tissue is about 10% water and muscle tissue is about 75% water, a person's total body water depends upon their body composition (98). In addition, muscle water will vary with glycogen content because of the osmotic pressure exerted by glycogen granules within the muscle's sarcoplasm (74). As a result, trained athletes have a relatively greater total body water, than their sedentary counterparts, by virtue of a larger muscle mass and a higher skeletal muscle glycogen concentration.

The water contained in body tissues is distributed between the intracellular and extracellular fluid compartments. The intracellular fluid compartment is larger and contains about 30 l of water, whereas the extracellular fluid compartment contains about 15 l of water for the average 75-kg male. Sweating reduces total body water if adequate amounts of fluid are not consumed. As a consequence of free fluid exchange, hypohydration should affect the water content of each fluid compartment. Figure 2 illustrates the findings of two studies (20,27) on the redistribution of water between fluid compartments when hypohydrated (98). Costill et al. (20) dehydrated subjects by using a combination of cycle ergometer exercise and heat exposure. Shortly after completing cycle ergometer exercise, blood and skeletal muscle samples were obtained from their subjects. Durkot et al. (27) dehydrated rats by using a passive heat exposure for up to 11 h. The intent of Figure 2 is to present data trends and not to imply that a given percent decrease of total body water is similar between man and rat. At low volumes of body water loss, the water deficit primarily comes from the extracellular compartment; as the body water loss increases, a proportionately greater percentage of the water deficit comes from the intracellular compartment.

Nose and colleagues (83) determined the distribution of body water loss among the fluid spaces as well as among different body organs. These investigators thermally dehydrated rats by 10% of body weight, and after the animals regained their normal core temperature, the body water measurements were obtained. The water deficit was apportioned between the intracellular (41%) and extracellular (59%) compartments; and among the organs, 40% from muscle, 30% from skin, 14% from

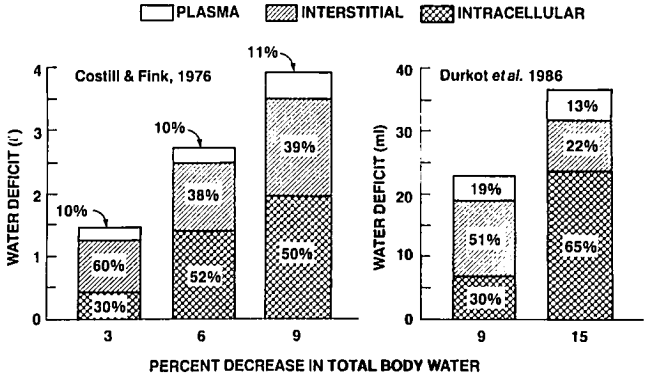

Figure 2—The findings of two studies (22,27) concerning the partitioning of water deficit between fluid compartments during resting conditions (98).

viscera, and 14% from bone. Neither the brain nor liver lost significant water content. Those investigators concluded that hypohydration results in water redistribution largely from the intra- and extracellular compartments of muscle and skin in order to maintain blood volume.

The method of dehydration might affect the partitioning of the remaining water between the body fluid spaces (60). For example, it is estimated that 3–4 g of water are associated with each gram of glycogen (84), although this relationship is variable (115). Several papers (21–23) report a substantial decrease of intracellular water content in skeletal muscle following 1.5–2.5 h of exercise dehydration. These investigators suggest that the loss of intracellular water may be the result of water released with the breakdown of muscle glycogen. Thus, exercise-induced hypohydration may result in a greater intracellular water loss than thermally induced hypohydration. Kozlowski and Saltin (60) have reported data to support this view, but Costill and Saltin (22) found no difference between exercise and thermal dehydration for the partitioning of water between the fluid compartments. Therefore, whether exercise and thermally induced hypohydration cause a difference in the redistribution of water between fluid compartments remains unresolved.

The redistribution of water between the intracellular and the extracellular compartment is dependent upon the osmotic gradient between these spaces. Cell membranes are freely permeable to water but only selectively permeable to various solutes. As a result, transient alterations in the solute concentration cause water redistribution across the cell membrane until the two fluid spaces are in equilibrium with respect to osmotic pressure. Therefore, if the methods used to attain hypohydration lead to differences in the intracellular and/or extracellular solute losses, the partitioning of water loss between the fluid compartments will vary accordingly.

It is known that exercise or heat-induced hypohydration will increase the osmotic pressure in the plasma (98,114). Eccrine sweat is ordinarily hypotonic relative to plasma (55); therefore, the plasma becomes hypertonic when dehydration is induced by sweat output (61,112). For humans, plasma osmolality can increase from about 283 mosmol·kg^{-1} when euhydrated to values exceeding 300 mosmol·kg^{-1} when hypohydrated (see Fig. 3) (110). Sodium, potassium, and their principal anion (chloride), are primarily responsible for the elevated plasma tonicity during hypohydration (61,112). As recently reemphasized by Nose et al. (81), it is the plasma hypertonicity that mobilizes fluid from the intracellular to the extracellular compartments to enable the defense of the blood (plasma) volume in hypohydrated subjects.

The electrolytes, sodium and chloride, that are lost

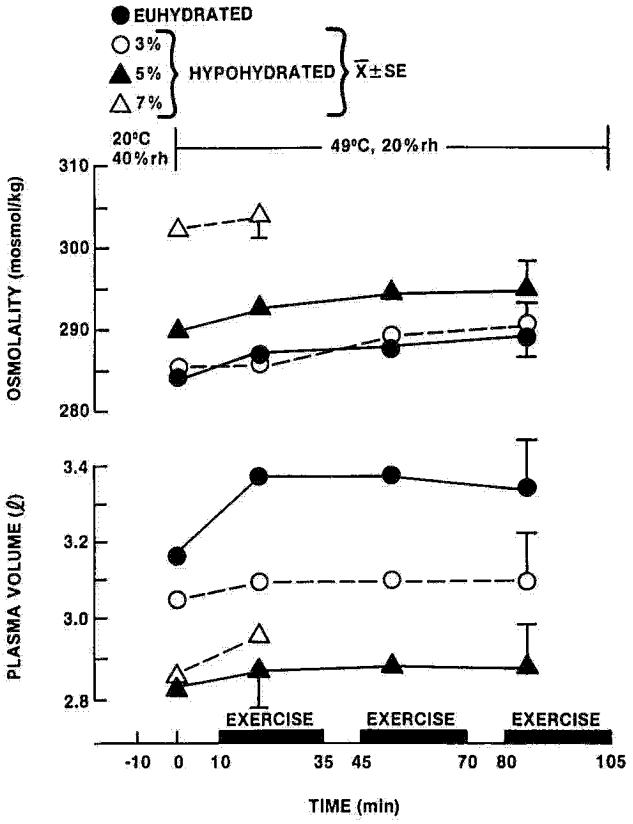

Figure 3—Plasma osmolality and plasma volume values (±SE) at rest and exercise when euhydrated (0%) and hypohydrated by percentage of body weight (110).

with sweat are primarily found in the extracellular fluid (98). The amount of electrolytes lost in sweat are modified by the individual's state of heat acclimation (2,55). For example, Kirby and Convertino (55) report that over the course of heat acclimation the sweat sodium concentration decreases; thus, despite a 12% increase of sweating rate, the sodium losses decreased by 59%. Therefore, for a given sweating rate the heat acclimated individual loses less solute from the plasma. It seems logical that for a given amount of body fluid loss via sweat, the greater amount of sodium retained in heat acclimated individual would cause more fluid to move from the intracellular to the extracellular compartment (as compared with an unacclimated individual) and perhaps cause a better defense of blood (plasma) volume when hypohydrated.

In the early 1930s, investigators from the Harvard Fatigue Laboratory found that for a given loss of body water, via sweating, the blood volume decreased more in winter than in summer (49). This observation was made on one subject who voluntarily dehydrated over an extended period by performing physical exercise in the desert. Figure 4 presents recent data (98,106) showing the magnitude of hypovolemia associated with hypohydration of 5% of body weight both before and after a 10-d heat acclimation program. These data were

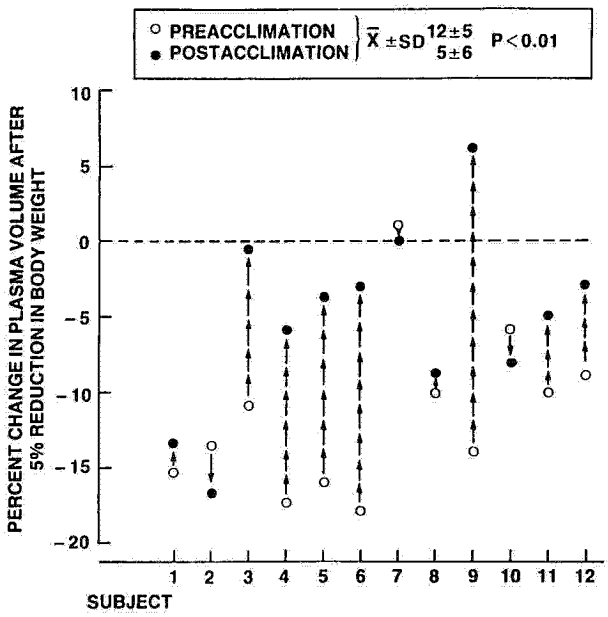

Figure 4—Magnitude of plasma volume reduction, while at rest, associated with a hypohydration of 5% body weight both before and after a heat acclimation program (data from ref. 106 as presented in ref. 98).

obtained while the individuals were resting; hypohydration was achieved by voluntary food and fluid denial combined with exercise in a hot environment. Subjects rested 15 h in a comfortable environment (while hypohydrated) prior to the measurements. An identical program of exercise-heat stress, but with full fluid replacement, was completed on the day prior to the euhydration measurements (*dotted line*). After the subjects were heat acclimated, there was a smaller plasma volume reduction for a given body water loss. It can be theorized that, since hypohydration was induced by exercise-heat stress, a more hypotonic sweat secretion in heat acclimated subjects resulted in a greater amount of solute remaining in the plasma that enabled redistribution of fluid from the intracellular compartment. Therefore, an individual's state of heat acclimation probably alters the magnitude of hypovolemia associated with hypohydration.

Figure 3 presents plasma volume responses for heat acclimated subjects when euhydrated and hypohydrated by 3%, 5%, and 7% of their body weight during rest and exercise (110). Note that plasma volumes were generally reduced with increased hypohydration, although there was some evidence of a plasma volume defense during the 7% hypohydration. The most important point from this figure is that the hypohydration mediated plasma volume reduction that occurs at rest continues throughout the subsequent light intensity (25% O_2max) exercise. In fact, the differences between the euhydration and hypohydration plasma volume values are greater during exercise than rest because of the small exercise hemodilution that occurs when subjects are euhydrated but not when hypohydrated (99,109,110).

There is evidence that plasma volume can be partially defended despite a progressive dehydration during intense (65–75% O_2max) running exercise (36,50,104). For example, Sawka and colleagues (104) showed that during 100 min of treadmill running, the plasma volume remained stable despite a 4% reduction in body weight. Likewise, Kolka et al. (58) reported that during a competitive marathon race the plasma volume remained stable despite a 7% reduction in body weight. Reasons for the stable plasma volume during intense exercise, despite progressive dehydration, might include the release of water from glycogen breakdown, metabolic water (90), and the redistribution of water from inactive skeletal muscle (98). The higher the exercise intensity, the greater the use of muscle glycogen as a metabolic substrate (16) and therefore the greater the water release from glycogen breakdown. Also, the greater amount of substrate oxidation, including fat, will result in a greater amount of metabolic water release.

Convertino (17) has speculated that the endocrine system could contribute to the redistribution of water into the intravascular space during intense exercise. The plasma concentration of angiotensin and vasopressin are increased in relation to the exercise intensity (18,19) and the magnitude of water deficit (9,34,35). Both of these hormones are potent vasoconstrictors, and their increased circulating concentrations causes vasoconstriction in inactive tissues. Vasoconstriction increases the ratio of pre- to post-capillary resistance, and favors fluid absorption from the inactive tissues (98). Therefore, during high-intensity exercise while hypohydrated, the elevated circulating concentration of these hormones favors additional fluid absorption from the inactive tissues. Regardless of the physiological mechanisms, exercise intensity may influence the availability of water for redistribution to the intravascular space.

Some athletes may use diuretics to reduce their body weight. Diuretics are drugs that increase the rate of urine formation and generally result in the loss of solutes (127). The commonly used thiazide (e.g., Diuril), carbonic anhydrase inhibitors (e.g., Diamox), and furosemide (e.g., Lasix) result in natriuresis. Diuretic-induced hypohydration generally results in an iso-osmotic hypovolemia, with a much greater ratio of plasma loss to body water loss than either exercise or heat induced hypohydration (51). As a result, with a diuretic-mediated loss of body water, there is not a solute excess in the plasma to exert an osmotic pressure for the redistribution of body water. Therefore, diuretic-induced hypohydration results in a relatively greater loss of extracellular and thus plasma water than sweat-induced hypohydration.

EXERCISE PERFORMANCE

Generally, body water deficits adversely influence exercise performance (105). The critical water deficit and magnitude of performance decrement are related to the environmental temperature and exercise task; the warmer the environment the greater the potential for exercise performance decrements (105). Prolonged aerobic exercise tasks are more likely to be adversely influenced by hypohydration than short-term anaerobic exercise tasks (105). In addition, the thermoregulatory advantages conferred by high aerobic fitness and heat acclimation are negated by hypohydration during exercise in the heat (11,12,106). Cognitive performance is also adversely influenced by body water deficits (1,38,62). For many complex athletic and industrial tasks, both the mental decision making and physiological function are closely related. As a result, hypohydration probably has a more profound effects of real-life tasks than on solely physiological performance measures.

Table 2 presents a summary of investigations concerning the influence of hypohydration on maximal aerobic power and physical work capacity (4,11,13, 24,46,50,88,96,126). In a temperate environment, a body water deficit of less than 3% body weight did not alter maximal aerobic power. Maximal aerobic power was decreased (11,13,126) in three of the five studies when hypohydration equaled or exceeded 3% body weight. Therefore, a critical water deficit (3% body weight) might exist before hypohydration reduces maximal aerobic power in a temperate environment. In a hot environment, Craig and Cummings (24) demonstrated that small (2% body weight) to moderate (4% body weight) water deficits resulted in large reduction of maximal aerobic power. Likewise, their data indicate a disproportionately larger decrease in maximal aerobic power with an increased magnitude of body water deficit. It seems that environmental heat stress has a potentiating effect on the reduction of maximal aerobic power elicited by hypohydration.

The physical work capacity for progressive intensity aerobic exercise was decreased during hypohydration in all of the studies presented in Table 2. Physical work capacity was decreased by marginal (1–2% body weight) water deficits that did not alter maximal aerobic power (4,13). The more pronounced the water deficit, the larger the reduction in physical work capacity. Clearly, hypohydration resulted in much larger decrements of physical work capacity in hot as compared with temperate environments (24,88). It appears that the thermoregulatory system, perhaps via increased body temperatures, has an important role in the reduced exercise performance mediated by a body water deficit.

A reduced maximal cardiac output might be the physiological mechanism by which hypohydration decreases an individual's maximal aerobic power and physical work capacity. Remember, hypohydration is associated with a decreased blood (plasma) volume during both rest and exercise. A decreased volume can increase blood viscosity (125) as well as possibly reduce venous return. During maximal exercise, a viscosity-mediated increased resistance and a reduced cardiac filling could decrease both stroke volume and cardiac output. Several investigators (3,119) have reported a tendency for reduced cardiac output for hypohydrated subjects during short-term moderate intensity exercise in temperate environments.

It is not surprising that environmental heat stress potentiates the hypohydration mediated reduction in maximal aerobic power. For euhydrated individuals, environmental heat stress alone decreases maximal aerobic power by ~7% (108). In the heat, the superficial skin veins reflexively dilate to increase the cutaneous blood flow and volume. The redirection of blood flow to the cutaneous vasculature could decrease maximal aerobic power by: (a) reducing the portion of cardiac output perfusing the contracting musculature or, (b) decreasing the effective central blood volume, central venous pressure, and thus reduce venous return and cardiac output. A hypohydrated person performing exercise in the heat would be hypovolemic and still have

TABLE 2. Hypohydration effects on maximal aerobic power and physical work capacity.

Study	Year	Dehydration Procedure	ΔWT	Test Environment	Exercise Mode	Maximal Aerotic Power	Physical Work Capacity
Armstrong et al.	1985	Diuretics	−1%	Neutral	TM	ND	↓ (6%)
Caldwell et al.	1984	Exercise,	−2%	Neutral	CY	ND	↓ (7 W)
		diuretics,	−3%	Neutral	CY	↓ (8%)	↓ (21 W)
		sauna	−4%	Neutral	CY	↓ (4%)	↓ (23 W)
Saltin	1964	Sauna, heat, exercise, diuretics	−4%	Neutral	CY	ND	↓ (?)
Pichan et al.	1988	Fluid restriction	−1%	Hot	CY	—	(↓6%)
			−2%	Hot	CY	—	(↓8%)
			−3%	Hot	CY	—	(↓20%)
Craig and Cummings	1966	Heat	−2%	Hot	TM	↓ (10%)	↓ (22%)
			−4%	Hot	TM	↓ (27%)	↓ (48%)
Buskirk et al.	1958	Exercise, heat	−5%	Neutral	TM	↓ (0.22 l·min^{-1})	—
Webster et al.	1988	Exercise in heat, sauna	−5%	Neutral	TM	↓ (7%)	↓ (12%)
Herbert and Ribisl	1971	?	−5%	Neutral	CY	—	↓ (17%)
Houston et al.	1981	Fluid restriction	−8%	Neutral	TM	ND	—

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