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Exercise-Associated Hyponatremia in Endurance Sports: Risk Factors, Pathophysiology and Prevention – A Narrative Review

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Abstract

Background. Exercise-associated hyponatremia (EAH) is a potentially serious disturbance of water and sodium homeostasis occurring during or after prolonged endurance exercise. Although excessive fluid intake is an established risk factor, EAH results from interactions between fluid intake, arginine vasopressin activity, sodium balance, exercise duration, environmental conditions, and individual characteristics.

Aim. This narrative review aimed to summarize current evidence on the epidemiology, pathophysiology, risk factors, and prevention of EAH in endurance sports, with emphasis on recent findings and modifiable factors.

Methods. Relevant literature was identified primarily through PubMed searches and reference-list screening. Particular emphasis was placed on studies published between 2020 and 2026, while selected earlier studies were retained as foundational evidence. Original studies, systematic and narrative reviews, clinical guidelines, and relevant physiological research were considered.

Results. EAH occurs in marathon running, triathlon, ultramarathon, and mountain endurance events, with substantial variation in reported prevalence. Excessive fluid intake relative to free-water excretion remains the most consistently identified modifiable risk factor. However, EAH may also occur despite body mass loss, indicating that sodium losses and other physiological mechanisms contribute in some athletes. Exercise-associated arginine vasopressin activity facilitates water retention during prolonged exercise. Current evidence does not support universal sodium supplementation as a reliable preventive strategy. Hydration beliefs and planned drinking behaviors may also influence risk.

Conclusions. EAH is a multifactorial disorder rather than simply a consequence of sodium depletion or excessive water consumption. Prevention should emphasize avoiding forced drinking, individualized hydration strategies, and athlete education regarding fluid and sodium balance.

Keywords: exercise-associated hyponatremia; endurance sports; endurance exercise; hydration; fluid balance; sodium balance; arginine vasopressin; risk factors; prevention

1. Introduction

Exercise-associated hyponatremia (EAH) is a potentially serious disturbance of fluid and electrolyte homeostasis that may develop during or shortly after prolonged physical exercise. It is commonly defined as a serum or plasma sodium concentration below 135 mmol/L occurring during exercise or within 24 h after exercise [1,2,8]. Although EAH was initially recognized predominantly among participants in prolonged endurance events, particularly

marathon and ultramarathon races, it has subsequently been reported across a broader range of sporting and physically demanding activities [1,2]. Nevertheless, prolonged endurance exercise remains a particularly relevant setting because of the combination of sustained physiological stress, prolonged exposure to fluid intake, and substantial changes in water and sodium balance. The clinical importance of EAH is related not only to the biochemical abnormality itself but also to the wide spectrum of associated manifestations. Mild or moderate cases may remain asymptomatic or present with relatively nonspecific symptoms such as headache, nausea, vomiting, fatigue, dizziness, or weakness, whereas more severe cases may involve altered mental status and neurological manifestations associated with cerebral edema [1,2,8]. The clinical presentation may also overlap with other causes of exercise-associated collapse, which can complicate recognition of the condition in the field [2,4,9]. Accordingly, EAH represents an important issue in sports medicine, particularly in endurance events where large numbers of participants may be exposed to prolonged exercise under variable environmental conditions. The epidemiology of EAH appears to be heterogeneous and is strongly influenced by the characteristics of the sporting event and the population studied. Studies conducted in marathon runners, triathletes, and ultramarathon participants have reported substantially different prevalence estimates, reflecting differences in exercise duration, environmental conditions, athlete characteristics, and hydration practices [1,4,5,7,10,16]. Recent evidence further suggests that EAH should not be regarded as a uniform phenomenon across endurance sports. In particular, the large contemporary review by Armstrong et al. demonstrated substantial variability in serum sodium concentrations, symptomatology, and severity across different types of prolonged endurance activities [1]. Similarly, studies performed during ultramarathons have shown considerable inter-individual variability in changes in serum sodium and body mass, even among athletes exposed to comparable exercise durations and environmental conditions [3,5,16,23].

The pathophysiology of EAH is multifactorial. Excessive fluid intake relative to the capacity for renal water excretion is considered one of the major mechanisms, particularly when fluid consumption exceeds losses incurred through sweating and urinary excretion [1,2,8]. However, the development of EAH cannot be adequately explained by fluid intake alone. Non-osmotic secretion of arginine vasopressin (AVP) during prolonged exercise may impair renal free-water excretion despite low or normal plasma osmolality, thereby facilitating water retention and dilution of serum sodium [2,8,23]. Importantly, recent physiological observations support the concept that exercise itself may provide non-osmotic stimuli for AVP secretion. In male ultra-endurance athletes, substantial increases in copeptin, a stable surrogate marker of AVP

secretion, have been observed during prolonged mountain races despite relatively modest changes in serum sodium [23]. This finding reinforces the view that EAH reflects a complex interaction between exercise-induced hormonal responses and fluid intake rather than a simple consequence of sodium depletion.

Sodium losses through sweat may also contribute to disturbances in serum sodium concentration, although their relative importance in the development of EAH remains debated [1,2,18]. Sweat composition is highly variable between individuals and is influenced by physiological and environmental factors, making the relationship between sweat sodium loss and systemic sodium balance complex [20,22]. Moreover, studies of endurance athletes have shown that sodium supplementation does not consistently prevent EAH or other exercise-associated disturbances [11,12,18]. In some settings, changes in serum sodium appear to be more closely related to body mass and fluid balance than to the absolute amount of sodium consumed [1,3]. These findings suggest that sodium intake should be considered within the broader context of water and electrolyte homeostasis rather than as an isolated preventive intervention.

Individual susceptibility to EAH may also be influenced by sex, body size, exercise duration, environmental conditions, previous experience, and hydration behavior [1,4,5,7]. For example, studies of marathon and triathlon populations have identified differences in the clinical presentation and prevalence of EAH between men and women, while ultramarathon studies have demonstrated that EAH may occur both in athletes who gain body mass and in those who finish with a reduction in body mass [4,5]. The latter observation is particularly relevant because it challenges an overly simplistic view of EAH as synonymous with overt overhydration. Furthermore, differences in hydration beliefs and pre-planned drinking behaviors may influence fluid intake during endurance competition, emphasizing the importance of athlete education as part of preventive strategies [13,14].

Prevention of EAH has therefore increasingly shifted away from universal recommendations to consume large amounts of fluid toward more individualized approaches based on thirst, exercise duration, environmental conditions, and expected fluid losses [2,9,15,21]. However, uncertainty remains regarding the optimal balance between fluid and sodium intake in different endurance settings. Athletes may also receive inconsistent information concerning the role of sodium supplementation, dehydration, and drinking strategies, potentially contributing to inappropriate hydration practices [13,14,18].

Despite the growing body of evidence, important areas of uncertainty remain. Contemporary studies continue to demonstrate substantial variation in the incidence, physiological

mechanisms, and clinical characteristics of EAH across endurance disciplines [1,3–7,16,23]. In addition, recent research has expanded the understanding of the role of AVP, body mass changes, hydration behavior, and sodium handling, while raising questions about the extent to which traditional risk factors can be generalized across different athletic populations [2,3,23]. A focused synthesis of these findings may therefore be useful for integrating physiological mechanisms with modifiable risk factors and current prevention strategies.

The aim of this narrative review is to summarize and critically discuss current evidence concerning exercise-associated hyponatremia in endurance sports, with particular emphasis on its epidemiology, pathophysiology, risk factors, fluid and sodium balance, and strategies for prevention. Particular attention is given to recent evidence from endurance athletes and to findings that challenge traditional assumptions regarding the relationship between fluid intake, sodium loss, and the development of EAH.

2. Materials and methods

This narrative review was conducted to summarize and critically discuss the available evidence concerning exercise-associated hyponatremia (EAH) in endurance sports, with particular emphasis on epidemiology, pathophysiology, risk factors, fluid and sodium balance, hydration behavior, and prevention. Because the purpose of the review was to provide an integrated and clinically relevant synthesis rather than to perform a quantitative analysis of the available evidence, a narrative review methodology was adopted.

Relevant publications were identified primarily through searches of the PubMed database, supplemented by examination of the reference lists of key review articles and primary studies. Additional literature was considered when identified through citation tracking of the selected publications. Particular attention was given to recent evidence published between 2020 and 2026, reflecting the aim of incorporating contemporary findings into the review. Earlier publications were retained when they provided seminal epidemiological, physiological, or conceptual evidence that remained relevant to the current understanding of EAH.

The literature considered for inclusion comprised peer-reviewed original research articles, systematic reviews, narrative reviews, clinical practice guidelines, and other relevant scientific publications addressing EAH or closely related aspects of fluid and sodium homeostasis during prolonged exercise. Studies involving endurance athletes, including marathon runners, ultramarathon participants, triathletes, and athletes participating in prolonged mountain or trail events, were prioritized. Publications investigating hydration behavior, sodium intake, sweat composition, exercise-induced hormonal responses, and physiological regulation of serum

sodium were also considered when they provided information directly relevant to the development or prevention of EAH.

Particular emphasis was placed on studies evaluating serum sodium concentration, changes in body mass, fluid intake, sodium intake or loss, markers of arginine vasopressin activity, environmental conditions, exercise duration, sex-related factors, and hydration practices. Publications focused primarily on the acute treatment of severe EAH were not considered a major focus of the present review, as detailed management of established symptomatic EAH was beyond the scope of the article.

The selected literature was assessed qualitatively. Findings from individual studies were compared with those reported in contemporary reviews and guidelines, with attention given to areas of agreement, conflicting observations, and limitations of the available evidence. Because of substantial heterogeneity in study populations, endurance disciplines, environmental conditions, definitions of EAH, and methods used to assess hydration and sodium balance, no quantitative meta-analysis was performed.

3. Results

3.1. Definition and epidemiological characteristics of exercise-associated hyponatremia

Exercise-associated hyponatremia (EAH) is generally defined as a serum or plasma sodium concentration below 135 mmol/L during exercise or within 24 h after exercise [1,2,8,9]. It has been described across a variety of prolonged physical activities, although the majority of available evidence concerns marathon running, ultramarathons, triathlons, and long-distance trail events [1,4–7]. Reported prevalence varies considerably between studies, reflecting differences in exercise duration, environmental conditions, athlete characteristics, hydration practices, and methods of sodium assessment [1,7].

The clinical relevance of EAH was demonstrated in the Boston Marathon, where Almond et al. reported sodium concentrations ≤ 135 mmol/L in 13% of runners and identified longer race duration, greater fluid intake, and body mass gain as important risk factors [10]. Subsequent studies have confirmed that EAH also occurs during ultramarathons and triathlons, although prevalence varies markedly between events [4–7,16]. In a high-altitude 161-km ultramarathon, EAH was identified in 20% of participants, with a higher incidence among women in univariable analysis [5]. In IRONMAN® triathletes, clinical manifestations ranged from asymptomatic biochemical abnormalities to neurological symptoms [4].

Recent evidence emphasizes that EAH is heterogeneous rather than uniformly distributed across endurance sports. Armstrong et al. reported substantial differences in serum sodium concentrations and clinical severity between sporting disciplines [1]. Importantly, changes in serum sodium do not necessarily reach the diagnostic threshold: Chlíbková et al. observed a progressive decline in plasma sodium during a seven-stage ultramarathon without development of EAH in female runners [3]. Severe neurological manifestations are uncommon but clinically important, and fatal exercise-associated hyponatremia has been reported [1,2,17].

3.2. Fluid balance and water intake

Excessive fluid intake relative to the ability to excrete free water is one of the most consistently recognized modifiable factors associated with EAH [1,2,8,9]. Early marathon data demonstrated a strong association between body mass gain during exercise and hyponatremia [10]. However, subsequent ultramarathon studies have shown that EAH may also occur in athletes who finish with reduced body mass [4,5,16]. Therefore, although body mass gain is an important marker of excess fluid retention, body mass alone cannot explain all cases.

Several studies support the use of individualized or thirst-guided drinking strategies. In a 40-km military route march, participants allowed to drink ad libitum did not develop EAH while avoiding clinically significant dehydration [15]. This is consistent with recommendations against forced drinking schedules [9,21]. At the same time, endurance athletes report heterogeneous hydration beliefs and practices. Chlíbková et al. observed substantial variation in hydration-related behaviors [13], while Namineni et al. demonstrated considerable differences in marathon runners' knowledge and planned hydration strategies [14].

Overall, the available evidence indicates that fluid intake is an important determinant of EAH, but its effect depends on exercise duration, body size, sweat losses, renal water handling, and the hormonal regulation of water balance.

3.3. Arginine vasopressin and regulation of water balance

Non-osmotic secretion of arginine vasopressin (AVP) is a central component of the current pathophysiological model of EAH. AVP promotes renal water reabsorption and may limit free-water excretion despite normal or reduced plasma osmolality [2,8]. Recent field data support substantial activation of this pathway during prolonged endurance exercise. Schenk et al. demonstrated marked increases in copeptin, a stable surrogate of AVP secretion, during two ultra-endurance mountain races of different distances [23]. However, most participants did not develop hyponatremia, indicating that AVP activation alone is insufficient and that fluid intake and other factors determine whether clinically relevant sodium dilution occurs.

These findings support a multifactorial model in which exercise-induced hormonal responses interact with water intake and sodium balance [1,2,23].

3.4. Sodium loss, sweat composition, and sodium intake

Sodium balance during prolonged exercise is influenced by dietary or supplemental sodium intake and losses through sweat. Sweat sodium concentration varies considerably between individuals and is determined by the physiology of eccrine sweat secretion and ductal sodium reabsorption [20]. Recent experimental evidence suggests that mild dehydration does not necessarily produce major acute changes in sweat electrolyte concentrations [22].

The available evidence does not support the assumption that low sodium intake is the primary cause of most EAH cases. Sodium intake during an ultramarathon was not associated with protection against hyponatremia in the study by Hoffman et al. [11], while the WASSUP study found no significant association between weight-based sodium supplementation and post-race dysnatremia, including hyponatremia [12]. A broader review similarly emphasized the context-dependent role of sodium intake during endurance exercise [18]. More broadly, current evidence suggests that sodium intake and fluid intake should be considered as related but distinct components of hydration strategies during prolonged endurance exercise [18,19,21]. Thus, sodium losses may contribute to changes in serum sodium, but sodium intake cannot be considered independently of fluid consumption and renal water handling.

3.5. Individual and environmental risk factors

The available studies suggest that EAH risk is influenced by body size, sex, exercise duration, and environmental conditions, although associations are not entirely consistent across populations [1,4,5,7]. Smaller body size may increase the relative impact of a given absolute fluid intake [19], while some studies have reported a higher incidence or different clinical presentation among women [4,5]. Experimental work suggests that hormonal status may modify fluid-regulatory responses in women, although its specific relevance to EAH remains uncertain [24].

Longer exercise duration increases the period during which fluid can be consumed and non-osmotic AVP secretion can remain elevated [1,2]. Environmental stress may further modify these responses. EAH has been documented during high-altitude ultramarathon competition, although altitude and heat should be regarded as contextual modifiers rather than independent causes [5].

3.6. Prevention strategies

Current evidence favors prevention based primarily on avoiding excessive fluid intake and individualizing hydration according to thirst, exercise duration, environmental conditions, body size, and expected losses [1,9,21]. There is insufficient evidence that universal sodium supplementation prevents EAH [11,12,18].

Athlete education is therefore an important component of prevention. Variability in hydration beliefs and planned drinking behaviors suggests that some athletes may still associate greater fluid intake or sodium consumption with protection against all forms of exercise-associated dehydration and electrolyte disturbance [13,14]. Available evidence supports avoiding rigid drinking schedules and discouraging intentional overconsumption of fluids [9,15].

Overall, EAH appears to result from an interaction between exercise-induced impairment of free-water excretion, fluid intake, sodium balance, and individual characteristics. Neither fluid intake, body mass change, nor sodium intake alone is sufficient to predict the development of EAH across all endurance populations [1–5,11,16,23].

4. Discussion

The available evidence supports a multifactorial model of exercise-associated hyponatremia (EAH) in which disturbances in water homeostasis develop from the interaction between fluid intake, renal water handling, sodium balance, exercise duration, environmental stress, and individual characteristics. The central finding emerging from both recent and historical studies is that EAH should not be reduced to a simple state of “sodium deficiency” or, conversely, to excessive water intake alone. Rather, the development of clinically relevant hyponatremia appears to depend on whether water retention and sodium losses occur in a physiological context in which free-water excretion is insufficient to maintain normal serum sodium concentration [1,2,8].

This interpretation is particularly important because the epidemiology of EAH varies considerably between endurance disciplines. Marathon runners, triathletes, mountain ultramarathoners, and participants in multistage events are exposed to different combinations of exercise duration, intensity, environmental stress, and opportunities for fluid consumption. The large body of field data summarized by Armstrong et al. demonstrates that both the magnitude of sodium reduction and the clinical presentation of EAH differ according to sporting context [1]. This heterogeneity is also apparent in individual studies. EAH has been documented in marathon running [7,10], triathlon [4], high-altitude ultramarathon [5], and

ultra-trail competition [6,16], but the reported prevalence varies substantially even among superficially similar events. Consequently, prevalence figures from one endurance discipline should not be automatically extrapolated to another.

One of the most consistent observations across the literature is the association between excessive fluid intake and the development of EAH. The Boston Marathon study provided an important early demonstration that runners who gained weight during the race were at substantially greater risk of hyponatremia [10]. Subsequent evidence has reinforced the concept that consumption of fluid in excess of physiological requirements is a major modifiable factor [1,2,8,9]. However, the findings should not be interpreted as meaning that all cases of EAH are caused by overt overhydration. Studies in ultramarathoners have repeatedly identified athletes with hyponatremia who finished with a reduction in body mass [4,5,16]. This observation is difficult to reconcile with a model based exclusively on retained free water and indicates that sodium losses and other regulatory mechanisms may contribute in selected individuals.

The distinction between water-driven and sodium-related mechanisms has practical implications. Sodium is undoubtedly lost during prolonged exercise, particularly through sweating, but the evidence does not support the assumption that inadequate sodium intake is the primary explanation for most EAH cases [1,2,11,18]. The study by Hoffman et al. demonstrated that sodium intake during an ultramarathon did not prevent hyponatremia or other common exercise-associated complaints [11]. Likewise, the WASSUP study examined a weight-based sodium supplementation strategy during ultramarathon running but did not establish sodium replacement as a specific preventive measure against EAH [12]. These findings are consistent with the broader evidence synthesis by Veniamakis et al., in which the role of sodium during endurance exercise was considered important but highly context-dependent [18].

The physiologic explanation for these observations lies in the regulation of water excretion. During prolonged exercise, AVP secretion may remain elevated despite the absence of an osmotic stimulus. This reduces renal free-water clearance and increases the likelihood that ingested hypotonic fluid will be retained [2,8]. The study by Schenk et al. provides contemporary field evidence supporting this mechanism. Copeptin concentrations, reflecting AVP activity, increased markedly during two ultra-endurance mountain races of different distances, demonstrating substantial activation of the vasopressinergic system during prolonged exercise [23]. Importantly, this hormonal response occurred without universal development of hyponatremia. This supports the interpretation that AVP activation is a facilitating mechanism rather than a sufficient cause of EAH by itself. The development of

hyponatremia likely depends on the interaction between AVP-mediated water retention and the amount and composition of fluid consumed.

The role of exercise-induced AVP secretion also helps explain why the same amount of fluid may produce different physiological outcomes in different circumstances. Exercise duration, cardiovascular stress, nausea, pain, environmental exposure, and other non-osmotic stimuli can affect AVP release [2,8,23]. Accordingly, an individualized hydration strategy should not be based solely on a predetermined estimate of sweat loss. The capacity to excrete free water changes dynamically during prolonged exercise, and this may be particularly important during ultramarathons and other events lasting many hours.

The relationship between sweat sodium loss and serum sodium concentration is similarly complex. Sweat sodium concentration varies substantially among athletes as a consequence of individual differences in eccrine gland function and ductal sodium reabsorption [20]. The work of Baker and Wolfe emphasizes that sweat composition is regulated rather than representing a fixed concentration of plasma sodium loss [20]. Recent experimental observations further suggest that mild dehydration does not necessarily produce a major acute alteration in sweat electrolyte concentrations [22]. Therefore, sweat sodium concentration may provide useful contextual information for individualized nutrition planning, but it should not be interpreted as a direct surrogate for the risk of EAH.

This point is particularly relevant to the growing interest in individualized sodium supplementation. Sodium replacement and fluid replacement should not be regarded as interchangeable strategies. Sodium intake may form part of an individualized hydration plan, but its role should be considered in the context of overall fluid intake, exercise duration, environmental conditions, and individual losses [18,19,21]. An athlete can consume an appropriate amount of sodium and still be at risk of hyponatremia if fluid intake substantially exceeds the capacity for water excretion. Conversely, an athlete with considerable sweat sodium losses does not necessarily develop EAH if fluid intake remains physiologically appropriate.

The importance of fluid behavior is therefore difficult to separate from the physiology itself. The literature suggests that athletes do not always base their drinking on thirst or on individualized estimates of fluid needs. Chlíbková et al. demonstrated substantial variation in hydration beliefs and reported behaviors among endurance athletes [13], while Namineni et al. found marked heterogeneity in marathon runners' knowledge and planned hydration practices [14]. Such findings suggest that EAH prevention is partly a behavioral issue. Athletes may follow rigid drinking schedules or intentionally consume more fluid because they believe that

any degree of dehydration is harmful. These practices may be particularly relevant in prolonged events where drinking opportunities are frequent and exercise duration is sufficient for cumulative fluid excess to develop.

The available evidence supports a shift away from universal drinking targets toward individualized strategies. The study by Nolte et al. provides an important practical example: participants allowed to drink ad libitum during a 40-km route march did not develop EAH and did not experience clinically important dehydration [15]. Although military route marching is not identical to competitive endurance sport, the study supports the broader physiological principle that drinking according to thirst may help limit both excessive fluid intake and clinically important dehydration. This concept is also consistent with recommendations emphasizing avoidance of forced fluid consumption [9,21].

At the same time, “drink to thirst” should not be interpreted as a universal prescription independent of context. Endurance events differ considerably in duration, intensity, environmental conditions, access to fluids, and individual sweat rates. The literature therefore supports a more nuanced interpretation in which drinking according to thirst is preferable to forced overconsumption, while athletes may still benefit from understanding their typical fluid requirements and how these change with environmental conditions and exercise duration [1,18,21]. This distinction is important because an overly simplistic formulation of “drink to thirst” could obscure substantial individual variability.

Sex and body size are additional factors that deserve attention, although the available evidence does not support treating either as an isolated, deterministic risk factor. Some studies have reported a greater prevalence or different clinical presentation of EAH among women [4,5], whereas other ultramarathon studies have found less pronounced or inconsistent sex differences [1,7]. Smaller body size may increase the relative effect of a given absolute fluid intake, making body mass an important consideration when interpreting hydration practices [19]. Experimental work on female fluid regulation also suggests that endocrine factors may modify responses to dehydration, although the extent to which these mechanisms translate into clinically relevant differences in EAH susceptibility remains uncertain [24]. Taken together, the evidence supports considering sex and body size as potential modifiers of risk rather than as independent causes.

Environmental conditions further complicate interpretation of EAH risk. Heat increases sweating and cardiovascular strain, while altitude adds hypoxic stress and may alter the physiological response to prolonged exercise. EAH observed during a 161-km high-altitude ultramarathon illustrates that extreme environmental exposure can coexist with substantial

disturbances in serum sodium [5]. However, the available evidence does not justify assuming that heat or altitude directly causes EAH. Instead, environmental stress likely interacts with duration of exercise, fluid intake, sweating, pace, and hormonal regulation [1,2,5]. This is particularly relevant when comparing studies conducted under different environmental conditions, because apparently conflicting prevalence estimates may reflect differences in the physiological context rather than genuine contradictions.

The recent literature also challenges another common assumption: that progressive decreases in serum sodium necessarily indicate impending EAH. In female runners participating in a seven-stage ultramarathon, plasma sodium declined continuously without crossing the threshold for hyponatremia [3]. Similar observations in other ultra-endurance settings suggest that moderate reductions in serum sodium may represent a physiological response to prolonged exercise rather than a pathological state [1,3]. The distinction is important because it demonstrates that the relationship between exercise duration and sodium concentration is continuous, whereas EAH is defined using a specific biochemical threshold. The mere presence of a decline in sodium should therefore not be interpreted in isolation.

A related issue is the coexistence of EAH with other complications of prolonged endurance exercise. The systematic review by Lecina et al. showed that hyponatremia and acute kidney injury are both encountered in ultra-trail populations [6]. Although the available evidence does not establish a simple causal relationship between the two conditions, their coexistence highlights the complexity of the physiological response to prolonged exercise. Athletes participating in ultra-endurance events may simultaneously experience alterations in renal function, fluid balance, thermoregulation, and electrolyte homeostasis. This further supports the need to interpret EAH as part of a broader disturbance of physiological regulation rather than as an isolated sodium abnormality.

From a preventive perspective, the strongest overall message emerging from the literature is that prevention should primarily focus on avoiding excessive fluid intake while recognizing individual variation in sodium losses and physiological responses. There is insufficient evidence to support a universal sodium intake target that guarantees protection from EAH [11,12,18,19]. Likewise, there is little justification for drinking large quantities of fluid solely to avoid any reduction in body mass [1,9,10]. Instead, hydration strategies should consider expected exercise duration, environmental conditions, body size, individual experience, access to fluids, and previous responses to prolonged exercise [18,19,21].

Education may represent one of the simplest and most scalable preventive measures. The persistence of misconceptions surrounding hydration and sodium intake suggests that athletes

may not always distinguish between preventing dehydration and preventing hyponatremia [13,14]. Educational interventions for endurance athletes should therefore emphasize that both insufficient and excessive fluid intake can be problematic, and that sodium supplementation cannot compensate for inappropriate fluid consumption. This principle may be particularly relevant for recreational and less experienced athletes, who may be more likely to rely on generalized rather than individualized hydration advice.

Another important implication is that prevention strategies should acknowledge the difference between endurance sports themselves. The available evidence encompasses conventional road marathons, triathlons, long-distance trail races, mountain ultramarathons, multistage events, and military endurance activities [1,4–7,15,16]. These settings differ substantially in exercise intensity, duration, environmental exposure, access to food and fluids, and typical athlete profile. Consequently, a single hydration recommendation applied uniformly across all endurance sports may be inappropriate. The most defensible approach is to identify common principles—particularly avoidance of forced drinking—while allowing the practical implementation to vary according to the event and athlete.

The present body of evidence also reveals several important uncertainties. First, many studies rely on post-race serum sodium measurements, making it difficult to determine the precise temporal relationship between changes in fluid intake, AVP activity, and the onset of hyponatremia [1,3,23]. Second, individual studies differ in the timing and method of assessing hydration status, making direct comparisons difficult. Third, severe symptomatic EAH is relatively uncommon compared with biochemical EAH, which limits the ability of most field studies to identify reliable predictors of the most clinically important outcomes [1,4]. Finally, there remains no simple physiological marker that can independently predict which athlete exposed to prolonged exercise and fluid intake will develop EAH.

Overall, the evidence supports a model in which EAH develops when prolonged exercise-associated stimulation of water-retaining mechanisms is combined with fluid intake that exceeds the ability to maintain free-water balance, with sodium losses and individual physiological characteristics modifying the final serum sodium concentration. This model accommodates both the classic pattern of EAH associated with body mass gain and the less intuitive cases occurring despite body mass loss [1,3–5,16]. It also explains why sodium supplementation alone has limited preventive value and why hydration education and avoidance of excessive fluid consumption remain central elements of prevention [9,11,13–15,18,21].

For endurance athletes, the practical implication is therefore not that hydration or sodium should be minimized, but that both should be individualized and interpreted in the context of prolonged exercise physiology. The available evidence favors strategies that avoid forced fluid intake, recognize variability in sweat and sodium losses, and account for exercise duration and environmental conditions. This approach is more consistent with the current understanding of EAH than universal prescriptions based solely on body mass, sweat sodium concentration, or a fixed volume of fluid or sodium per hour.

5. Limitations of available evidence

The available evidence on exercise-associated hyponatremia (EAH) has several important limitations that should be considered when interpreting the findings summarized in this review. First, substantial heterogeneity exists between studies with regard to participant characteristics, endurance discipline, exercise duration, environmental conditions, and timing of serum sodium assessment [1,3–7,16]. Studies conducted during marathons, triathlons, mountain ultramarathons, and multistage races therefore cannot always be directly compared. Differences in study design and sampling procedures may also contribute to the wide variation in reported prevalence.

Second, much of the available evidence is observational and based on field studies performed during individual sporting events. Although such studies provide valuable information under real-world conditions, they generally cannot establish causality between individual risk factors and the development of EAH. This is particularly relevant for variables such as fluid intake, sodium intake, body mass changes, and environmental exposure, which are strongly interrelated during prolonged exercise [1,3,5,10,16]. In addition, severe symptomatic EAH represents a relatively small proportion of all reported cases, limiting the statistical power of most studies to identify reliable predictors of clinically serious outcomes [1,4].

Third, assessment of hydration and sodium balance is itself methodologically challenging. Body mass change is widely used as an indirect indicator of fluid balance, but it does not directly distinguish between changes in total body water and other components of body mass [1,3,16]. Similarly, sweat sodium concentration varies considerably between individuals and cannot be interpreted as a direct measure of total sodium requirements without considering sweat rate, exercise duration, and environmental conditions [20,22]. Measurements of AVP are also technically difficult in field studies; consequently, much of the contemporary evidence relies on copeptin as a surrogate marker rather than direct assessment of AVP activity [23].

Another limitation concerns the heterogeneity of hydration and sodium strategies used by athletes. Individual beliefs and behaviors may strongly influence fluid and sodium intake, but these factors are often self-reported or estimated rather than directly measured [13,14]. Furthermore, studies evaluating sodium supplementation have used different doses, timing strategies, and individualized approaches, making direct comparison difficult [11,12,18,21]. This heterogeneity limits the possibility of defining a single sodium or fluid intake strategy that can be universally recommended for all endurance athletes.

Finally, although this review placed particular emphasis on contemporary evidence published from 2020 onward, several foundational studies were necessarily retained because they established key epidemiological and physiological concepts relevant to EAH [8,10,11]. The present review is narrative in nature and therefore does not provide the systematic risk-of-bias assessment or quantitative synthesis characteristic of a systematic review or meta-analysis. The conclusions should consequently be interpreted as an evidence-informed synthesis of heterogeneous literature rather than as definitive estimates of the magnitude of individual risk factors or the effectiveness of specific preventive interventions.

6. Conclusions

Exercise-associated hyponatremia is a multifactorial disturbance of water and sodium homeostasis that may occur during or after prolonged endurance exercise. Current evidence indicates that its development is determined by the interaction between fluid intake, exercise-induced impairment of free-water excretion, sodium balance, exercise duration, environmental conditions, and individual characteristics rather than by isolated sodium depletion or excessive water consumption alone [1,2,23].

Excessive fluid intake relative to the capacity for water excretion remains the most consistently identified modifiable risk factor. However, the occurrence of EAH in athletes who finish endurance events with reduced body mass demonstrates that the condition cannot be explained exclusively by overt fluid retention [3–5,16]. Exercise-induced arginine vasopressin activity, reflected by increases in copeptin during prolonged endurance exercise, provides an important physiological mechanism linking prolonged exercise with impaired free-water excretion [23]. The available evidence does not support universal sodium supplementation as an effective strategy for preventing EAH [11,12,18,21]. Sodium intake should instead be considered together with fluid intake, sweat losses, exercise duration, environmental conditions, and individual characteristics. Prevention should primarily focus on avoiding forced fluid consumption, adopting individualized hydration strategies, and improving athletes'

understanding of the differences between preventing dehydration and preventing hyponatremia [9,13–15].

Further research should prioritize prospective studies performed across different endurance disciplines and environmental conditions, with standardized assessment of fluid intake, body mass, sodium balance, and markers of vasopressin activity. Better characterization of individual susceptibility may help refine prevention strategies and reduce the risk of EAH without promoting either excessive fluid consumption or unnecessary sodium supplementation. Overall, contemporary evidence supports a balanced and individualized approach to hydration during endurance exercise. Rather than aiming to eliminate all exercise-related reductions in body mass or relying on fixed sodium prescriptions, athletes and practitioners should focus on maintaining an appropriate balance between fluid intake, physiological losses, and the body's capacity to regulate water and sodium homeostasis.

Supplementary Materials

Not applicable.

Author Contributions

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Jarosław Dudek – investigation, formal analysis, project administration

Damian Grębosz – formal analysis, resources

Aleksandra Kinga Łysak – writing, investigation

Krzysztof Lis – resources, visualisation

Aleksandra Kalisz - writing, investigation

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