

## Evidence-Based Approach to Lingering Hydration Questions

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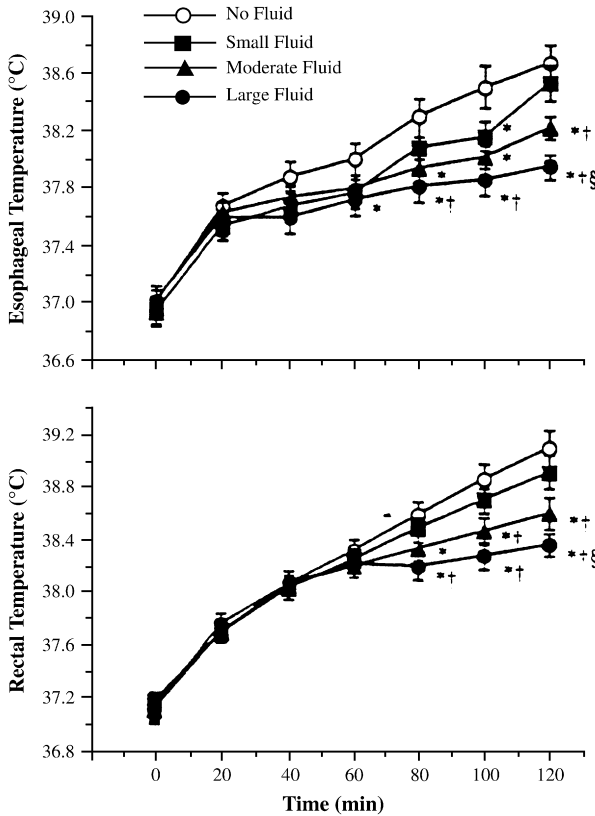
**S**tudies related to fundamental hydration issues have required clinicians to re-examine certain practices and concepts. The ingestion of substances such as creatine, caffeine, and glycerol has been questioned in regards to safety and hydration status. Reports of overdrinking (hyponatremia) also have brought into question the practices of drinking appropriate fluid amounts and the role that fluid-electrolyte balance has in the etiology of heat illnesses such as heat cramps. This article offers a fresh perspective on timely topics related to hydration, fluid balance, and exercise in the heat.

### CORE TEMPERATURE AND HYDRATION

Proper hydration is important for optimal sport performance [1] and may play a role in the prevention of heat illnesses [2]. Dehydration increases cardiovascular strain and increases core temperature ( $T_c$ ) to levels higher than in a state of euhydration [3]. These increases, independently [4] and in combination [3,5], impair performance and put an individual at risk for heat illness [6]. Exercise in the heat in which dehydration occurs before [3] or during exercise [7] results in  $T_c$  that is directly correlated ( $r = 0.98$ ) [7] with degree of dehydration (Fig. 1). The link between dehydration and hyperthermia has shown that independently and additively they result in cardiovascular instability that puts individuals at risk for heat exhaustion [3].

Despite laboratory evidence linking dehydration with increased  $T_c$ , some authors argue that this physiologic phenomenon does not occur in field settings [8–10]. This may be because field studies fail to control exercise intensity [8–11].  $T_c$  is driven by metabolic rate, and when the same subject is tested in a controlled laboratory environment, a higher metabolic rate produces a higher  $T_c$  [12]. Without controlling or measuring relative exercise intensity, a hydrated individual could exercise at a higher metabolic rate and drive his or her  $T_c$  to the same level as a dehydrated individual working at a lower intensity. Without

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**Fig. 1.** The degree of dehydration that occurs during exercise is correlated with the increase in esophageal (*top graph*) and rectal (*bottom graph*) temperatures. Subjects cycled for 120 minutes in a 33°C environment at approximately 65%  $\dot{V}O_{2max}$  while replacing 0% (No Fluid), 20% (Small Fluid), 48% (Moderate Fluid), or 81% (Large Fluid) of the fluid lost in sweat. Subjects lost 4.2%, 3.4%, 2.3%, and 1.1% body weight in the conditions. (From Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992;73(4):1340–50; with permission.)

a randomized crossover experimental design that controls exercise intensity, field studies cannot validly conclude that hydration is not linked to  $T_c$ .

Field studies disputing relationships between  $T_c$  and dehydration also cite that laboratory studies use environments that are too hot, and that the physiologic relationship does not exist in temperate environments (approximately 23°C) often associated with field studies [8]. Laboratory studies have shown that the increase of  $T_c$  with dehydration is exacerbated in hot environments, but still observed in cold environments (8°C) [13]. Dehydration impairs thermoregulation independent of ambient conditions, but the effect is seen especially at high ambient temperatures when the thermoregulatory system is

more heavily stressed. Laboratory-based studies have clearly shown that when exercise intensity and hydration state are controlled,  $T_c$  increases at a faster rate when subjects are dehydrated [7].

## CAFFEINE

Caffeine and its related compounds, theophylline and theobromine, have long been recognized as diuretic molecules [14], which encourage excretion of urine via increased blood flow to the kidneys [15]. The recommendation that caffeine be avoided by athletes because hydration status would be compromised [6] is based on several studies examining the acute effects of high levels (>300 mg) of caffeine [16]. More recent studies have tested the credibility of this recommendation by re-examining hydration status in varying settings after short-term caffeine intake and, for the first time, after long-term intake.

Using increased urine output as an indicator of diuresis and dehydration, early studies showed that the threshold for an increase of urine output was 250 to 300 mg of caffeine intake [17]. Urine output was greater for the first 3 hours after ingestion [17], but when urine was collected for 4 hours, the difference in urine output between caffeine and placebo was negated [18]. When double the caffeine was ingested (612 mg or 8.5 mg/kg), urine volume increased over the next 4 hours [19]. The molecular properties of caffeine do not refute the fact that it may act as an acute diuretic, but when observations span a short time (<24 hours), it is difficult to understand long-term changes in hydration [15].

When 24-hour urine volume is examined, the ingestion of caffeine at levels of 1.4 to 3.1 mg/kg does not increase urine output or change hydration status [20]. When large amounts of caffeine are ingested (8.2–10.2 mg/kg), the increases in urine excretion vary from person to person, but may be 41% greater than control levels [21]. It cannot be concluded from these studies that “caffeine causes dehydration” because acute increases in urine volume with large caffeine intake (>300 mg) may be offset later by decreased urine output and result in no change in long-term hydration status [16].

Acute ingestion of caffeine before exercise (1–2 hours) at levels up to 8.7 mg/kg does not alter urine output and fluid balance [19,22–24] when subjects exercise at 60% to 85%  $VO_{2max}$  for 0.5 to 3 hours [19,22–24]. The possible mechanism for a lack of a diuretic effect with caffeine during exercise is most likely due to an increase in catecholamines and diminished renal blood flow [19]. There is little evidence to suggest that short-term use of caffeine alters hydration status at rest or during exercise.

Because most Americans consume caffeine on a regular basis [15], it is surprising that few studies have examined the effects of controlled caffeine intake over several days. In 2004, the authors' research team conducted a field study involving a crossover design in which subjects exercised for 2 hours, twice a day, for 3 consecutive days [25]. Subjects rehydrated ad libitum and consumed a volume equal to 7 cans daily of either caffeinated or decaffeinated soda. Throughout the 3 days, no differences of urine volume, body weight, plasma volume, and urine specific gravity were observed between the two

conditions. The authors reported similar results in an investigation in which subjects consumed 3 mg caffeine/kg/d for 6 days; during the following 5 days, 20 subjects decreased their intake to 0 mg/kg/d, 20 maintained intake at 3 mg/kg/d, and 20 doubled their intake to 6 mg/kg/d [26]. Urine volume and other markers of hydration status showed that, regardless of caffeine ingestion, hydration status did not change throughout the 11 days (Fig. 2). Heat tolerance and thermoregulation examined on the 12th day during exercise in a hot environment did not differ between conditions [27].

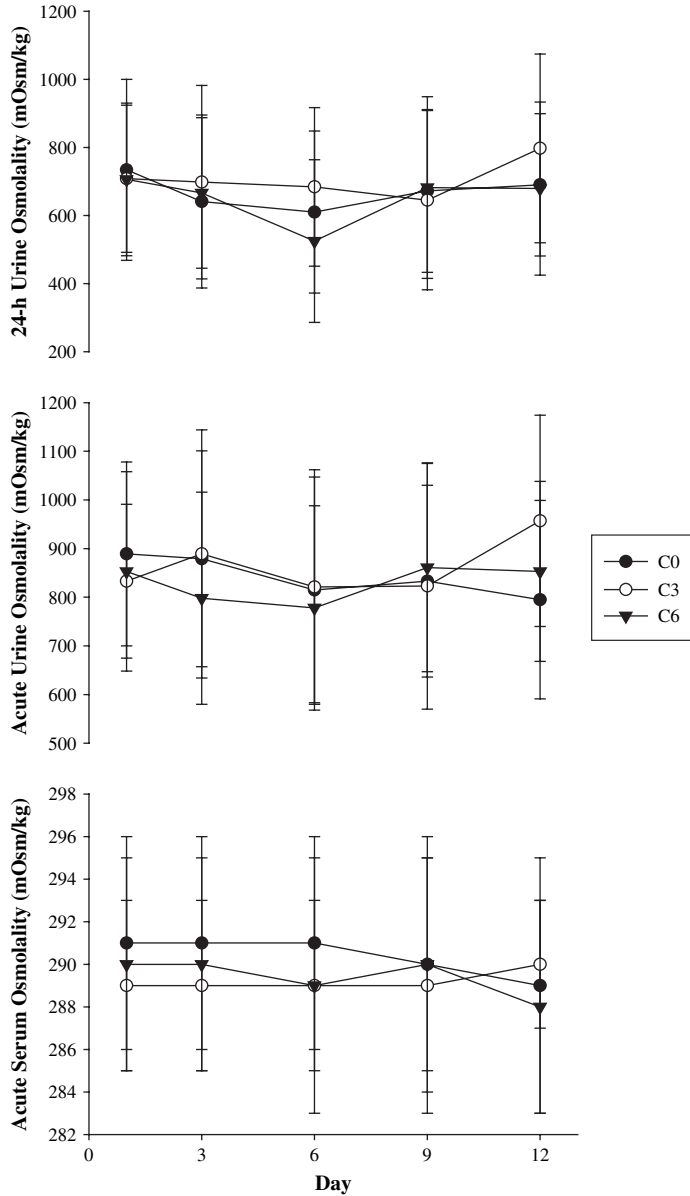
Acute ingestion of moderate to low levels of caffeine (<300 mg) does not promote dehydration at rest or during exercise. Long-term ingestion of low to high levels of caffeine does not compromise hydration status and thermoregulation at rest and during exercise. Varying one's level of caffeine ingestion (either increasing or decreasing) also does not seem to change hydration status [15,16]. There is no evidence to support caffeine restriction on the basis of impaired thermoregulation or changes of hydration status at levels less than 300–400 mg/d.

## **HYPONATREMIA**

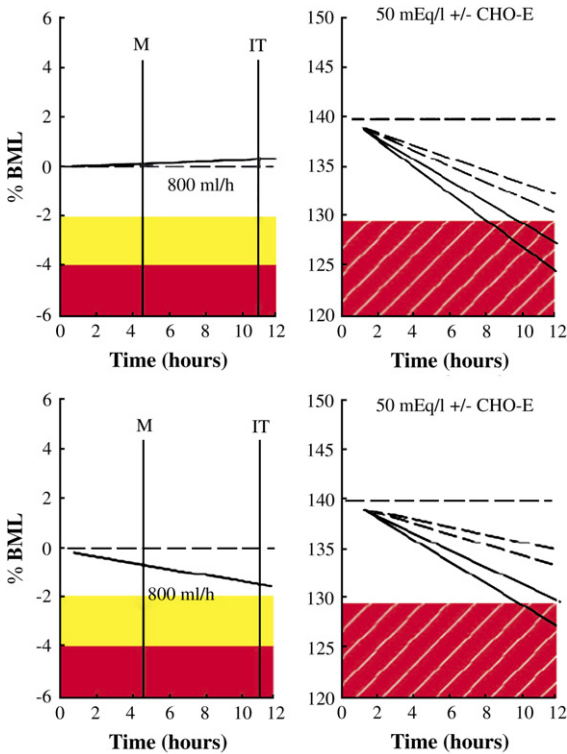
Hyponatremia has received attention in the media as a result of its occurrence in popular road running races [28]. Hyponatremia is a serious complication of low plasma sodium levels (<130 mEq/L) [29]. The exact cause is likely multifaceted and circumstantial [30]. Hyponatremia has been observed in exercising individuals who became dehydrated [31,32], maintained hydration [32], and became overhydrated [31,32]. Asymptomatic hyponatremia is the most common type of hyponatremia [32] and is defined as a decrease in sodium level (<130 mEq/L) that occurs in the absence of life-threatening symptoms [33]. Asymptomatic hyponatremia per se is not harmful or detrimental to performance [34]. When plasma sodium decreases to less than 125 mEq/L, hyponatremic illness may occur. Hyponatremic illness is a medical emergency that is symptomatic and requires immediate medical treatment [32,33,35].

Overdrinking, identified as an increase in body mass, significantly increases one's risk for developing hyponatremia and should be avoided [32,35,36]. Some observational studies have found that increased dehydration results in higher sodium levels [31,32,37], but this does not mean that dehydration prevents hyponatremia. The increased risk of heat illnesses associated with dehydration does not warrant dehydration as a method for preventing hyponatremia. High sweat rates or sodium-concentrated sweat may lead to large losses of sodium and put one at risk for hyponatremia, especially in events lasting more than 3 hours [38]. It is recommended that one should ingest fluid at a rate that closely matches fluid loss (ie,  $\leq 2\%$  body weight loss) [39].

Replacing large fluid losses with equal amounts of pure water may dilute the plasma sodium level [36], so it has been suggested that replacement of electrolytes can be achieved through sports drinks or salt tablets [30,34]. Mathematical modeling has shown that in a variety of conditions the ingestion of sodium may attenuate the decline of serum sodium over time (Fig. 3) [40]. However, recent



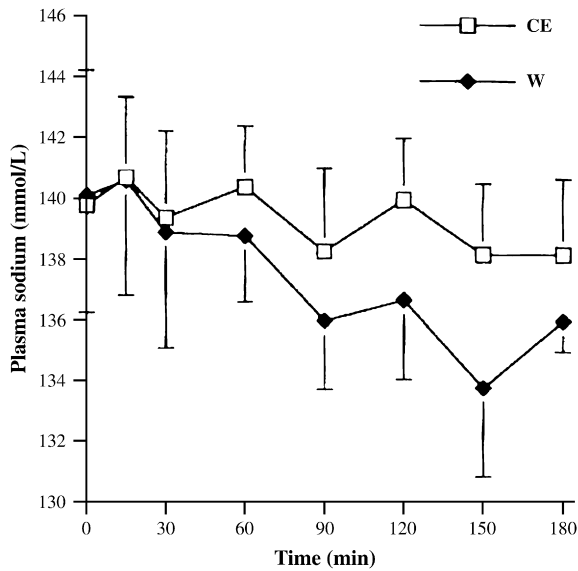
**Fig. 2.** Controlled consumption of caffeine at a level of 3 mg/kg/d for 6 days and then decreased to 0 mg/kg/d (C0), maintained at 3 mg/kg/d (C3), or increased to 6 mg/kg/d (C6); none of these conditions altered hydration status. Urine osmolality (top graph) and volume (data not shown) during repeated 24-hour collection periods did not change over the course of the investigation. Acute urine (middle graph) and serum (bottom graph) osmolality also did not differ as a result of the level of caffeine consumption. (Data from Armstrong LE, Pumerantz AC, Roti MW, et al. Fluid, electrolyte, and renal indices of hydration during 11 days of controlled caffeine consumption. *Int J Sport Nutr Exerc Metab* 2005;15(3):252–65.)



**Fig. 3.** Predicted effectiveness of a carbohydrate-electrolyte sports drink (CHO-E) containing 17 mEq/L of sodium and 5 mEq/L of potassium for attenuating the decline in plasma sodium concentration (mEq/L) expected for a 70-kg person drinking water at 800 mL/h when running 10 km/h in cool (18°C; upper panel) and warm (28°C; lower panel) environments. The solid shaded areas depict water loss that would be sufficient to diminish performance modestly and substantially. The hatched shaded area indicates the presence of hyponatremia. *M* indicates the finishing time for the marathon run. *IT* indicates the approximate finishing time for an ironman triathlon. For the sodium figures, the solid lines reflect the effect of drinking water only, and hatched lines illustrate the effect of consuming the same volume of a sports drink. The pair of lines of similar type represent the predicted outcomes when total body water accounts for 50% and 63% of body mass. BML, body mass loss. (From Montain SJ, Chevront SN, Sawka MN. Exercise associated hyponatremia: quantitative analysis to understand the etiology. *Br J Sports Med* 2006;40(2):98–105; with permission.)

studies involving consumption of sodium through sports drinks and salt tablets have confirmed [30,34,41] and refuted [37,42,43] this relationship (Fig. 4). Some of these differences in results may lie in methodologic differences, [30] assumptions, and conflicting conclusions [44].

Understanding the etiology and cause of hyponatremia may help to understand its prevention better. It is well agreed that overconsumption of fluids is the primary, but not the only, cause [35,40]. Whether replacement of sweat losses with equal volumes of sodium-containing beverages would prevent or



**Fig. 4.** Ingestion of a carbohydrate-electrolyte beverage (CE) slightly attenuated the decline of plasma sodium observed with ingestion of plain water (W) over 180 minutes of exercise at a moderate intensity in a hot environment (34°C). [Adapted from Vrijens DM, Rehrer NJ. Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. *J Appl Physiol* 1999;86(6):1847–51; with permission.]

attenuate hyponatremia is still debated [35]. More studies that look at varying environmental conditions, sweat rates, and body masses may help shed light on this complex picture. Some authorities have suggested that allowing dehydration would prevent hyponatremia because the contraction of extracellular fluid would increase sodium concentration. Until further studies are conducted, promoting dehydration (ie, >2% of pre-exercise weight) is not warranted and may put some individuals at greater risk for exertional heat illnesses and could compromise performance [2].

## CREATINE

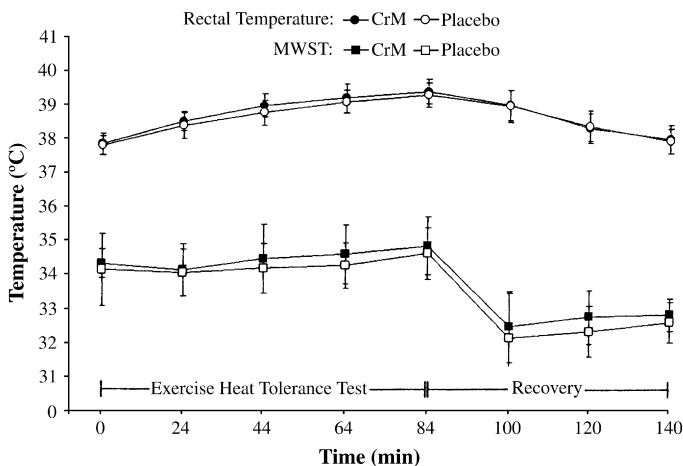
Creatine is one of the most popular nutritional supplements on the market. Athletes of all levels and varieties of sports are using it in hopes of gaining a competitive edge. During creatine supplementation, 90% of the increase in body weight (0.7–2.0 kg) is accounted for by increases of total body water (TBW) [45]. The increase of TBW during the “loading phase” results from increases of intracellular water stores [46], but prolonged use of creatine results in TBW increases in all body fluid compartments [45]. Some authors speculate that creatine use while exercising in the heat impairs heat tolerance and may be a contributing factor for heatstroke [47,48]. Those authors propose that

creatine increases one's risk for heat injury because the increases of intracellular water stores deplete intravascular volume [49]. Before any published conclusive studies concerning creatine's effect on hydration status and use in the heat, the American College of Sports Medicine published a consensus statement stating that "high-dose creatine supplementation should be avoided during periods of increased thermal stress . . . there are concerns about the possibility of altered fluid balance, and impaired sweating and thermoregulation . . ." [48].

Paradoxically, studies using short-term and long-term creatine supplementation have shown that subjects exercising in the heat (30–37°C) for 80 minutes have either no change or an advantageous lower heart rate and  $T_c$  [46,50–52]. Work from our laboratory also has shown that creatine supplementation does not alter exercise heat tolerance, even when subjects begin exercise in a dehydrated state (Fig. 5) [51]. One study that found lower  $T_c$  with creatine use during exercise in heat suggests that the increases of TBW with supplementation may hyperhydrate the body and lower  $T_c$  [46]. Despite early concerns about creatine supplementation and exercise in the heat [48], more recent studies have shown conclusively that heat storage does not increase as a result of creatine use [46,50–52]. There is no evidence to support restriction of creatine use during exercise in the heat.

## EXERCISE-ASSOCIATED CRAMPS

Although the exact mechanism is unknown, skeletal muscle cramps are associated with numerous congenital and acquired conditions, including hereditary



**Fig. 5.** The use of creatine monohydrate (CrM) does not compromise exercise heat tolerance. After becoming dehydrated, rectal temperature and mean weighted skin temperature (MWST) had similar responses in CrM and placebo treatments when subjects exercised in the heat and recovered in a cool environment. (From Watson G, Casa D, Fiala KA, et al. Creatine use and exercise heat tolerance in dehydrated men. *J Athl Train* 2006;41(1):18–29; with permission.)

disorders of carbohydrate and lipid metabolism, diseases of neuromuscular and endocrine origins, fluid and electrolyte deficits (ie, owing to diarrhea or vomiting), pharmacologic agents (ie,  $\beta$ -agonists, ethanol, diuretics), and toxins [53]. The medical treatments for these various forms of muscle cramps are as varied as their etiologies. McGee [54] specifically classified leg muscle cramps as contractures (ie, electrically silent cramps caused by myopathy or disease), tetany (ie, sensory plus motor unit hyperactivity), dystonia (ie, simultaneous contraction of agonist and antagonist muscles), or true cramps (ie, motor unit hyperactivity). The last category includes skeletal muscle cramps that are due to heat, fluid-electrolyte disturbances, hemodialysis, and medications.

The International Classification of Diseases [55] defines heat cramps, a form of motor unit hyperactivity, as painful involuntary contractions that are associated with large sweat (ie, water and sodium) losses. Heat cramps occur most often in active muscles (ie, thigh, calf, and abdominal) that have been challenged by a single prolonged event (ie, >2–4 hours) or during consecutive days of physical exertion. A high incidence of heat cramps occurs among tennis players [56], American football players [57], steel mill workers [58], and soldiers who deploy to hot environments [59,60]. These activities result in a large sweat loss, consumption of hypotonic fluid or pure water, and a whole-body sodium and water imbalance [59,61]. The distinctions between heat cramps and other forms of exercise-associated cramps are subtle [54,59,62], but sodium replacement usually resolves heat cramps effectively [56,59,61–63]; successful treatment via sodium administration confirms a preliminary diagnosis of heat cramps.

Bergeron [62] described a tennis player who was plagued by recurring heat cramps. This athlete secreted sweat at a rate of 2.5 L/h and had a sweat sodium ( $\text{Na}^+$ ) concentration of 83 mEq/L. This sweat  $\text{Na}^+$  concentration is high, in that most heat-acclimatized athletes exhibit 20 to 40 mEq  $\text{Na}^+$ /L of sweat (ie, heat acclimatization reduces sweat  $\text{Na}^+$  concentration), but occurs naturally in a small percentage of humans. During 4 hours of tennis match play, this young athlete lost 10 L of sweat and a large quantity of electrolytes (ie, 830 mEq of  $\text{Na}^+$ ; 19,090 mg of  $\text{Na}^+$ ; 48.6 g of sodium chloride). Given that the average sodium chloride intake of adults in the United States is 8.7 g (3.4 g  $\text{Na}^+$ ) per day, it is not difficult to see how this athlete could experience a whole-body  $\text{Na}^+$  deficit. To offset his 4-hour sodium chloride loss in sweat, this athlete would require 1.6 L of normal saline, 7.8 to 9.8 cans of canned soup (85–107 mEq per can), 12.6 servings of tomato juice (66 mEq of  $\text{Na}^+$  per serving), or 39.5 to 127.7 L of a sport drink (6.5–21 mEq  $\text{Na}^+$ /L). These options are unreasonable. A long history of heat cramps ended when this tennis player began consuming supplemental salt during meals. Other tennis players have been successfully treated using a similar course of action [63].

In 2004, the authors' research team evaluated a female varsity basketball player (body mass 78.5 kg, height 187 cm) who experienced exercise-induced cramps during the winter months in New England, with signs and symptoms

identical to heat cramps. The authors measured her sweat rate as 1.16 L/h, her sweat sodium concentration (ie, via whole-body washdown) as 42 mEq/L, and her daily consumption of sodium. These values were normal and typical of winter sport athletes. Three days of observations indicated that her dietary intake of  $\text{Na}^+$  per day was similar to her daily sweat  $\text{Na}^+$  loss (ie, both 3200–3600 mg). Because she did not train or compete in a hot environment, the authors hesitated to diagnose her malady as heat cramps. When she began ingesting supplemental sodium (ie, by liberally salting each meal at midseason), however, the skeletal muscle cramps resolved permanently. This case suggests that a history of skeletal muscle cramps, with a large daily  $\text{Na}^+$  turnover owing to a high sweat rate, indicates the need for an evaluation of whole-body  $\text{Na}^+$  balance. It further suggests that heat cramps may have been named because they usually occur in hot environments, but they also may occur in mild environments when sweat  $\text{Na}^+$  concentration and sweat losses are large.

A study by Stofan and colleagues [57] examined the link between sweat sodium losses and heat cramps. Sweat rate, sodium content, and percent body weight loss were measured on a single day of a “two-a-day” practice in subjects who had a history (episode within the last year) of severe heat cramps. Although heat cramps were not observed, football players with a history of heat cramps had sweat sodium losses two times greater than matched controls. Although the exact etiology of heat cramps may be unknown, sodium deficits seem to contribute to their development. In most cases, restoration and compensation of sodium losses seems to prevent further heat cramps.

## FLUID NEEDS AND HYDRATION PLAN

Water losses during exercise should be replaced at a rate equal to (not greater than) the sweat rate [39]. Loss of sweat during exercise needs to be replaced after exercise, but dehydration ( $\geq 2\%$  body weight) during exercise can be detrimental to performance in part by increases in  $T_c$ . It is difficult to replace 100% of fluid loss during exercise, especially if it occurs in hot environments for long durations or if sweat loss is great [11,39]. Authorities have suggested that a minimal amount of dehydration ( $< 2\%$  body weight) may be tolerated without compromising performance [64]. Regardless, knowledge of sweat rate is necessary to develop a hydration plan (Table 1) [65], but without this it has been recommended to ingest 200 to 300 mL every 10 to 20 minutes [6]. Thirst lags behind changes in hydration (termed *voluntary dehydration*) [66]. When individuals have high sweat rates, and large volumes of fluid cause gastrointestinal stress, it may be advantageous for them to train themselves to tolerate consumption of fluids at a rate similar to their sweat losses [67].

In attempts to optimize endurance performance in the heat, glycerol has been used to increase TBW. It is an osmotically active molecule that acutely ( $< 4$  hours) increases TBW stores [68]. Although using glycerol plus water is an effective prehydration strategy, it does not increase sweat rate or reduce performance time or  $T_c$  in a race setting [69]. Using glycerol as a part of

a rehydration strategy between exercise bouts increases exercise time to exhaustion in the heat (37°C). The increase is likely due to increases of plasma volume, not because of cardiovascular effects, thermoregulatory effects, or differences in fluid-regulating hormones [70]. It is generally accepted that glycerol, although a hyperhydrating agent, is not an ergogenic aid in most situations [64]. Future research should examine the importance of timing in glycerol ingestion for performance benefits.

When multiple, dehydrating exercise sessions are occurring over a short time (ie, two workouts per day in football or track and field), athletes must rehydrate immediately and quickly between bouts. Intravenous rehydration has been used in the belief that direct administration of fluid into the central circulation optimally replaces lost fluid. Contrary to this belief, when hydrating with equal amounts of intravenous and oral fluid ingestion, intravenous is not superior to restore plasma volume after dehydration [71]. Oral rehydration results in better cardiovascular stability, lower  $T_c$ , rating of perceived exertion, thirst, and thermal sensation than intravenous rehydration. However, these changes do not translate into improved exercise time to exhaustion [71,72]. Regardless, oral hydration is preferred (versus intravenous) for individuals who would be exercising subsequently in the heat [71,72]. An exception occurs when large amounts of fluid must be replaced in a short time, and gastric emptying and intestinal absorption rates may limit the ingestion of fluids orally. In such cases, a combination of intravenous and oral rehydration may be warranted so that fluid requirements are met, and the oropharyngeal reflex is stimulated [73].

Athletes often supplement with glycerol or choose to use intravenous rehydration because of the difficulty of matching fluid intake with fluid losses during intense exercise in the heat. This makes theoretical sense given the possibility of large sweat rates (ie, >1.5 L/h) and the likelihood that fluid consumption could not match the sweat rate given gastric emptying and intestinal absorption rates, especially when the ingestion must occur when the exercise is intense. An individualized rehydration plan that considers sweat rate, the semantics of the actual competition parameters, and personal preferences and tolerance is recommended to ensure that rehydration is optimized in these circumstances [65]. When the individualized rehydration plan is practiced and rehearsed in practices and preliminary competitions, the need for glycerol and intravenous rehydration will likely be eliminated because of the benefits associated with the “rehydration training,” and ultimately the degree of dehydration would be minimized [65,74].

## SUMMARY

Hydration status affects exercise performance in the heat and may influence the development of exertional heat illnesses. However, numerous factors that influence hydration state are not understood by the public. Field-based studies may lead athletes to believe that  $T_c$  is not influenced by hydration, but these studies contradict well-controlled laboratory experiments. For many years, recommendations have been published that active individuals should avoid caffeinated

**Table 1**  
Self-testing program for optimal hydration\*

1. Make sure you are properly hydrated *before* the workout—your urine should be pale yellow
2. Do a warmup run until you begin to sweat, then stop. Urinate if necessary
3. Weigh yourself naked on a floor scale (accurate to 0.1 kg)
4. Run for 1 h at an intensity similar to your targeted race or training run
5. Drink a measured amount of a beverage during the run, if you are thirsty. It is important that you measure exactly how much fluid you consume during the run
6. Do not urinate until post-body weight is recorded
7. Weigh yourself naked again on the same scale you used in step 3
8. You may now urinate and drink fluids as needed. Calculate your fluid need using the following formula

A. Enter your body weight from step 3 in Kg (To convert from lb to kg, divide lb by 2.2)			_____
B. Enter your body weight from step 7 in Kg (To convert from lb to kg, divide lb by 2.2)			_____
C. Subtract B from A	A - B =		_____
D. Convert your total in step C to g by multiplying by 1000	C × 1000 =		_____
E. Enter the amount of fluid consumed during the run in mL (To convert from oz to mL, multiply oz by 30)			_____
F. Add E to D	E + D =		_____

This final figure is the number of ml that you need to consume per hour to remain well hydrated. If you want to convert mL back to oz, divide by 30

\*This table may be used to calculate the amount of fluid needed during an exercise bout to remain hydrated.

Adapted from Casa D. Proper hydration for distance running—identifying individual fluid needs. *Track Coach* 2004;167:5321–8; with permission.

beverages with little supporting scientific evidence. Research from the authors' laboratory shows that long-term intake of moderate levels of caffeine does not compromise hydration status. Hyponatremia also has received a lot of attention, but until more is known about its etiology and prevention, it is recommended that athletes drink an amount of fluid to minimize dehydration (but not overdrink). The use of creatine as an ergogenic aid initially was overshadowed by questions regarding its safety during exercise in the heat. Research shows no reason for these concerns. Although the mechanism of heat cramps is still not fully understood, it seems that deficits in sodium from sweating and/or diet is a predisposing factor. The reader is encouraged to read thorough review articles on these topics [64,75]. Ultimately, clinical practice should be dictated by evidence in the literature and not perpetuate unproven myths.

## References

- [1] Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc* 1985;17(4):456–61.

- [2] Binkley HM, Beckett J, Casa DJ, et al. National Athletic Trainers' Association position statement: exertional heat illnesses. *J Athl Train* 2002;37(3):329–43.
- [3] Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, et al. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. *J Appl Physiol* 1997;82(4):1229–36.
- [4] Gonzalez-Alonso J, Teller C, Andersen SL, et al. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 1999;86(3):1032–9.
- [5] Gonzalez-Alonso J. Separate and combined influences of dehydration and hyperthermia on cardiovascular responses to exercise. *Int J Sports Med* 1998;19(Suppl 2):S111–4.
- [6] Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *J Athl Train* 2000;35(2):212–24.
- [7] Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992;73(4):1340–50.
- [8] Laursen PB, Suriano R, Quod MJ, et al. Core temperature and hydration status during an ironman triathlon. *Br J Sports Med* 2006;40(4):320–5.
- [9] Godek SF, Bartolozzi AR, Burkholder R, et al. Core temperature and percentage of dehydration in professional football linemen and backs during preseason practices. *J Athl Train* 2006;41(1):8–17.
- [10] Sharwood KA, Collins M, Goedecke JH, et al. Weight changes, medical complications, and performance during an ironman triathlon. *Br J Sports Med* 2004;38(6):718–24.
- [11] Godek SF, Godek JJ, Bartolozzi AR. Hydration status in college football players during consecutive days of twice-a-day preseason practices. *Am J Sports Med* 2005;33:843–51.
- [12] Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. *Human performance physiology and environmental medicine at terrestrial extremes*. Traverse City: Cooper Publishing Group; 1988. p. 97–151.
- [13] Gonzalez-Alonso J, Mora-Rodriguez R, Coyle EF. Stroke volume during exercise: interaction of environment and hydration. *Am J Physiol Heart Circ Physiol* 2000;278(2):H321–30.
- [14] Eddy NB, Downs AW. Tolerance and cross-tolerance in the human subject to the diuretic effect of caffeine, theobromine, and theophylline. *J Pharmacol Exp Ther* 1928;33:167–74.
- [15] Armstrong LE. Caffeine, body fluid-electrolyte balance, and exercise performance. *Int J Sport Nutr Exerc Metab* 2002;12(2):189–206.
- [16] Maughan RJ, Griffin J. Caffeine ingestion and fluid balance: a review. *J Hum Nutr Dietet* 2003;16:411–20.
- [17] Robertson D, Frolich JC, Carr RK, et al. Effects of caffeine on plasma renin activity, catecholamines and blood pressure. *N Engl J Med* 1978;298(4):181–6.
- [18] Passmore AP, Kondowe GB, Johnston GD. Renal and cardiovascular effects of caffeine: a dose-response study. *Clin Sci (Lond)* 1987;72(6):749–56.
- [19] Wemple RD, Lamb DR, McKeever KH. Caffeine vs caffeine-free sports drinks: effects on urine production at rest and during prolonged exercise. *Int J Sports Med* 1997;18(1):40–6.
- [20] Grandjean AC, Reimers KJ, Bannick KE, et al. The effect of caffeinated, non-caffeinated, caloric and non-caloric beverages on hydration. *J Am Coll Nutr* 2000;19(5):591–600.
- [21] Neuhauser B, Beine S, Verwied SC, et al. Coffee consumption and total body water homeostasis as measured by fluid balance and bioelectrical impedance analysis. *Ann Nutr Metab* 1997;41(1):29–36.
- [22] Graham TE, Hibbert E, Sathasivam P. Metabolic and exercise endurance effects of coffee and caffeine ingestion. *J Appl Physiol* 1998;85(3):883–9.
- [23] Falk B, Burstein R, Rosenblum J, et al. Effects of caffeine ingestion on body fluid balance and thermoregulation during exercise. *Can J Physiol Pharmacol* 1990;68(7):889–92.
- [24] Gordon NF, Myburgh JL, Kruger PE, et al. Effects of caffeine ingestion on thermoregulatory and myocardial function during endurance performance. *S Afr Med J* 1982;62(18):644–7.

- [25] Fiala KA, Casa DJ, Roti MW. Rehydration with a caffeinated beverage during the nonexercise periods of 3 consecutive days of 2-a-day practices. *Int J Sport Nutr Exerc Metab* 2004;14(4):419–29.
- [26] Armstrong LE, Pumerantz AC, Roti MW, et al. Fluid, electrolyte, and renal indices of hydration during 11 days of controlled caffeine consumption. *Int J Sport Nutr Exerc Metab* 2005;15(3):252–65.
- [27] Roti MW, Casa DJ, Pumerantz AC, et al. Thermoregulatory responses to exercise in the heat: chronic caffeine intake has no effect. *Aviat Space Environ Med* 2006;77(2):124–9.
- [28] Almond CS, Shin AY, Fortescue EB, et al. Hyponatremia among runners in the Boston Marathon. *N Engl J Med* 2005;352(15):1550–6.
- [29] Armstrong LE. Exertional hyponatremia. In: Armstrong LE, editor. *Exertional heat illnesses*. Champaign (IL): Human Kinetics; 2003. p. 103–35.
- [30] Vrijens DM, Rehrer NJ. Sodium-free fluid ingestion decreases plasma sodium during exercise in the heat. *J Appl Physiol* 1999;86(6):1847–51.
- [31] Speedy DB, Noakes TD, Kimber NE, et al. Fluid balance during and after an ironman triathlon. *Clin J Sport Med* 2001;11(1):44–50.
- [32] Speedy DB, Noakes TD, Rogers IR, et al. Hyponatremia in ultradistance triathletes. *Med Sci Sports Exerc* 1999;31(6):809–15.
- [33] Armstrong LE. Exertional hyponatremia. *J Sports Sci* 2004;22(1):144–5.
- [34] Twerenbold R, Knechtle B, Kakebeeke TH, et al. Effects of different sodium concentrations in replacement fluids during prolonged exercise in women. *Br J Sports Med* 2003;37(4):300–3.
- [35] Hew-Butler T, Almond C, Ayus JC, et al. Consensus statement of the 1st international exercise-associated hyponatremia consensus development conference, Cape Town, South Africa 2005. *Clin J Sport Med* 2005;15(4):208–13.
- [36] Weschler LB. Exercise-associated hyponatremia: a mathematical review. *Sports Med* 2005;35(10):899–922.
- [37] Hew-Butler TD, Sharwood K, Collins M, et al. Sodium supplementation is not required to maintain serum sodium concentrations during an ironman triathlon. *Br J Sports Med* 2006;40(3):255–9.
- [38] Montain SJ, Sawka MN, Wenger CB. Hyponatremia associated with exercise: risk factors and pathogenesis. *Exerc Sport Sci Rev* 2001;29(3):113–7.
- [39] 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(1):i–vii.
- [40] Montain SJ, Chevront SN, Sawka MN. Exercise associated hyponatremia: quantitative analysis to understand the aetiology. *Br J Sports Med* 2006;40(2):98–105.
- [41] Baker LB, Munce TA, Kenney WL. Sex differences in voluntary fluid intake by older adults during exercise. *Med Sci Sports Exerc* 2005;37(5):789–96.
- [42] Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: effects of water, saline, or no fluid. *Med Sci Sports Exerc* 1991;23(7):811–7.
- [43] Speedy DB, Thompson JM, Rodgers I, et al. Oral salt supplementation during ultradistance exercise. *Clin J Sport Med* 2002;12(5):279–84.
- [44] Weschler LB, Rehrer NJ. What can be concluded regarding water versus sports drinks from the Vrijens-Reher experiments? *J Appl Physiol* 2006;100(4):1433–4.
- [45] Powers ME, Arnold BL, Weltman AL, et al. Creatine supplementation increases total body water without altering fluid distribution. *J Athl Train* 2003;38(1):44–50.
- [46] Kilduff LP, Georgiades E, James N, et al. The effects of creatine supplementation on cardiovascular, metabolic, and thermoregulatory responses during exercise in the heat in endurance-trained humans. *Int J Sport Nutr Exerc Metab* 2004;14(4):443–60.
- [47] Bailes JE, Cantu RC, Day AL. The neurosurgeon in sport: awareness of the risks of heatstroke and dietary supplements. *Neurosurgery* 2002;51(2):283–8.

- [48] Terjung RL, Clarkson P, Eichner ER, et al. American College of Sports Medicine roundtable: the physiological and health effects of oral creatine supplementation. *Med Sci Sports Exerc* 2000;32(3):706–17.
- [49] Demant TW, Rhodes EC. Effects of creatine supplementation on exercise performance. *Sports Med* 1999;28(1):49–60.
- [50] Kern M, Podewils LJ, Vukovich M, et al. Physiological response to exercise in the heat following creatine supplementation. *J Exerc Physiol* 2001;4:18–27.
- [51] Watson G, Casa D, Fiala KA, et al. Creatine use and exercise heat tolerance in dehydrated men. *J Athl Train* 2006;41(1):18–29.
- [52] Weiss CA, Powers ME, Horodyski MB. Creatine supplementation does not alter the physiological response to exercise in the heat. *J Athl Train* 2003;38(2):S29.
- [53] Schweltnus MP, Derman EW, Noakes TD. Aetiology of skeletal muscle ‘cramps’ during exercise: a novel hypothesis. *J Sports Sci* 1997;15(3):277–85.
- [54] McGee SR. Muscle cramps. *Arch Intern Med* 1990;150(3):511–8.
- [55] American Medical Association. International classification of diseases: manual of the international statistical classification of diseases, injuries, and causes of death, 9th revision. Chicago: AMA; 1998.
- [56] Bergeron MF. Heat cramps during tennis: a case report. *Int J Sport Nutr* 1996;6(1):62–8.
- [57] Stofan JR, Zachwieja JJ, Horswill CA, et al. Sweat and sodium losses in NCAA football players: a precursor to heat cramps? *Int J Sport Nutr Exerc Metab* 2005;15(6):641–52.
- [58] Talbot JH. Heat cramps. *Medicine*. Baltimore: Williams and Wilkins; 1935. p. 323–76.
- [59] Hubbard RW, Armstrong LE. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, editors. *Human performance physiology and environmental medicine at terrestrial extremes*. Traverse City: Cooper Publishing Group; 1988. p. 305–59.
- [60] Armstrong LE. Considerations for replacement beverages: fluid-electrolyte balance and heat illness. In: *Fluid replacement and heat stress*. Washington, DC: National Academy Press; 1993. p. 37–54.
- [61] Ladell WSS. Heat cramps. *Lancet* 1949;2:836–9.
- [62] Bergeron MF. Exertional heat cramps. In: Armstrong LE, editor. *Exertional heat illnesses*. Champaign (IL): Human Kinetics; 2003. p. 91–102.
- [63] Bergeron MF. Heat cramps: fluid and electrolyte challenges during tennis in the heat. *J Sci Med Sport* 2003;6(1):19–27.
- [64] Coyle EF. Fluid and fuel intake during exercise. *J Sports Sci* 2004;22(1):39–55.
- [65] Casa D. Proper hydration for distance running—identifying individual fluid needs. *Track Coach* 2004;167:5321–8.
- [66] Armstrong LE, Hubbard RW, Szlyk PC, et al. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. *Aviat Space Environ Med* 1985;56(8):765–70.
- [67] Rehrer NJ. Fluid and electrolyte balance in ultra-endurance sport. *Sports Med* 2001;31(10):701–15.
- [68] Riedesel ML, Allen DY, Peake GT, et al. Hyperhydration with glycerol solutions. *J Appl Physiol* 1987;63(6):2262–8.
- [69] Wingo JE, Casa DJ, Berger EM, et al. Influence of a pre-exercise glycerol hydration beverage on performance and physiologic function during mountain-bike races in the heat. *J Athl Train* 2004;39(2):169–75.
- [70] Kavouras SA, Armstrong LE, Maresh CM, et al. Rehydration with glycerol: endocrine, cardiovascular, and thermoregulatory responses during exercise in the heat. *J Appl Physiol* 2006;100(2):442–50.
- [71] Castellani JW, Maresh CM, Armstrong LE, et al. Intravenous vs. oral rehydration: effects on subsequent exercise-heat stress. *J Appl Physiol* 1997;82(3):799–806.
- [72] Casa DJ, Maresh CM, Armstrong LE, et al. Intravenous versus oral rehydration during a brief period: responses to subsequent exercise in the heat. *Med Sci Sports Exerc* 2000;32(1):124–33.

- [73] Figaro MK, Mack GW. Regulation of fluid intake in dehydrated humans: role of oropharyngeal stimulation. *Am J Physiol* 1997;272(6 Pt 2):R1740–6.
- [74] Murray BM. Training the gut for competition. *Curr Sports Med Rep* 2006;5:161–4.
- [75] Casa DJ, Clarkson PM, Roberts WO. American College of Sports Medicine roundtable on hydration and physical activity: consensus statements. *Curr Sports Med Rep* 2005;4(3):115–27.