

Updated Fluid Recommendation: Position Statement From the International Marathon Medical Directors Association (IMMDA)

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Controversy exists regarding optimal fluid guidelines for athletes engaging in different sports. Most published recommendations emphasize the detrimental consequences of dehydration,^{1–4} while more recent reports warn of the morbid consequences of hyperhydration.^{5–7} Accordingly, individualized recommendations emphasizing a balance between the two extremes have evolved.^{8–10} These revised guidelines, however, continue to promote static recommendations for what in reality are very dynamic athletic situations.

Marathon running epitomizes a dynamic situation that requires a constant adjustment to constantly changing homeostatic requirements. Real-time assessments of fluid and sodium homeostasis are physiologically represented by changes in plasma osmolality (POsm). Pituitary arginine vasopressin (AVP) secretion is stimulated when POsm increases by only 1% to 2%, representing the body's attempt to prevent dehydration by decreasing kidney water excretion. After maximal antidiuresis is achieved, thirst is then stimulated to replace water losses that are in excess of the ability of AVP-stimulated antidiuresis to conserve body water. In general, the osmotic threshold for thirst is 5 to 10 mOsm/g H₂O higher than that of AVP secretion. Thus, thirst is

stimulated with decreases in body water of approximately 1.7% to 3.5%.^{11–13} Concomitant performance decrements and cardiovascular strain are also documented when baseline body fluid losses exceed approximately 2%.⁴

The failure of athletes to replace 100% of body-weight losses from ad libitum fluid intake has been well-described as “involuntary”^{14,15} or “voluntary” dehydration;^{16–18} this phenomenon has resulted in the devaluation of thirst as a “poor” indicator of body fluid needs. The combination of laboratory with recent field data (see Table 1 and subsequent discussion), however, suggests that the body primarily defends POsm, and not blood or extracellular fluid volume during prolonged endurance exercise. Because it is well established that thirst is stimulated in response to changes in tonicity in most land mammals,^{11,13,19–22} it seems reasonable to conclude that thirst would be the predominant physiologic and dynamic regulator governing fluid balance during exercise.

This article will focus on the physiology of normal fluid balance as the ultimate guide toward the evolution of optimal fluid recommendations. Six physiologic considerations of fluid and sodium balance will be detailed followed by 6 practical recommendations. The neuroendocrine regulation of homeostasis will be emphasized in defense of the behavioral drives for thirst and sodium palatability during dynamic activities such as prolonged endurance exercise. The behavioral drives to maintain fluid and sodium balance are evolutionary stable, essential for the safety and survival of the species, and deeply rooted within the human genetic makeup.²³

PHYSIOLOGIC CONSIDERATIONS OF FLUID AND SODIUM BALANCE

1. *Bodyweight is not an accurate surrogate for body fluid volume during prolonged exercise:* The field of exercise science promotes fluid replacement at 100% of bodyweight losses under the premise that sweat loss is an accurate surrogate for changes in body fluid volume.^{1,2,4,8,24–28} This recommendation—to externally defend bodyweight during exercise—remains entrenched in the literature despite initial observations in 1932 stating that voluntary water consumption replaced only 56% of sweat lost during exercise.²⁹ Research on ad libitum fluid intakes over the last 74 years have repeatedly verified that

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humans, when given free access to fluids, replace no more than 75% of net water losses during physical activity.^{15-17,30-32} Abdominal distress, nausea and vomiting have been reported in highly trained distance runners who have tried to match fluid intakes to sweat rates.^{17,33} The ongoing discrepancy between thirst, satiation and the inability to inherently maintain bodyweight during prolonged endurance activity suggests that bodyweight is only a marker of body fluid homeostasis and is not regulated in itself. The remarkable capacity of the body to maintain circulating plasma volume despite large deficits in bodyweight due to “sweat losses” during prolonged endurance exercise exemplifies the complex regulation of body fluid volume and distribution³⁴ that are unrelated to the maintenance of bodyweight.

2. *Blanket fluid guidelines with fixed ranges will not protect the diverse population currently participating in athletic events:* The American College of Sports Medicine's (ACSM) current guidelines promote a fluid intake of 600 to 1200 mL/h; a range largely based on laboratory studies performed on elite male athletes.¹ The popularity of marathon running increased markedly at the time these guidelines were released, with charity running groups enticing more recreational runners into the sport. Slower, more inexperienced, female athletes heeded the upper limit of the ACSM guidelines and developed exercise-associated hyponatremia (EAH) by ingesting more fluid than they could excrete over the course of a marathon run.^{35,36} In 2001, the International Marathon Medical Directors Association (IMMDA) approved guidelines lowering the “acceptable” range to 400 to 800 mL/h to adjust the upper limit and protect smaller athletes (mainly female) from overdrinking.⁷

Recently, mathematical models have proposed that these ranges may not be ideal to cover the current population of runners participating in marathon events.³⁷ Close inspection of a marathon field today illustrates the variety of shapes, sizes and speeds that must be taken into consideration when formulating fluid intake guidelines. For example, the average weight of 7299 runners registering for the 2005 Comrades Marathon was 73 kg. The lightest competitor weighed 43 kg whereas the heaviest runner weighed 119 kg. The fastest runner averaged 16.4 km/h (winning time 5:27) whereas the slowest runners ran at a 7.4 km/h pace (the official 12-h cut-off pace). The 11,728 runners who completed the 89.2 km Comrades Marathon ran, on average, 49 minutes slower during the second half of the race than they ran the first half of the race with a minimum and maximum range of -11 (negative split) and 180 minutes (positive split), respectively. The wide variation between split times exemplifies the late-race functional deterioration that has been described elsewhere³⁸ and highlights the fact that modern marathon running is a dynamic situation, including participants with widely diverse physical attributes and fitness levels. An even wider spread of running speeds and bodyweights would be expected in a standard 42.2 km marathon, as the Comrades Marathon is more than double the distance, requires a qualifying

time for entry, has a defined cut-off and is held over a grueling terrain; all of which precludes the participation of unfit and novice runners. Conversely, relatively unfit and untrained runners can often complete a standard 42 km marathon, amplifying the wide range of participant profiles.

The 3 main factors governing fluid loss during exercise are mass (bodyweight), running speed (metabolic rate), and ambient temperature.^{26,27} Race day temperatures in the New York City Marathon have varied between 1 and 29°C and have fluctuated as much as 17°C from start to finish.³⁹ Therefore, it would seem very unlikely that ANY single “range” could successfully encompass this wide spectrum of weather conditions, bodyweights, and running speeds that characterize modern day marathon running.

3. *Drinking to thirst will preserve POsm within the normal range and maintain intracellular volume and homeostasis:* Thirst is a subjective sensation characterized by a deep-seated desire for water^{11,20,23} and is generally associated with oral sensations such as dryness, irritation, and an “unpleasant taste” in the mouth.^{21,22,40} Thirst has been quantified using geometric and visual analog scales^{11,21,40,41} and documented to increase when POsm is 5 to 10 mOsm/kg above the threshold for AVP secretion; well within the normal physiologic range of ~280 to 295 mOsm/kg H₂O.^{11,22,42}

POsm is maintained within this narrow range to protect intracellular volume. Serum sodium concentrations [Na⁺] mirror POsm⁴³ because sodium is the principal solute of extracellular fluid; thus [Na⁺] and POsm are viewed as interchangeable in this discussion, although the appropriate calculation is: POsm = 2 × [Na⁺] + [BUN] + [glucose] (all in mmol/L).¹³

The maintenance of intracellular volume is of vital importance for cell function and survival. Hypertonicity causes intracellular dehydration; a bodyweight loss of 5.5% during prolonged severe exercise elicits a reduction in red cell volume by 3.2%.⁴⁴

A reduced intracellular volume can reduce the rates of glycogen and protein synthesis⁴⁵ and hypertonicity beyond serum sodium concentrations of 160 mmol/L can cause encephalopathy and death.⁴⁶ Conversely, hypotonicity causes intracellular expansion. Although high cell volume can stimulate glycogen and protein synthesis, excessive and acute cell swelling from serum sodium levels below 125 mmol/L can lead to hyponatremic encephalopathy, noncardiogenic pulmonary edema, and death.⁴⁷

Thirst and AVP secretion are intimately related: synergistically lowering elevations in POsm by stimulating fluid intake and promoting antidiuresis. The threshold for AVP release (~280 to 285 mOsm/kg H₂O) is normally set 5-10 mOsm/kg H₂O below that for thirst (~290 to 295 mOsm/kg H₂O), despite significant variation between individual set points.¹¹ This evolutionary design liberates individuals from constantly seeking water, as thirst is only stimulated when antidiuresis is maximal and hypertonicity exceeds the capacity of the kidneys to cope with rising tonicity. The strength of an intact thirst mechanism is

exemplified in patients with the disorder of diabetes insipidus, either due to inadequate AVP secretion or an impaired kidney response to AVP, where a normal POsm is maintained by stimulated thirst despite the excretion of up to 20 L of fluid per day.¹¹ Oropharyngeal metering^{48–50} and stomach fullness^{21,22,33} provide inhibitory feedback to terminate drinking more rapidly than the return of POsm to normal levels, perhaps as a safety measure to prevent overdrinking.

The strength and precision of the thirst mechanism has been demonstrated both at rest and during exercise in young adults. In one study involving seven men an increase in POsm, in response to hypertonic saline infusion, promoted a dipsogenesis proportional to the increase in POsm; with 82% of the total volume of water consumed within the first 5 minutes of rehydration with the immediate cessation of intake due to “stomach fullness.”²² In a separate fluid deprivation study, 65% of total water intake was consumed during the first 2.5 minutes of rehydration in 5 males.²¹ Subjective ratings of dry mouth and plasma protein concentrations returned to baseline levels in 2.5 minutes, plasma volume returned to baseline levels within 5 minutes and serum sodium concentrations began to decrease within 5 minutes; returning to baseline values within 12.5 minutes. This initial drinking bout was also interrupted by sensations of “stomach fullness”. During prolonged endurance exercise, 8 female and 10 male athletes responded to graded levels of hypohydration with ad libitum fluid intakes sufficient enough to restore POsm, replace sweat losses, and attenuate thermal and circulatory strain.^{17,30,31}

Thus, humans respond to increases in serum $[Na^+]$ with AVP secretion, thirst and water intake that occur when POsm is still well within normal ranges. The precision and magnitude of the thirst drive is related to ancestral areas of the brain that are associated with vegetative functions such as the hunger for air and food, micturition and pain.⁵¹ To assume that the thirst drive would be an “inaccurate index” of fluid balance during exercise (the dynamic homeostatic imbalance alternatively described as the “flight or fight” response) would seem contradictory to the evolution of our species.

Although there is concern that thirst due to intracellular dehydration or extracellular volume depletion may be confused with the sensations of a dry mouth (xerostomia) during exercise, thirst due to xerostomia has not been shown to lead to severe polydipsia.⁴¹ The sensation of a dry mouth can promote more frequent drinking episodes, but total fluid intake is similar, or counter regulated by increased urinary output, in tested clinical and experimental settings.⁵² Recent studies on xerostomia, thirst, and interdialytic weight gain have not factored in osmolality and therefore do not provide convincing evidence that a dry mouth without changes in POsm can contribute to excessive fluid intakes or to hyponatremia from fluid overload without inappropriate AVP secretion.^{53–56}

4. *The body defends POsm and volume over bodyweight. “Involuntary dehydration” is a homeostatic*

mechanism designed to protect POsm and cellular volume in response to hypotonic sweat losses: The reluctance of man to voluntarily replace fluids at amounts equivalent to bodyweight losses during exercise was recognized in 1933, when Dill³² hypothesized that humans only drunk as much fluid to maintain constant osmolality of the extracellular fluid. This “failure” of man to replace 100% of body fluid losses, generally using bodyweight as a surrogate marker of body fluid volume, has been subsequently recognized as “voluntary” or “involuntary” dehydration; this has had the effect of minimizing the importance of thirst as an adequate indicator of body fluid needs.^{14–16}

Actual field study data from multiple endurance studies indicates that the maintenance of bodyweight will actually lead to reductions in serum sodium concentrations from prerace to postrace (Table 1). Most athletes who lose less than 2% of bodyweight experience a decrease in serum $[Na^+]$.^{57,58} Athletes who gain weight progress from normonatremia to hyponatremia.⁵⁹ In contrast, POsm is maintained within normal ranges (± 3 mmol/L) when percent bodyweight losses are between 2% and 4%.^{60–67} Whereas most athletes who lose more than 4% of bodyweight demonstrate increases in serum $[Na^+]$ above normal ranges.^{44,68–71} Thus, the cumulative data in Table 1 suggest that the body defends POsm over body fluid volume, as reflected by changes in bodyweight, during prolonged endurance activity lasting between 2.7 and 38.2 hours, and can maintain POsm and therefore intracellular volume, best between 2% and 4% cumulative bodyweight losses. Acute bodyweight changes below 2% or above 4% cannot be compensated for internally, and may lead to dysregulation of serum $[Na^+]$ with resultant clinical symptomatology.

Concordant with these field data, 3 laboratory studies replacing fluids to 100% of bodyweight losses confirm a reduction in serum sodium concentrations during prolonged endurance exercise lasting between 2 and 6 hours.^{72–74} Although these decreases in serum sodium concentrations were generally within normal physiologic ranges, fluid replacement at or above 100% does not seem to offer any performance benefits.^{73,75}

Advocates of replacing 100% of bodyweight losses argue that cardiovascular drift—the downward drift in central venous pressure and stroke volume with concomitant rise in heart rate—commences with a 1% decrease in bodyweight losses.⁷⁶ Subsequent studies normalizing blood volume while inducing hypohydration have confirmed, however, that cardiac drift during exercise can and often does result from factors other than hypovolemia.^{76,77} The corresponding increase in heart rate with progressive dehydration is accompanied by a proportional decrease in stroke volume which serves to maintain cardiac output.⁷⁷ These reductions in stroke volume are more related to elevations in core temperature, circulating catecholamine concentrations and heart rate than due to the redistribution of blood flow or circulating blood volume.⁷⁸ Only at degrees of dehydration beyond $\sim 3\%$ is cardiac output significantly

TABLE 1. Field Study Data: Serum Sodium and Bodyweight Change

Study	Race Distance (km)	[Na ⁺] Prerace (mmol/L)	[Na ⁺] Postrace (mmol/L)	[Na ⁺] Change (Prepost Race)	Bodyweight Loss (%)	Finish Time (h)
Twerenbold et al ⁵⁹ (N = 13)	~41 run	137 ± 1	133 ± 2	-4	2 ± 1 weight gain	4
Glance et al ⁵⁷ (N = 13)	160 run	144	140	-4	1	26
Gerth et al ⁵⁸ (N = 51)	100 run	137 ± 5	131 ± 2	-6	1	14
Hew-Butler (unpublished) (N = 33)	109 cycle	139 ± 3	138 ± 3	-1	2 ± 1	5 ± 1
Stuempfle et al ⁶¹ (N = 20)	161 snow race	141 ± 1	138 ± 2	-3	2	38 ± 7
Nelson et al ⁶² (N = 45)	42 run	139 ± 0	142 ± 0	3	3	4
Refsum et al ⁶³ (N = 41)	90 ski	142	141	-1	3	8
Cohen and Zimmerman ⁶⁴	42 run	139 ± 2	142 ± 2	3	3	3
Noakes and Carter ⁶⁵ (N = 13)	160 run	144 ± 1	140 ± 3	-4	3	16 ± 6
Kavanagh and Shephard ⁶⁶ (N = 9)	42 run	146 ± 2	148 ± 2	2	3	4 ± 1
Hew-Butler (unpublished) (N = 82)	56 run	139 ± 3	138 ± 3	-1	4 ± 1	6 ± 1
Hew-Butler et al ⁶⁰ (N = 413)	226 triathlon	141 ± 2	141 ± 3	0	4 ± 2	12 ± 2
Maron et al ⁶⁷ (N = 6)	42 run	141 ± 1	141 ± 1	0	4 ± 1	3 ± 0
Gastmann et al ⁶⁸ (N = 9)	453 triathlon	138 ± 4	133 ± 4	-5	5	26
Rocker et al ⁶⁹	42 run	144	149	5	5	3
Beckner and Winsor ⁷⁰	42 run	141	156	7	5	No time
Riley et al ⁷¹	32-42 run	143 ± 1	148 ± 1	5	5	4
Astrand and Saltin ⁴⁴ (N = 6)	85 ski	144	152	8	6	9

diminished as a result of a reduction in circulating blood volume.^{78,79} This bodyweight loss exceeds the threshold level at which AVP secretion and thirst are generally stimulated.

Equations predicting ad libitum fluid intake from multiple regression analyses identify POsm as the primary factor driving voluntary fluid intake during exercise or under stressful environmental conditions; with plasma volume and subjective symptoms of secondary importance.^{15,40} Thus, under acute situations involving sweat sodium losses, the body will defend osmolality and intracellular volume through fluid shifts from the extracellular (primarily interstitial) fluid compartments⁸⁰⁻⁸² until ~4% bodyweight losses through sweating accrue; after which intracellular fluid volume is compromised and an external fluid supply is necessary.

Athletes rehydrating with either water or an electrolyte-containing beverage restore 68% and 82% of fluid losses, respectively, after an exercise-induced dehydration of 2.3%.¹⁴ Changes in free water clearance followed changes in POsm and rehydration with only water restored POsm to control (isotonic) levels. Conversely, extracellular fluid space was fully restored only through consumption of the electrolyte-containing beverage. Ad libitum fluid intake did not match 100% bodyweight losses during 3 hours of rehydration with either beverage (hence the "involuntary" dehydration), illustrating the immediate defense of the body to preserve POsm first and only secondarily intravascular volume.

Metabolic water formation, from the combustion of fat and carbohydrate combined with the liberation of glycogen-bound water, may contribute an internal water source to offset sweat losses and sustain internal fluid balance during prolonged or intense physical activity.⁸³⁻⁸⁵ The activation or inactivation of internal sodium stores has also been hypothesized as a mechanism aiding in the defense of POsm during prolonged endurance exer-

cise.^{86,87} Data from 2135 weighed competitive athletic performances demonstrate that athletes who overhydrate, underhydrate, or euhydrate during prolonged endurance races generally maintain serum sodium concentrations within normal ranges (Fig. 1 reprinted with permission).⁸⁶ Despite this wide variation in bodyweight changes, 80% of the athletes in this large and diverse pool maintained POsm and intracellular volume during prolonged continuous athletic activity.

Thus, the combination of laboratory work with field data requires a re-examination of the phrase "voluntary dehydration" to more accurately reflect the preservation of POsm (and therefore intracellular volume) over extracellular fluid (ECF) and plasma volume, secondary to sweat NaCl losses during endurance exercise. The body does *not* defend ECF and plasma volume over POsm during an acute exercise bout, and the immediate replacement of body fluids at 100% of bodyweight losses is not beneficial to performance. AVP secretion and thirst are stimulated when POsm increases 1% to 4%; well within the physiologic ranges of homeostatic compensation. Performance and cardiovascular decrements are clearly documented when bodyweight losses exceed 4%; however, this range is well beyond the protective capacity of the synergistic AVP and thirst mechanisms, whose primary physiologic interaction is to preserve osmolality within relatively narrow physiologic ranges. Only after POsm is stabilized will the body then activate mechanisms to *gradually* restore plasma volume by increasing sodium consumption and fluid intake over the next 24 hours.^{20,88}

5. *Plasma volume contraction, resulting from the preservation of POsm during exercise, will be restored over a period of 24 hours by hormonal mechanisms which will increase the palatability for sodium-containing foods and beverages. When sodium is ingested, plasma volume will gradually return to baseline levels:* The primary rationale for the inclusion of sodium into rehydration beverages is

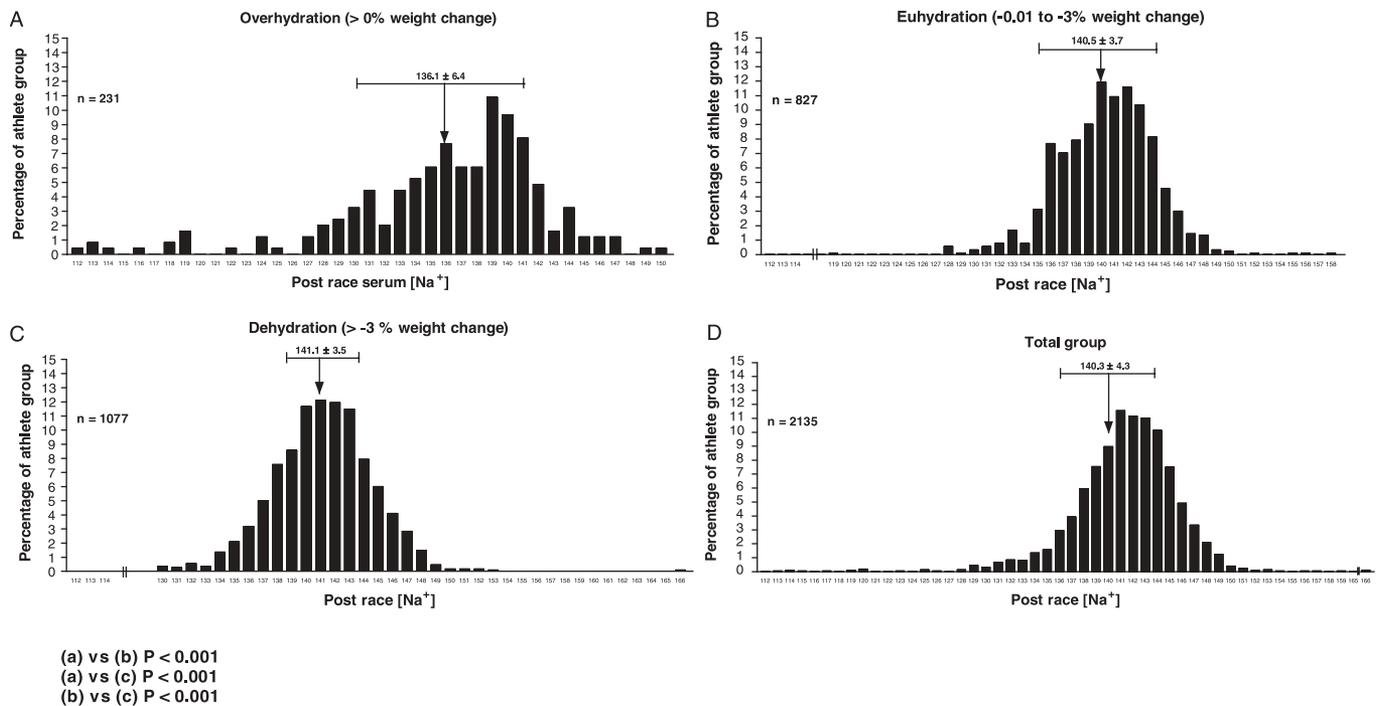


FIGURE 1. Distributions of postrace $[Na^+]$ in 2135 overhydrated, euhydrated, and dehydrated athletes. Reprinted with permission from T. D. Noakes.⁸⁶

to aid in the restoration of plasma volume⁴⁵ rather than prevent the development of EAH.⁵ The presence of a sodium appetite is equivocal in humans,⁸⁹⁻⁹¹ but studies on sodium palatability and preference have revealed significant associations with sodium loss and deficiency.^{88,90,92-97} A peculiar theme echoing throughout these studies is the delayed expression of the attractiveness of sodium-containing items after cumulative sodium losses, particularly in studies involving sweat sodium losses after exercise.⁸⁸⁻⁹⁵

A study by Takamata et al⁸⁸ confirms that the body protects osmolality over extracellular volume after 7 hours of intermittent exercise at 35°C. Elevated serum sodium concentrations after exercise increased the palatability for water and decreased the pleasantness of concentrated sodium beverages during the first hour of rehydration. POsm returned to baseline levels 3 hours after rehydration had begun, at which time the palatability rating for sodium-containing beverages significantly increased over baseline levels. The palatability of highly concentrated sodium beverages continued to increase at 6, 17, and 23 hours after the cessation of exercise which correlated with significant increases in aldosterone secretion, but not AVP or POsm. The authors concluded that “increased H₂O palatability is only associated with osmotically induced thirst and thus contributes to body fluid osmoregulation. In contrast, ECF thirst accompanied by an increased Na⁺ preference appears after a delay of many hours after osmoregulatory responses occur and may contribute to ECF volume regulation”.

Thus, the palatability for sodium is delayed after exercise until POsm is restored to normal levels from the ingestion of, and preference for, plain water. Only after POsm returns to baseline levels—through the ingestion of plain water to dilute elevated serum sodium concentrations—will the body seek sodium (by a palatability increase) to return plasma volume to baseline levels in the succeeding hours after the exercise bout. This is presumably facilitated by the natriorexogenic and diposonic actions of aldosterone and angiotensin II, which are known to stimulate these actions.²³ This phenomenon has also been supported by studies of exercising students, with low sweat rates, who expressed a decreased avidity for sodium when given high doses of salt before exercise,⁹⁴ and has also been clearly demonstrated in rats made hypovolemic by polyethylene glycol injection.²⁰ Figure 2 illustrates that hypovolemic rats, on a normal sodium diet, will respond to isotonic hypovolemia by first ingesting only water until the decline in POsm inhibits water intake. Thereafter, the rats will drink saline until POsm increases, which will again stimulate water intake. The rat will then alternate between saline and water consumption until plasma volume is returned to baseline levels; maintaining a stable POsm throughout the gradual return to euolemia. Thus, it has been shown in both animals and humans that sodium ingestion is necessary to restore plasma volume once osmolality has been stabilized. This restoration normally progresses over the course of 24 hours after the hypovolemic insult.

Because sodium is clearly necessary to restore and maintain plasma volume after sweat sodium losses from

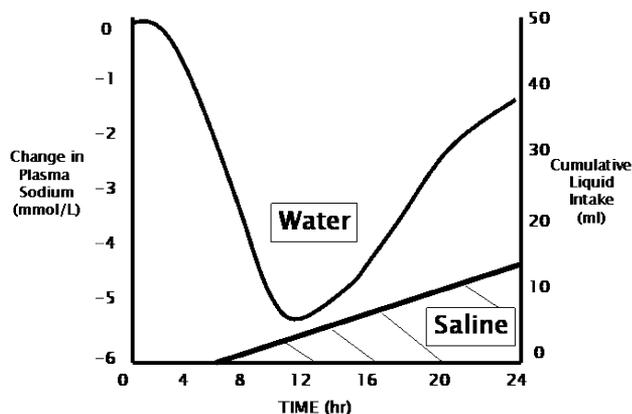


FIGURE 2. Changes in $[Na^+]$ and cumulative fluid intake in hypovolemic rats (after subcutaneous injection of 30% polyethylene glycol solution) with free access to water and concentrated saline solutions over a 24-hour period. Redrawn from *American Scientist*. 1988;76:261–267 with permission from J. G. Verbalis.

exercise, the beneficial effect of sodium ingestion during exercise is a topic of substantial debate. Studies have shown that consumption of hypotonic sodium-containing beverages do not prevent the development of hyponatremia in athletes replacing 100% fluid losses or less^{72,74,98} or in polydipsic psychiatric patients,^{99,100} because most of the sodium ingested is rapidly lost through the urine.^{74,101} In conditions of fluid overload, however, a mild blunting of serum sodium decline occurs with consumption of sodium-containing beverages. This blunting does not eliminate the development of hyponatremia in athletes who continue to overdrink, however.⁵⁹

There are no studies documenting that sodium ingestion during exercise provides a clinical, physiological or performance benefit.^{60,102} On the contrary, there are several studies linking sodium ingestion with *negative* physiologic and performance effects.^{103–107} Konikoff et al¹⁰⁵ supplemented normal dietary intakes with 10.2 g (173 mmol) of sodium 3 days before a 2-hour exercise bout (60% VO_2 Max on a cycle ergometer) with fluid intake matching sweat rate. The authors concluded that the “salt loading” did not have a beneficial effect on temperature and fluid balance and in fact, elicited undesirable effects including significant increases in bodyweight, heart rate, and rectal temperature.¹⁰⁵ Furthermore, the extra sodium was rapidly excreted in the urine, suggesting that the body had no need for the extra NaCl. Similarly, Pitts and Consolazio¹⁰³ administered 9 g of NaCl to 11 subjects just before a 10-mile march. Water was replaced at equal volumes to sweat loss every 2 miles. The supplemental sodium ingestion also led to elevated heart rates and rectal temperatures, along with complaints of “gastrointestinal uneasiness.”¹⁰³ Performance in a VO_2 Max test was impaired after rapid infusion of 30 mL/kg of isotonic saline 30 minutes before the exercise bout.¹⁰⁷ There was a consistent increase in ventilation after every exercise work level

after rapid saline infusion, as well as significant reductions in forced vital capacity and forced expiratory volume. All of these subjects felt more fatigued at maximal effort and reported sensations of “increased leg tightness” These authors hypothesized that the symptoms were due to increased intramuscular tissue pressure and resultant edema from the saline infusion.

Thus, sodium supplementation has no documented advantages when consumed during exercise, with over-replacement exhibiting detrimental cardiorespiratory and thermoregulatory effects during exercise. Because sodium is necessary for plasma volume restoration in the 24 hours after exercise, once $POsm$ has been normalized, electrolyte-containing food and beverages should be freely available after exercise and ingested according to palatability and tolerability.

6. *Conditions where changes in plasma volume alter the set-point between thirst and tonicity: advanced age, the syndrome of inappropriate antidiuretic hormone secretion (SIADH) and exercise in extreme heat or cold:* It is well documented that individuals over the age of 65 years demonstrate a reduced thirst drive—with corresponding decreases in fluid intake—in response to rising tonicity, when compared with younger adults.^{108–112} Evidence suggests that this “reduced thirst drive” alternatively represents an upward shift in the operating set point rather than an age-related defect in osmoregulation.^{109,113} Studies confirm that healthy older men demonstrate appropriate, although delayed, reflex adjustments serving to maintain osmotic homeostasis during conditions of hypertonic hypovolemia¹⁰⁹ and hypervolemia,¹¹⁴ when the rehydration period is *sufficiently long* (3 h). Osmotic stimulation of AVP has been shown to elicit a 3-fold greater increase per unit rise in $POsm$ in older men¹¹² but follows the same pattern as that of younger men when exposed to similar osmotic stimuli.^{111,112,114} Thus, evidence suggests that it is not a reduction in the osmotic drive that is primarily responsible for the attenuation of thirst during aging, but rather a reduction in the sensitivity of cardiopulmonary baroreceptors to plasma volume expansion that may contribute to an upward shift in the operating set point.^{108,110} The diminished inhibitory effect of central volume expansion on thirst may result from an age-related impairment of plasma volume maintenance and expansion; perhaps from the inability to mobilize or maintain proteins in the vascular space.^{113,115} This impairment in older males to expand or maintain plasma volume has been demonstrated in situations of hypertonic saline infusion,^{111,114} thermal dehydration,¹¹³ and during repeated exercise/heat stress.^{108,115} The inability of most elderly people to expand plasma volume may necessitate elevation of the osmotic thirst threshold because age-related declines in renal function could concomitantly facilitate the development of fluid overload hyponatremia if lower, more “physiologic,” osmotic thresholds continued to exist.

Exercise in cold conditions may also elevate the osmotic threshold for which thirst and AVP secretion are

stimulated. Subjects hypohydrated by ~3% to 4% experience a decline in both AVP secretion and perceived thirst sensations when entering a cold (4°C) chamber, despite resting plasma osmolalities above 295 mosmol/kg H₂O.¹¹⁶ These subjects relate significantly lower thirst ratings at 40 and 60 minutes of treadmill exercise—when euhydrated and hypohydrated—in cold compared with temperate environments (27°C), despite similar plasma osmolalities. Thus, the authors of this study hypothesize that thirst and AVP secretion are “attenuated” from central volume expansion, secondary to peripheral vasoconstriction in the cold. These findings suggest that the osmotic operating set point in cold conditions could be elevated due to increased central blood volume.

Conversely, nonosmotic pituitary AVP secretion, leading to dilutional hyponatremia and SIADH, may cause a downward resetting of the osmotic threshold for thirst.¹¹⁷ The presence of thirst at low osmolalities has been hypothesized to pathologically contribute to the development of hyponatremia,¹¹⁸ as patients with chronic SIADH relate the sensation of thirst and report daily fluid intakes similar to healthy individuals. The study by Smith et al¹¹⁷ demonstrates, however, that SIADH patients with chronic dilutional hyponatremia (average serum sodium concentration: 125.8 ± 2.7 mmol/L) respond appropriately to osmotic challenge by hypertonic saline infusion. Baseline plasma osmolalities and the osmotic thresholds for both thirst and AVP release were 20 mosmol/kg H₂O lower in the SIADH group when compared with a group of healthy matched controls. Thirst ratings, elevations in AVP, urine osmolalities, and fluid intakes were similar between the 2 groups with parallel elevations in POsm. Perceived ratings of thirst returned to baseline levels in both groups after 30 minutes of drinking and fluid consumption led to an immediate fall in thirst ratings within 5 minutes in both cohorts. Thus, the suppression of thirst in the SIADH group was similar to that of the control group, suggesting that the neural inhibition of thirst and drinking is preserved in patients with SIADH. These laboratory-based conclusions have been verified in the field setting, where marathon runners diagnosed with EAH reportedly consumed fluids in excess of excretion rates not because they were thirsty, but because they were fearful of becoming dehydrated.¹¹⁹ The sensation of thirst should not be confused with the act of drinking. The act of drinking occurs from regulated (drinking to physiologic thirst) and unregulated (social, habitual, palatability) fluid intake which may be governed by factors (marketing, commercialism) unrelated to the osmotic set point.¹³

Work performance at the initial stages of acclimatization during exercise in extreme heat may improve with “forced” fluid intakes—beyond thirst—at volumes equivalent to sweat loss.^{103,120,121} The environmental temperatures in these trials were extreme, ranging from 38¹⁰³ to 46°C^{120,121} and involved a limited number of subjects. Subjects in the Bean and Eichna trial¹²¹ ingested water equivalent to weight lost (1200 mL) and were able to complete 5 work cycles “without great difficulty” on the first day of exercise compared with subjects receiving

only enough fluid to quench thirst (600 mL). On the fourth day of exercise, however, the degree of acclimatization—as measured by heart rate, rectal temperature and blood pressure—was similar between the two groups. The one subject in the Pitts et al¹⁰³ trial also reported less fatigue and had lower rectal temperatures when “forced” to drink water equal to sweat losses. The fact that the rectal temperatures between ad libitum fluid replacement and “full” replacement varied greatly between trials (~0.3 to 1°C) suggests that initial acclimatization requires fluid intakes beyond thirst to accommodate plasma volume expansion. After plasma volume is appropriately expanded, the need to ingest fluid beyond ad libitum intakes is not necessary to maintain similar rectal temperatures, heart rates and blood pressures. Alternatively, Moroff and Bass¹²⁰ administered a water preload of 2000 mL to nonacclimatized subjects and reported that when these subjects were water loaded, they had lower rectal temperatures and pulse rates compared with when not preloaded. Acclimatized subjects given the same preload, however, displayed lower heart rates with similar rectal temperatures for reasons that are unclear. Thus, initial exercise in extreme heat may require *transient* fluid intakes beyond thirst to facilitate the expansion of plasma volume—and heat acclimatization—irrespective of the current operating osmotic set point.

PRACTICAL RECOMMENDATIONS

1. Drinking to thirst is the body’s *dynamic* physiologic fluid calculator and in most cases will protect athletes from the hazards of both over and underdrinking by providing real-time feedback on tonicity.
2. A *static* fluid calculator can provide an estimate of body fluid losses thereby providing a numeric range as a *generalized* strategy for fluid replacement during racing and training.
3. Athletes are advised to understand their individualized fluid needs through use of a static fluid calculator but ALWAYS defer to physiologic cues to increase fluid intake (thirst) or decrease fluid consumption (increased urination, bloating, weight gain) while running.
4. Water, sodium and glucose (in foods and or beverages) should be freely available at fluid replacement stations, spaced 1.6 km (minimum) to 5 km (maximum) apart. The quantity and amount of food and fluid consumed should be guided by individual palatability and tolerance for such items.
5. Calibrated scales along a marathon course should be at the discretion of the medical team; a weight loss of > 4% or any weight gain constitutes justification for medical consultation.
6. Exercise in extreme heat (> 38°C) may require hydration beyond thirst during the initial days of heat acclimatization whereas advanced age (> 65 y) and cooler environmental temperatures (< 5°C) may elevate the operating set point for the stimulation of thirst.

FINAL WORD

Marathon running is a serious endeavor that requires a personal commitment to sustained physical training. The hazards of marathon running have been highlighted by the recent deaths of 4 novice female runners from EAH. Understanding individual fluid requirements before tackling a 42.2-km race is pivotal toward ensuring the successful and safe completion of such an event.

Although our society revolves around rules and algorithms to guide us through different situations, individuals should not be confined to these rules in a dynamic setting. There are no shortcuts toward great achievement, and marathon running is no exception. Clinicians and scientists must resist handing out unrealistic “blanket advice” to individuals seeking simple answers, but rather should encourage athletes to explore, understand and be flexible toward their own needs. By providing guidelines and advice on how to appropriately understand individual fluid replacement needs, we can eliminate future fluid balance problems by avoiding the temptation to generalize one rule for every situation and every athlete.

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