HYDRATION & AEROBIC PERFORMANCE: IMPACT OF ENVIRONMENT

Michael N. Sawka, PhD | School of Applied Physiology | Georgia Institute of Technology | United States of America
Samuel N. Cheuvront, PhD and Robert W. Kenefick, PhD | Thermal and Mountain Medicine Division | U.S. Army Research Institute | United States of America

KEY POINTS

- Athletes exercising in warm-hot conditions have high sweat rates that are a function of the metabolic intensity and environmental heat load.
- When sweat rates are high, ad libitum fluid consumption is often not sufficient to fully replace sweat losses ("voluntary dehydration") and results in cumulative body water deficits.
- A body water deficit of >2% of body mass (~3% of total body water for the average athlete) is defined as hypohydration.
- Hypohydration does not alter aerobic exercise performance in cold-cool environments, and sometimes impairs aerobic exercise performance in temperate conditions.
- Hypohydration usually impairs aerobic exercise performance in warm-hot environments.
- When skin temperature exceeds 27°C (81°F), hypohydration impairs aerobic performance by an additional ~1% for every 1°C (1.8°F) skin temperature elevation.

INTRODUCTION

Body water and electrolyte imbalances are common when performing strenuous physical exercise and/or during exposure to the environmental extremes of heat (Sawka et al., 2007), cold (Freund & Young, 1996) and high altitude (Hoyt & Honig, 1996). In warm-hot environments, high sweat rates may be sustained for many hours resulting in body water and electrolyte imbalances (Montain et al., 2006). During cold and high-altitude exposures, body water deficits are partially due to increased urine production. However, sweat rates can also be elevated while performing strenuous physical work in high-altitude, cold environments, due to high radiant heat loads (Gonzalez et al., 2012) and wearing heavy clothing or equipment (Young et al., 2000). When body water loss exceeds 2% of body mass, aerobic exercise performance can be impaired (Sawka et al., 2007; Sawka et al., 2015).

FLUID BALANCE AND BODY WATER

Water (total body water) is the principal chemical constituent of the human body. For an average young adult male, total body water represents 50–70% of body weight (Institute of Medicine, 2005). Variability in total body water is primarily due to differences in body composition. Lean body mass is ~73% water and fat body mass is ~10% water (Institute of Medicine, 2005). Thus for an average athlete, a body water deficit of 2% of body mass will be ~3% of total body water and ~5-10% of total body water is turned over daily, distributed via obligatory (non-exercise) fluid loss avenues.

Table 1 provides the sources of daily water losses and production for sedentary and active populations (Sawka et al., 2005). Metabolic water is formed by oxidation of substrates and is roughly offset by respiratory water losses. Urine output generally approximates 1–2 L (1.05–2.11 qt.) per day but can be markedly increased when consuming large volumes of fluid. This large capacity to vary urine output represents the primary avenue to regulate net body water and solute balance across a broad range of fluid intake volumes and losses from other avenues (Institute of Medicine, 2005). Sweat losses vary widely and depend upon the physical activity level and environmental conditions with ambient temperature, radiant heat load and high humidity, all markedly elevating sweating requirements (Gonzalez et al., 2009). Figure 1 provides an approximation of hourly sweat rates for athletes running at different speeds and exposed to different environmental conditions (Sawka, 1992). Sweat rates of >1 L/h (1.05 qt.) are common due to either high metabolic intensities and/or environmental heat stress.

Source	Loss (mL/d)	Production (M1/d)
Respiratory Loss	-250 to -350	
Urinary Loss	-500 to -1,000	
Fecal Loss	-100 to -200	
Insensible Loss	-450 to -1,900	
Metabolic Production		+250 to +350*
Total	**-1,300 to -3,450**	**+250 to +350**
Net Loss (Sedentary)	**-1,050 to -3,100**	
Sweat Losses in Various Sports	-455 to -3,630	
Net Loss (Athlete)	**-1,550 to -6,730**	

Table modified from Sawka et al. (2005). *Metabolic water production based on 2,500-3,000 kcal daily energy expenditure. Additional water production with exercise is assumed offset by parallel respiratory losses (as illustrated above with rest).
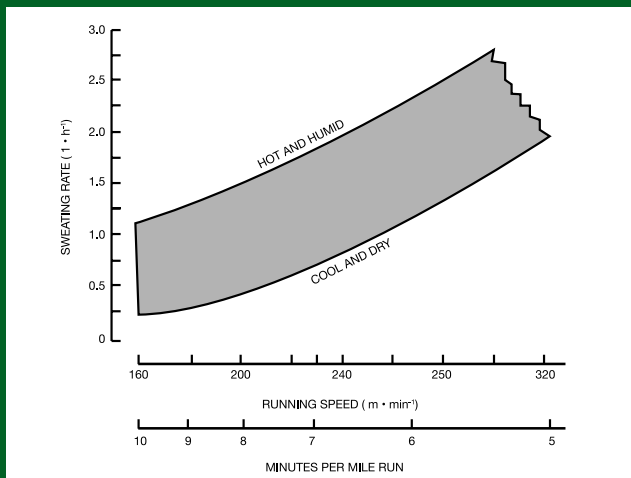
Table 1: Daily water losses and production.

Figure 1: Approximation of hourly sweat rates for runners at different paces and heat stress conditions (Sawka, 1992).

et al., 2004) and represent an approximate threshold (based on plasma volume reductions and plasma osmolality increases) where compensatory fluid regulatory actions occur (Cheuvront et al., 2013, 2014).
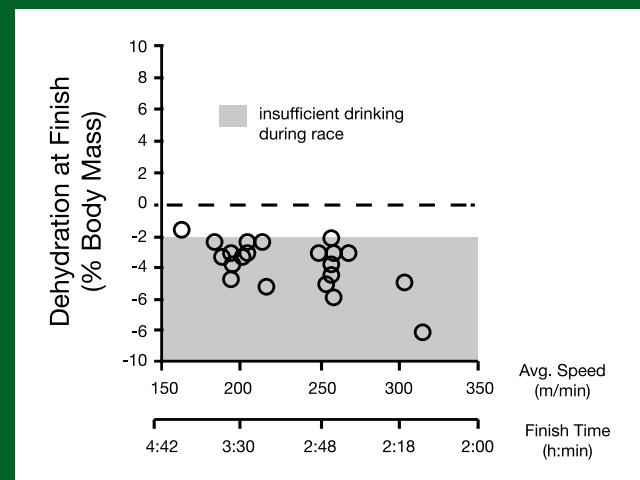

Figure 2: Plot of average running speed of finishing time for 42 km against the magnitude of post-race hypohydration when drinking ad libitum (Cheuvront et al., 2007).

Net body water balance (loss = gain) is regulated remarkably well on a day-to-day basis as a result of thirst and hunger drives, coupled with ad libitum access to food and beverage to offset water losses (Institute of Medicine, 2005). This is accomplished by an intricate interplay between neuroendocrine and renal responses to body water volume and electrolyte changes, as well as non-regulatory social-behavioral factors. These physiological homeostatic responses collectively ensure that small degrees of over- and under-hydration are readily compensated in the short term (Institute of Medicine, 2005).

During periods of high sweat rates (>1.0 L/h; 1.05 qt./h), humans practicing ad libitum drinking will often under-consume (Adolph, 1947; Adolph & Dill, 1938; Bean & Eichna, 1945; Greenleaf et al., 1983; Greenleaf & Sargent, II, 1965) and incur a body water deficit (Cheuvront & Haymes, 2001; Sawka et al., 2007). Figure 2 plots body water deficits incurred by runners practicing ad libitum drinking at different paces in marathon races contested across a range of cool-to-warm environmental conditions (Cheuvront et al., 2007). Note that most of the runners achieved body water deficits >2% of their initial body mass.

BODY WATER DEFICITS
Hypohydration is defined as a body water deficit greater than normal daily fluctuation (Cheuvront & Kenefick, 2014). Changes in hydration status can be assessed by a variety of body measures; however, they all have serious limitations (Cheuvront & Kenefick, 2014). Because of low measurement variability, change in body mass provides the most sensitive and simplest measure to determine acute changes in body water for all types of dehydration (Cheuvront et al., 2013; Cheuvront & Kenefick, 2014; Institute of Medicine, 2005; Sawka et al., 2007). Body water deficits >2% of body mass exceed two standard deviations in normal body mass variability (Adolph & Dill, 1938; Cheuvront

Incomplete fluid replacement decreases total body water, and as a consequence of free fluid exchange, affects each fluid space and will decrease blood (plasma) volume (Institute of Medicine, 2005). Plasma volume decreases because it provides the fluid for sweat, and as a result, osmolality increases because sweat is hypotonic (sodium poor) relative to plasma. The plasma hyperosmolality acts to pull fluid from the intracellular to the extracellular space to enable the defense of plasma volume when individuals become hypohydrated (Mack & Nadel, 1996). Use of diuretics (e.g., furosemide) for medical purposes increases urine formation and generally results in the loss of both electrolytes and water. Diuretic-induced hypohydration generally results in an iso-osmotic hypovolemia, with a much greater ratio of plasma loss relative to intracellular water loss that is typical of exercise or heat induced hypohydration (Cheuvront et al., 2013). Consistent with this, the environmental stressors of cold (Young et al., 1987) and high altitude (Hoyt & Honig, 1996) stimulate urine and electrolyte output, thus inducing an iso-osmotic hypovolemia (Cheuvront et al., 2013).

ENVIRONMENT AND AEROBIC PERFORMANCE
Ambient Temperature
During exercise in the heat, the most significant physiological burden is to support high skin blood flow for heat dissipation (Nybo et al., 2014; Sawka et al., 2011c). Skin temperature is elevated in proportion to ambient temperature and humidity. Figure 3A illustrates the generally linear relationship between ambient temperature and skin temperature (Adams, 1977) with a de novo 95% confidence interval calculation to illustrate the modifying effects of air flow and sun on

the grouped relationship. Skin temperatures will be elevated toward the high end of the confidence interval by high humidity, wearing uniforms/clothing that insulate and exposure to solar radiation. Skin temperatures will be reduced toward the lower end of the confidence interval by exposure to high air motion, which increases sweat evaporation. Warm-hot skin is associated with a greater skin blood flow and cutaneous venous compliance, which augments cardiovascular strain (Nybo et al., 2014; Sawka et al., 2011c). In general, the warmer the skin, the greater the skin blood flow response and the greater the heart rate elevation during exercise in the heat. The elevation in heart rate acts to reduce cardiac filling and stroke volume, thus providing a challenge to sustain blood pressure (Nybo et al., 2014; Sawka et al., 2011c). Therefore, during exercise in the heat with high sweat rates, there is the simultaneous problem of reduced plasma volume from dehydration in addition to elevated skin blood flow requirements. This dual perturbation (reduced plasma volume with increased skin blood flow) is likely an important physiological mechanism (via the cardiovascular system) contributing to impaired aerobic performance (Cheuvront et al., 2010; Cheuvront & Kenefick, 2014; Nybo et al., 2014; Sawka et al., 2015).

Figure 3: (Panel A) Approximate linear relationship between environmental conditions and skin temperature during aerobic exercise while wearing minimal clothing, redrawn from Adams (1977), dashed lines represent 95% confidence interval. **(Panel B)** Percent decrement in submaximal aerobic performance from euhydration as a function of skin temperature when hypohydrated by 4% of body mass from Sawka et al. (2011b).

It is generally accepted that heat stress alone will impair aerobic performance (Nybo et al., 2014), while cold stress alone does not impact aerobic performance unless the temperature is sufficient to adversely impair skeletal muscle function and nerve conduction (Sawka et al., 2011a). The earliest experiments regarding body water deficits and exercise capacity were conducted by the military and clearly concluded that in hot environments, replacement of fluids resulted in better sustainment of marching/military endurance performance in both laboratory and field trials (Adolph, 1947; Bean & Eichna, 1945; Ladell, 1955). Subsequent studies employing a variety of submaximal and maximal endurance test protocols have generally confirmed earlier observations (Cheuvront & Kenefick, 2014). The negative impact of hypohydration on aerobic performance is likely related to the environmental heat stress. During cold stress (2–10°C; 35–50°F) environments, hypohydration did not alter aerobic performance (Cheuvront et al., 2005; Kenefick et al., 2010). During temperate conditions (20–24°C; 68–75°F), hypohydration may (Cheuvront et al., 2005; Fallowfield et al., 1996; McConell et al., 1997; Merry et al., 2010) or may not (Kenefick et al., 2010; McConell et al., 1999; Oliver et al., 2007) impair aerobic performance. During warm-hot conditions (>25°C; >77°F) hypohydration usually (Below et al., 1995; Castellani et al., 2010; Cheung & McLellan, 1998; Ebert et al., 2007; Kenefick et al., 2010; Sawka, 1992; Walsh et al., 1994), but not always (Cheung et.al. 2015) impairs aerobic performance.

Figure 3B plots the impact of hypohydration on submaximal aerobic performance from several hypohydration studies (Castellani et al., 2010; Cheuvront et al., 2005; Kenefick et al., 2010) conducted in our laboratory (Sawka et al., 2011b). These studies employed similar procedures over a broad range of skin temperatures from 20–36°C (68–97°F). Segmented regression was used to approximate the statistical skin temperature threshold for performance impairment using individual study data points (n=53 paired observations). The threshold that best minimized the residual sums of squares was shown as 27.3°C (81°F) and warmer skin accentuated the performance impairment by ~1.5% for each additional 1°C (1.8°F) rise in skin temperature. Therefore, as ambient conditions become warmer, resulting in an elevation in cutaneous vasodilation, the adverse impact of hypohydration is clearly evident (Sawka et al., 2011b).

In addition to submaximal aerobic exercise performance, hypohydration has also been reported to consistently impair maximal intensity aerobic performance. Several review papers have addressed the impact of hypohydration on the impairment of maximal intensity aerobic exercise performance (Cheuvront & Kenefick, 2014; Sawka et al., 1984)

Terrestrial High Altitude
Physical exertion at high altitude likely induces sweat rates comparable to those at sea level for a given heat strain (Gonzalez et al.,
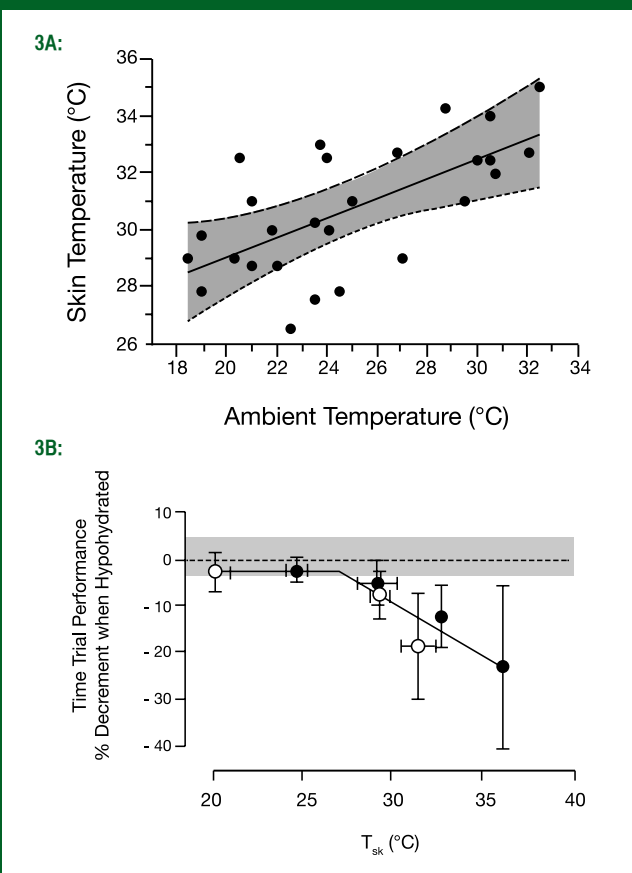
2013); however, respiratory water loss is elevated at high altitude (Hoyt & Honig, 1996). Further, with high-altitude exposure there is a proportionate diuretic reduction in plasma volume (Young & Sawka, 2002). Therefore, at high altitude hypohydration can occur from both sweat loss and adaptations to the environment.

Castellani and colleagues (2010) examined the impact of hypohydration (4% body mass loss) on submaximal aerobic performance at altitude. Their subjects performed time-trial tests in a warm environment (27°C; 80°F) when euhydrated (normal hydration) and hypohydrated at both sea level and in a hypobaric chamber to simulate high altitude (3,048 m; 10,000 ft.). They found that aerobic exercise performance when hypohydrated at sea level was impaired by 19% compared to that when euhydrated at sea level. Furthermore, aerobic exercise performance declined by 11% when euhydrated at high altitude and 34% when hypohydrated at high altitude. In addition, Girard and Racinais (2014) reported that exposure to heat and hypoxia (35°C, inspired oxygen concentration of 15%) resulted in a 51% decrement in a cycling to exhaustion task compared to control conditions (temperate/sea level), but also impaired performance compared to hot/sea level or temperate/altitude conditions. Therefore, altitude/hypohydration and altitude/heat exposure had additive effects on impairing performance.

MECHANISMS OF IMPAIRED AEROBIC PERFORMANCE

Hypohydration impairs aerobic performance when heat stress is present and this adverse impact is accentuated with high-altitude exposure. Heat stress is unique because it induces considerable cardiovascular strain to support skin blood flow requirements and hyperthermia (elevated skin and core temperatures). Table 2 briefly summarizes the physiological mechanisms impairing aerobic performance during heat stress. It is important to note that hypohydration exacerbates all of the proposed physiological mechanism(s) thought to limit aerobic performance from heat stress alone. However, it is clear that hypohydration-induced elevated cardiovascular strain is likely a primary critical factor needed to impair aerobic performance. In addition, the possible role of thirst has not been clearly defined (Cheung et al., 2015).

PRACTICAL IMPLICATIONS

- When it is hot outside and you are performing exercise or wearing heavy equipment, ensure you are well hydrated before starting exercise by monitoring your weight, urine and thirst (see SSE #97).
- When performing exercise, replace your sweat losses and do not dehydrate by more than 2% of your body weight.
- If an acute loss of ≥2% body mass occurs during endurance exercise, performance may be negatively affected.
- Regardless of hydration status, aerobic exercise performance

is impaired at altitude compared to sea level, while the negative effects on performance of heat and altitude are additive.
- Hypohydration does not alter aerobic exercise performance in cold-cool environments, and sometimes impairs aerobic exercise performance in temperate conditions.
- Hypohydration usually impairs aerobic exercise performance in warm-hot environments.
- When skin temperature exceeds 27°C (81°F), hypohydration impairs aerobic performance by an additional ~1% for every 1°C (1.8°F) skin temperature elevation.

Cardiovascular
e.g., Blood Pressure and Flow, Oxygen Delivery and Metabolite Removal
CNS & Neurobiological
e.g., Cerebral Metabolism, Neurotransmitter Levels, Temperature
Peripheral Muscular Factors
e.g., Temperature, Metabolic, Afferent Feedback
Psychological
e.g., Thermal Comfort, Rating of Perceived Exertion, Motivation and Expectations
Respiration
e.g., Hypocapnia, Alkalosis, Breathing Sensations

Table modified from Nybo et al., 2014. Where CNS refers to Central Nervous System.

Table 2: Physiological mechanisms potentially contributing to impaired exercise performance in warm-hot environments.

CONCLUSIONS

Hypohydration usually impairs aerobic performance during warm-hot conditions and high-altitude exposure will accentuate this performance impairment. The most important mechanism is likely the additive elevated cardiovascular strain induced from hypohydration, hyperthermia and hypoxia.

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