



Review

Effects of Sodium Intake on Health and Performance in Endurance and Ultra-Endurance Sports

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Abstract: The majority of reviews on sports nutrition issues focus on macronutrients, often omitting or paying less attention to substances such as sodium. Through the literature, it is clear that there are no reviews that focus entirely on the effects of sodium and in particular on endurance sports. Sodium intake, both at high and low doses, has been found to be associated with health and performance issues in athletes. Besides, there have been theories that an electrolyte imbalance, specifically sodium, contributes to the development of muscle cramps (EAMC) and hyponatremia (EAH). For this reason, it is necessary to create this systematic review, in order to report extensively on the role of sodium consumption in the population and more specifically in endurance and ultra-endurance athletes, the relationship between the amount consumed and the occurrence of pathological disorders, the usefulness of simultaneous hydration and whether a disturbance of this substance leads to EAH and EAMC. As a method of data collection, this study focused on exploring literature from 1900–2021. The search was conducted through the research engines PubMed and Scopus. In order to reduce the health and performance effects in endurance athletes, simultaneous emphasis should be placed on both sodium and fluid intake.

Keywords: sodium; endurance sports; ultra-endurance sports; hyponatraemia; muscle cramps; hydration



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1. Introduction

Metals are a group of minerals that cannot be produced by the body. Sodium, being an inorganic element, is an essential component in human nutrition. As such, excessive or very low intake of this ingredient can have adverse effects on the body. So, attention should be paid to this element as well. Most reviews on sports nutrition tend to praise protein (PRO), carbohydrates (CHO) and fat (FAT) without paying much attention to sodium [1–3]. The loss of body fluids during sport or exercise is largely due to sweating [4]. Thus, replacement of Na⁺ loss in sweat is recommended when the duration of exercise is longer than 2 h, when the climate is hot or during intense Na⁺ loss in sweat (e.g., >3–4 g Na⁺) [5,6]. In endurance sports, the duration of a race is longer than 5 min [7], while in ultra-endurance sports, the duration of a race is longer than 6 h [2]. So, it is important to fully replace the losses in fluids and Na⁺ to restore e-hydration. There are also theories [8,9] suggesting a positive association of sodium with muscle cramps and the occurrence of hyponatremia, while the causes are attributed to the long-duration intensity of exercise, which leads to muscle fatigue, and excessive fluid consumption, mainly pure water, respectively [10,11]. Thus, the following questions arise: (a) What should be the amount of sodium in the population? (b) Do they differ from that of athletes? (c) Is there any evidence that finally confirms the link between sodium and muscle cramps and hyponatremia? (d) Can sodium eliminate the occurrence of hyponatremia? (e) What are the hydration recommendations in endurance sports? The aim of this study is to collect research and literature from 1900 to

2021 in order to clarify the causes of muscle cramps, to record studies that clarify the role of sodium in hyponatremia. Hydration recommendations will also be made before, during and after exercise, with an emphasis on both the importance of fluid consumption and the importance of insufficient hydration leading to either over-hydration or dehydration. Thus, 130 bibliographies were used to complete this work.

2. Importance of Sodium and Ideal Composition

Sodium chloride (common salt) is an anionic compound with an extracellular fluid concentration adjusted to about 135–145 mmol/L [12]. More specifically, sodium is the main cation in extracellular fluid [13] with a multitude of benefits for both the general population and the sporting world, such as contributing to the release of digestive secretions and controlling the absorption of certain nutrients (amino acids, glucose, galactose and water) [12,14]. In addition, it ensures sufficient blood volume, blood pressure and, ultimately, organ perfusion [15]. In addition to its importance in terms of regulating water and fluid balance [16], it is vital for the stimulation of muscle and nerve cells and is also involved in the control of the acid–base balance [17]. In the sport section, sodium helps to maintain serum electrolyte concentrations resulting in a balance of intravascular osmotic pressure and plasma volume [18]. It increases the thirst stimulus and reduces the amount of urine produced [19], effects that ultimately reduce physical fatigue and medical problems associated with these homeostatic imbalances in endurance sports [20]. However, attention should be paid to excess sodium, which contributes to high blood pressure and damage to certain organs such as the heart, kidneys and bones [21]. In contrast, a low intake has been associated with an increased risk of cardiovascular events and death, independent of blood pressure levels [22,23].

3. Effects of Sodium Consumption

3.1. Very Low Sodium Consumption

There are studies [23,24] that show that even low sodium intake may not always be beneficial for the treatment of cardiovascular disease. Low sodium intake has been associated with an increased risk of cardiovascular events and death, independent of blood pressure levels [22,24]. In the study by Mente and colleagues, it was found that urinary sodium excretion of less than 3 g per day did not further reduce systolic blood pressure but actually tended to increase diastolic blood pressure in people with or without hypertension [25]. In both normotensive and hypertensive individuals, low sodium intake can cause insulin resistance and an increase in plasma or serum levels of renin, aldosterone, epinephrine and norepinephrine [22,26]. Subsequently, in the reviews by Yin et al. [27] and Van Horn et al. [28], it appears that sodium consumption of 0.5–1.0 g per day is attributed as an optimal physiological intake, in contrast to those of Mente et al. [29] and Mente et al. [25] where concerns have been raised about potential negative health effects due to a reduction in intake below the average global consumption level. These concerns mainly come from prospective observational studies [25,30], some of which report associations between sodium intake and cardiovascular disease [31,32]. Individuals reporting low levels of sodium intake are often patients with a history of disease who have been advised to reduce sodium. Among these individuals, there may be an increased risk resulting from concomitant disease leading to adverse cardiovascular outcomes rather than low sodium intake [25,29].

3.2. Very High Sodium Consumption

Increased sodium intake leads to an increase in intra-glomerular pressure, which can cause or exacerbate chronic kidney damage and increase the risk of progressive kidney disease [33]. Subsequently, the mechanisms by which a high-salt diet increases the risk of gastric cancer in humans are poorly understood [34]. One speculation is that foods such as processed meat, cured meat and dried fish exposed to salt are high in nitrite compounds, which may be involved in gastric carcinogenesis [35]. Still, studies [36,37] on

the association of sodium with osteoporosis suggest that increased sodium intake is a risk factor for the development of the disease (Table 1). Specifically, in postmenopausal women in Korea [37,38], it was shown that high sodium intake (>2000 mg) leads to increased urinary excretion (>2 g/day), which leads to hypercalciuria, thus increasing the risk for osteoporosis [39]. Finally, excessive salt intake by consumers has been associated with the development of hypertension [40,41] and, consequently, with a higher risk of cardiovascular disease, particularly for hypertensives and the elderly [42]. Reducing sodium intake is associated with a reduction in systolic and diastolic blood pressure, particularly in hypertensive and normotensive individuals [12,43].

Table 1. Relationship of clinical conditions with high sodium intake.

Clinical Condition	n	Average Age (Years)	Average Sodium Intake	Type of Study	Correlation	References
Kidney Disease	1384	≥20	11.5 gr	Observational	Positive	[44]
Cancer	2485	18–92	9 g	Case-control	Positive	[45]
Cancer	634	40–49	12.8 g	Cross-sectional	Positive	[46]
Hypertension	3230	22–73	9.4 g	Meta-analysis	Positive	[47]
Hypertension	10,074	20–59	Serum > 100 mmol/L	Cross-sectional	Positive	[12]
Osteoporosis	537	58 ± 6	>2 g/day	Cross-sectional	Positive	[37]
Osteoporosis	102	24 ± 3.4	2.6 ± 1.1 g/day	Cross checked	Positive	[36]

Although it is possible that both low and high levels of sodium intake are harmful, the mechanism of these effects is uncertain [23]. What does seem certain, however, is that after a retrospective search during the 2010–2019 period, the ideal amount of sodium was 1.5 g/day (Figure 1). It appears that, over time, the recommendations for both the healthy population and hypertensive patients do not make much difference.

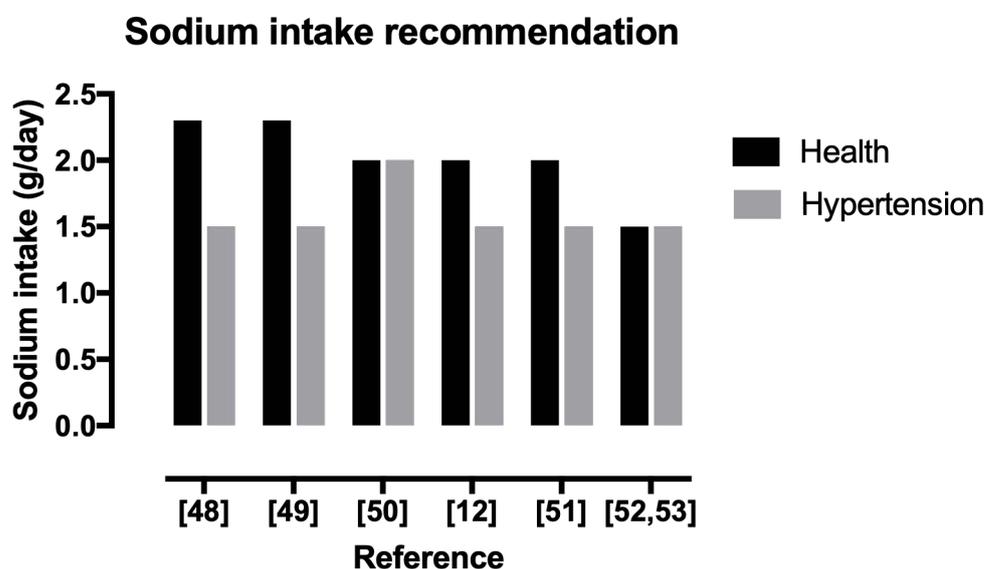


Figure 1. Recommendations of sodium intake [12,48–53].

4. Sodium in Sport

Turning to the main issue, despite the positive effects of sodium consumption, such as maintaining aldosterone and vasopressin production [54], increasing thirst stimulation and decreasing urine production [55], enhancing electrolyte balance and stimulating water retention in the body, resulting in a reduction of physical fatigue in endurance sports [56], it has been implicated by previous theories [8,9] that it contributes, positively, to the occurrence of muscle cramps and hyponatremia during exercise.

4.1. Exercise-Associated Muscle Cramps

Exercise-associated muscle cramps (EAMC) are defined as painful, spasmodic and involuntary contractions of skeletal muscles during or immediately after physical activity [10,57]. The prevalence of EAMC in different sports varies, as shown in Table 2. The basic etiological evidence in the literature by Schwellnus et al. [8] is that electrolyte depletion through excessive sodium loss in sweat along with dehydration causes this condition. These causes do not offer plausible pathophysiological mechanisms with supporting scientific evidence that could adequately explain their clinical presentation and management. Thus, studies were conducted [10,57] whose results showed that dehydration and sodium depletion do not appear to be associated with muscle cramps.

Table 2. Prevalence of exercise-associated muscle cramps (EAMC) in athletes.

Sports	Prevalence EAMC	References
Ultra-Marathon 166 km	14%	[58]
Marathon	18%	[10]
Ironman Triathlon	23%	[59]
Ultra-Marathon 100 km	23%	[60]
Ultra-Marathon 56km	41%	[59]
Cycling	60%	[8]
American football	30–53%	[61,62]

4.2. Exercise-Associated Hyponatremia

Exercise-associated hyponatremia occurs when plasma sodium concentration is <135 mmol/L or there is a decrease in serum sodium of 7–10% [63,64]. This can occur during or after prolonged exercise for 4 to 6 h or more [65] and can be detected up to 24 h after the end of exercise [63,66–68]. It is a disorder that has been widely described in marathon runners [67] but also in athletes and in other endurance and super-endurance events. Ultra-endurance athletes competing in events longer than 24 h, such as participants in the Ultraman, Titan Desert or Sables Marathon, are at a higher risk of developing Exercise-Associated Hyponatremia (EAH) compared to participants in shorter endurance events such as the marathon (Table 3.) [67–69]. However, the environment also contributes as a factor to hyponatremia [67,70]. The main causes of this condition appear to be excessive fluid consumption during exercise, increased sodium loss in sweat and loss of normal Antidiuretic hormone (ADH) suppression, called the syndrome of inappropriate ADH secretion (SIADH) [71]. In a more recent study, that of Buck and colleagues [72], EAH was found to be due to both increased consumption of hypotonic fluids [73] and inappropriate water retention [74]. The contribution of sodium loss from excessive sweat is controversial as sweat loss varies greatly between individual athletes but typically ranges between 15 and 65 mEq/L and sweat volume ranges from 250 mL/h to >2 L/h. Therefore, sodium lost from sweat is not thought to be responsible in itself for the development of EAH, but rather an additive effect along with hyperhydration. The symptoms of hyponatremia as seen in Table 4 vary [63,72,75–79]. Because some symptoms of hyponatremia can be identified with other conditions, the clinical sign that differentiates hyponatremia from other conditions that result in collapse is vomiting. Vomiting may be a reflex action in response to the increasing distension of large and unnecessary amounts of fluid within the gastrointestinal tract or may be caused by the central nervous system [80]. However, the presence of cognitive impairment, coma, seizures or respiratory distress suggests exercise-associated hyponatremic encephalopathy and should be recognized immediately [81], as it has been confirmed as a cause of at least fourteen deaths [77].

Table 3. Prevalence of exercise-associated hyponatremia.

Sports	Trial	Prevalence EAH	References
Marathon	Marathon	15%	[82]
	Houston Marathon 2000	<5%	[80]
	Boston Marathon	5%	[83]
	Houston Marathon 2000–2004	>20%	[84]
	Zurich Marathon	<5%	[85]
	Boston Marathon 2001–2018	<5%	[86]
	London Marathon	Up to 22%	[87]
Ultra-Marathon	Ultra-marathon in Asia	38%	[88]
			[89]
	161 km in North America	30–51%	[90]
Cycling	109 km	12%	[92]
	210–250 km	4.5% (4 to 90 persons)	[93]
Triathlon	Ironman-Triathlon	20%	[69]
	Ironman-Triathlon	1.8–28%	[94,95]
	Triple Ironman	26%	[67]

Table 4. Symptoms of EAH [65,69,72,75,77,81,96].

Mild	Severe	Clinical Appearance
Weariness	Mental disorder	Heat stroke
Dizziness	Ictus, collapse	Hypoglycemia
Slow urine production	Oliguria	Stress-related collapse
Sickness	Coma	Muscle cramps
Headache	Death	Edema
Weakness		

Prevention and Treatment

EAH can be prevented by avoiding over-hydration, ensuring adequate oral sodium intake and training athletes, focusing on sweat rate and sweat sodium content, exercise intensity and environmental conditions [11,72]. Estimates of individual athlete replacement needs can also be used by monitoring weight changes during training activities, although this may not be practical. Forced hydration, particularly in large quantities, should be discouraged [75,97]. Before treatment, it is very important to differentiate EAH from other exercise-related diseases such as heat exhaustion, heat stroke and exercise-induced collapse associated with sickle cell anemia, as their treatments are often contradictory [72]. If a patient has no neurological symptoms, EAH is considered mild and oral fluid restriction is required (Table 5). In studies by Siegel [86] and Bridges [98], oral HTS (hypertonic saline) reduces symptomatology from EAH faster than an IV bolus (intravenous administration). Unfortunately, oral hypertonic fluids may not be palatable [99], which limits their usefulness. Therefore, knowing that the main cause of EAH is the habit of excessive fluid consumption during a race combined with inadequate sodium intake [90], it is noteworthy to provide a full description of both hydration and incorrect fluid intake practices, as well as fluid and sodium quantities.

Table 5. Management of EAH symptoms.

	Mild Symptoms	Severe (Neurological Symptoms)	In Encephalopathy	Bibliography
Intravenous isotonic fluids of any type or volume are not recommended	recommended	is not recommended	is not recommended	[100]
Concentrated oral sodium replacement may be given (with reservation)	recommended	is not recommended	is not recommended	[86]
Bolus 100 mL of intravenous hypertonic saline (3% sodium chloride)	is not recommended	recommended	recommended	[63] [75]
Should be treated immediately with intravenous IV bolus infusion or HTS infusion for acute reduction of swelling in the brain	is not recommended	is not recommended	recommended	[58] [101]

5. Sodium and Hydration

There are some studies [102,103] that have been dedicated to determining the benefits of salt intake on endurance performance. Most of them report improved physical performance, an attenuated decrease in serum sodium concentration and enlarged plasma volume during endurance activities. It is worth noting that laboratory studies have always accompanied salt supplementation during exercise with a liquid ingestion pattern that matches sweat losses, which probably facilitated the occurrence of these benefits. In general, the water needs of athletes tend to vary according to individual characteristics and the type or intensity of exercise in which they participate, making individualized fluid replacement strategies necessary [102,103]. This should result in both preventing adverse effects and improving the performance of athletes [104] and maintaining proper hydration during exercise [105]. Athletes participating in endurance and ultra-endurance races should be aware that both prior acclimatization to race weather conditions and adequate fluid–electrolyte balance reduce the risk of dehydration and thus the risk of EAH [106]. The loss of body fluids during sport or exercise is largely due to sweating [107].

It is primarily a function of heat production by metabolism, but can be modified by the environment, clothing, acclimatization, hydration status, the size and composition of the athlete's body and the degree of training [108–110]. However, this heat dissipation is accompanied by typical fluid losses of 0.5–1.9 L/h according to Baker and colleagues [110]. Thus, the goals are to improve athlete performance, maintain proper hydration during exercise, prevent adverse effects (dehydration-hyperhydration) and avoid losses greater than 2–3% of body mass during exercise [104,105]. Dehydration causes a loss of intracellular and extracellular (plasma and interstitial) fluid in proportion to water loss, compromising cardiovascular function, reducing muscle blood flow and cardiac output [111]. Typically, sweat is hypotonic (i.e., lower concentration of electrolytes) compared to plasma [112]. Therefore, exercise-related sweat losses lead to a decrease in plasma volume and an increase in plasma electrolyte concentration (primarily sodium), known as hypertonic hypovolemia [112]. Low blood volume, due to dehydration, also prevents the transport of oxygen and glucose to muscle cells [111]. Dehydration of 2% of the body weight, which generally occurs during exercise lasting more than 90 min, appears to significantly reduce endurance performance in 20–21 °C environments [113]. Further, weight loss of more than 4% of the body weight during exercise can lead to heat illness, heat exhaustion, heat stroke and possibly death [114].

Athletes may drink large amounts of fluids in the hours before competition, often in combination with an osmotic agent such as glycerol or sodium, in order to temporarily increase total body water to compensate for sweat losses and delay the progression of absolute hypohydration [115]. This practice has been identified as a result of incorrect guidelines for fluid intake in sport, and when carried out in extreme situations, can

lead to serious consequences associated with hyponatremia [77]. Therefore, avoiding over-hydration or under-hydration is recommended for both health and performance in ultramarathon running. In some endurance and ultra-endurance races, it has affected up to 30% of participants [116]. However, various dosages have been suggested for before, during and after exercise as shown in Tables 6–8. These dosages are chosen according to the athlete’s tolerance to fluid volume.

Table 6. Pre-exercise hydration dosages.

Timing	Dosage	Bibliography
Before exercise	5 to 10 mL/kg body weight	[117]
Before exercise	5–7 mL/kg 4 h before exercise and more 3–5 mL/kg, 2 h before competition	[118]
4 h before exercise	5–7 mL/kg water or sports drink	[119]
Before exercise	400–600 mL cold water or sports drink 20–30 min before exercise	[120]

Table 7. Fluid intake during exercise.

Sports	Timing	Dosage	Bibliography
Ultra-Marathon	During exercise or competition, each 20 min	150–250 mliquids	[121]
Ultra-Marathon Competition	Each 1 h	300–600 mL	[70]
Marathon	Each 1 h	400–800 mL	[118]
Regardless of sport	During the exercise	450–675 mL, for every 0.5 kg of body weight lost	[122]

Table 8. Fluid intake after the exercise.

Sports	Timing	Dosage	Bibliography
Regardless of sport	After the exercise	1.25 to 1.5 L liquids for every 1 kg of weight loss	[117]
General for athletes in a warm climate	After the exercise	100–120% body mass losses	[123]
Regardless of sport	For fullrestoration	450–675 mL for every 0.5 kg of weight loss	[119]
General for athletes	After the exercise	Liquid with 150% or 200% of weight loss	[124]

6. Sources and Dosages of Sodium in Endurance–Ultra-Endurance Sports

In the review by Grozenski and Kiel [125], the consumption of drinks with 20 to 50 mEq-L sodium or small amounts of salted snacks helps to stimulate thirst, reabsorption of fluids and, by extension, support osmotic balance during endurance events. Additional salting of everyday foods is an inexpensive and effective method of increasing sodium intake (pickles, tomato juice, canned soups, baked beans and pizza) [56].

Furthermore, adding 3.2 gr (0.5 teaspoons) of table salt to every 960 mL (32 fl oz) of a sports drink will further increase sodium concentration without negatively affecting taste or absorption [56]. Additionally, Tiller’s [121] study argues that in order to reduce the risk of hyponatremia during long-duration exercise, runners should consume sodium at concentrations of 500–700 mg-L of fluid [118]. Slightly higher amounts of sodium (and other electrolytes) will be required under conditions of heat (e.g., >25 °C/77 °F) and/or humidity (e.g., >60%). When the sweat rate is elevated in such conditions, runners should aim for 300–600 mg-h-sodium (1000–2000 mg NaCl). If consumed in liquids, sodium concentrations greater than 1000 mg-L (50 mmol-L) should be avoided as this may reduce

the palatability of the drink [4]. The amounts ingested should also be offset against the sodium consumed from salt-containing foods, although it should be noted that it is unlikely that the recommended sodium intake rate from food alone will be achieved. The Academy of Nutrition and Dietetics (AND), Dietitians of Canada (DC) and The American College of Sports Medicine (ACSM) recommend sodium intake during exercise in athletes with high sweat rates (>1.2 L/h), subjective “salty sweating” and prolonged exercise >2 h [119]. Although highly variable, average sweat rates range from 0.3 to 2.4 L/h, and the average sweat sodium content is 1 gr/L (50 mmol/L) [119]. A sports drink containing sodium in the range of 10–30 mmol/L (230–690 mg/L) results in optimal absorption and prevention of hyponatremia [126], a concentration found in typical commercial sports drinks. ACSM recommendations for sodium intake are 300–600 mg/h (1.7–2.9 g salt) during prolonged exercise [120,126]. However, as shown in the previous chapters, sodium intake can be positively associated with the onset of the above disorders (EAH, EAMC) [8,9].

7. Discussion

Sodium is the main cation of extracellular fluid that has many advantages [12,17], with one of its main functions being maintaining fluid balance in the body [56]. Moreover, as mentioned above, the right amount plays an important role, as both high consumption and low consumption are health risks [23]. Many organizations (such as the Institute of Medicine, World Health Organization) recommend sodium intakes of up to 1.5 g/day. In contrast, in sports, amounts vary. It is well documented that the sweat rate and sweat electrolyte concentrations can vary significantly as a result of many factors, therefore individualized fluid replacement strategies are recommended [118]. Urine output after exercise decreases as the sodium concentration in the drink increases. Plain water is unlikely to be sufficient to restore fluid balance after exercise due to the subsequent reduction in sodium concentration and plasma osmolality that causes diuresis. Furthermore, sodium intake should ideally be equal to the sodium concentration lost in sweat. The sodium content of commercial sports drinks (~20–25 mmol-L, 460–575 mg-L) is lower than that normally lost in sweat [127] and should also be considered a conservative target. Regarding muscle cramps, there does not appear to be documented scientific evidence for the sodium–EAMC relationship. The most common cause of this condition is exercise at a higher relative intensity or exercise duration compared to normal training, resulting in muscle fatigue [10]. While sodium intake during a race can mitigate the drop in blood sodium concentrations, it cannot prevent EAH under conditions of excessive fluid intake [128]. Sodium intake during exercise will not prevent EAH in the presence of hyperhydration, but excessive sodium intake may actually increase the risk of EAH [129]. It is the amount of fluid, not the amount of sodium consumed, during exercise that increases final blood sodium concentrations. Sodium-containing sports drinks that are hypotonic will not prevent EAH in athletes who drink excessively during exercise [130]. Athletes should be trained to tolerate drinking larger amounts of water and ensure they consume more fluids in hotter and more humid environments. Sports nutritionists, dietitians and sports coaches can play an important role in educating athletes and coaches on proper hydration methods and overseeing fluid intake during training and competition. The goal is to limit weight loss to 2% [125].

8. Conclusions

Sodium is an element that should not be missing from people’s diets. Thus, the ideal amount of sodium intake in the largest range of the population appeared to be in the range of 1.5 g/day. However, it is equally important for endurance athletes to consume 300–600 mg/h. It was also noted that there is no documented scientific evidence on the relationship between sodium and muscle cramps. Sodium seems to be one, but not the only, factor contributing to this situation. For hyponatremia, its intake can mitigate the drop in blood concentrations, but cannot eliminate it. Finally, attention should first be paid to the individual amount of fluids consumed and then to the amount of sodium consumed.

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