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Exercise-Associated Hyponatremia in Endurance Athletes: Recognition, Prevention and Clinical Management – A Narrative Review

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Corresponding AuthorPiotr Ziajka, e-mail: ziajkapiotr@gmail.com**Abstract**

Background: Exercise-associated hyponatremia (EAH) is a potentially life-threatening electrolyte disorder occurring during or within 24 hours after prolonged endurance exercise. Despite increasing awareness, EAH remains underrecognized because its clinical presentation frequently overlaps with dehydration and other exercise-related conditions.

Aim: This narrative review aims to summarize current evidence regarding the epidemiology, pathophysiology, risk factors, clinical presentation, diagnosis, management, and prevention of exercise-associated hyponatremia in endurance athletes.

Materials and Methods: A narrative review of the literature was conducted using peer-reviewed publications retrieved from PubMed and Google Scholar. Original studies, review articles, international consensus statements, and clinical practice guidelines addressing exercise-associated hyponatremia in endurance athletes were analyzed and synthesized to provide a comprehensive overview of current knowledge.

Results: Current evidence indicates that EAH primarily results from excessive hypotonic fluid intake combined with impaired renal free water excretion due to persistent arginine vasopressin secretion. Individualized hydration strategies based on physiological thirst represent the most

effective preventive approach, whereas prompt administration of intravenous 3% hypertonic saline remains the treatment of choice in severe symptomatic cases.

Conclusions: Early recognition, appropriate differential diagnosis, and evidence-based management are essential for preventing severe neurological complications associated with EAH. Improved education of athletes, coaches, and healthcare professionals may further reduce the incidence and clinical consequences of this condition.

Keywords: exercise-associated hyponatremia; endurance athletes; endurance exercise; hydration; sports medicine

1. Introduction

Exercise-associated hyponatremia (EAH) is a potentially life-threatening electrolyte disorder characterized by a serum sodium concentration below 135 mmol/L occurring during or within 24 hours after prolonged physical activity. Although initially recognized primarily among marathon runners, EAH has been reported in a wide range of endurance disciplines such as ultramarathons, triathlons, cycling events, open-water swimming, and military training. As participation in recreational and competitive endurance sports continues to increase worldwide, EAH has become an increasingly important concern in sports medicine and emergency care [1, 3, 8, 11].

The pathogenesis of EAH is multifactorial, with excessive hypotonic fluid intake and impaired renal water excretion due to persistent secretion of arginine vasopressin representing the principal mechanisms. Additional contributing factors include prolonged exercise duration, low body mass, female sex, use of non-steroidal anti-inflammatory drugs (NSAIDs), and exposure to high ambient temperatures. Clinical manifestations range from mild symptoms, such as nausea, headache, and dizziness, to severe neurological abnormalities including seizures, cerebral edema, coma, and, in rare cases, death [1, 3, 4, 9, 13].

Despite growing awareness, EAH remains underrecognized because its clinical presentation frequently overlaps with other exercise-related conditions, including dehydration, exertional heat illness, and exercise-associated collapse. Misinterpretation of symptoms may lead to inappropriate management, particularly the administration of hypotonic or isotonic fluids to athletes with symptomatic hyponatremia, potentially worsening cerebral edema and increasing morbidity [1, 3, 12, 19].

Recent international guidelines have emphasized the importance of early recognition, appropriate differential diagnosis, and prompt treatment of symptomatic EAH, particularly in prehospital and field settings where laboratory testing may be unavailable. Furthermore, preventive strategies based on individualized hydration and drinking according to thirst have challenged the traditional recommendation of aggressive fluid replacement during endurance exercise [1, 2, 14, 18].

The aim of this narrative review is to summarize current evidence regarding the epidemiology, pathophysiology, clinical presentation, diagnosis, management, and prevention of exercise-associated hyponatremia in endurance athletes, with particular emphasis on practical recommendations for healthcare professionals involved in sports medicine and emergency care.

2. Epidemiology

The true incidence of exercise-associated hyponatremia (EAH) remains difficult to determine because many affected athletes are either asymptomatic or present with nonspecific symptoms that may go unrecognized. Consequently, the reported prevalence varies considerably depending on the type of sporting event, environmental conditions, participant characteristics, and diagnostic criteria used across studies [1, 3, 8, 11].

EAH has been documented in numerous endurance disciplines, including marathons, ultramarathons, triathlons, long-distance cycling, open-water swimming, and military training exercises. Among marathon runners, the reported prevalence generally ranges from 5% to 15%, although substantially higher rates have been observed in athletes participating in ultradistance events lasting several hours or days. In some ultramarathon cohorts, biochemical hyponatremia has been identified in more than one-quarter of participants, while severe symptomatic cases remain relatively uncommon [8, 11, 20, 21].

The growing popularity of endurance sports has contributed to an increasing number of reported EAH cases over the past two decades. Recreational athletes may be particularly vulnerable because they are often encouraged to consume large volumes of fluids before and during exercise, despite growing evidence that excessive hydration represents one of the principal risk factors for the development of EAH. Educational campaigns promoting individualized

hydration strategies have improved awareness; however, exercise-associated hyponatremia continues to be reported at major international sporting events [1, 7, 18, 22].

The incidence of EAH is influenced by several factors, including race duration, environmental temperature, hydration practices, and athlete experience. Longer events are consistently associated with a greater risk of hyponatremia, largely because they provide more opportunities for excessive fluid intake and prolonged secretion of arginine vasopressin. Furthermore, the prevalence of EAH may differ between elite and recreational competitors, with slower athletes generally demonstrating a higher risk due to longer exercise duration and greater cumulative fluid consumption [8, 9, 11, 20].

Although most cases of EAH are mild and resolve without permanent complications, severe hyponatremia accompanied by cerebral edema may result in significant morbidity or even death if not recognized promptly. Therefore, continued surveillance, education, and implementation of evidence-based hydration recommendations remain essential components of athlete safety during endurance events [1, 2, 14].

3. Pathophysiology

Exercise-associated hyponatremia develops when fluid intake exceeds the kidneys' ability to excrete free water, leading to dilution of serum sodium. During prolonged endurance exercise, physiological adaptations such as activation of the renin–angiotensin–aldosterone system (RAAS), increased arginine vasopressin (AVP) secretion, and reduced renal blood flow help preserve circulating blood volume and maintain cardiovascular stability. However, persistent AVP secretion limits renal water excretion, increasing the risk of dilutional hyponatremia when excessive hypotonic fluids are consumed [1, 3, 4, 9, 13].

Excessive intake of hypotonic fluids is considered the primary mechanism underlying EAH. For many years, athletes were encouraged to drink as much as possible to prevent dehydration, but current evidence indicates that overhydration poses a greater risk than mild dehydration in most endurance events. Consequently, modern guidelines recommend individualized hydration strategies based on thirst rather than fixed drinking schedules [1, 5, 16].

Several additional factors may further increase the risk of EAH, including prolonged exercise duration, high ambient temperature, use of non-steroidal anti-inflammatory drugs (NSAIDs), low body mass, and excessive sodium loss through sweat. Although sodium depletion may contribute in some individuals, current evidence suggests that water retention, rather than sodium loss alone, is the principal mechanism responsible for the development of EAH [1, 3, 4, 6, 9].

The neurological complications of EAH result primarily from osmotic water shifts between the extracellular and intracellular compartments. As serum sodium concentration decreases, plasma osmolality falls below intracellular osmolality, creating an osmotic gradient that promotes movement of water into brain cells. The resulting cerebral edema increases intracranial pressure and may lead to headache, nausea, vomiting, confusion, seizures, coma, brain herniation, and ultimately death in untreated cases. Because the skull provides limited space for tissue expansion, even relatively small increases in brain volume may produce significant neurological deterioration, emphasizing the importance of early recognition and appropriate treatment of symptomatic EAH. [1,4,19,24]

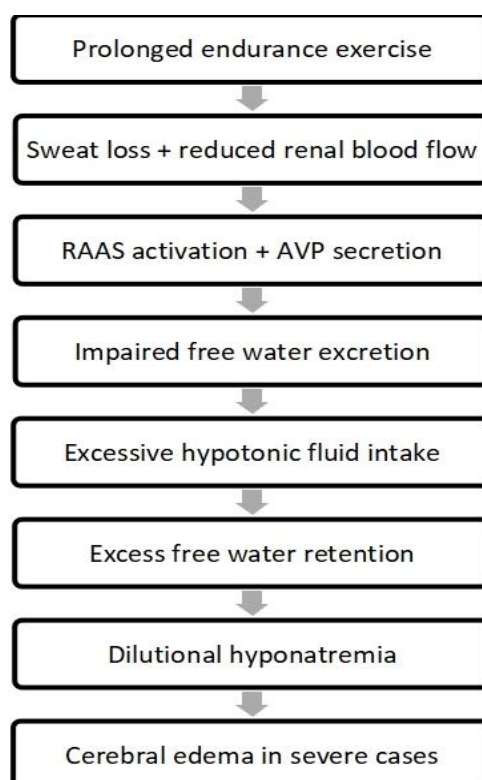


Figure 1. Pathophysiological mechanisms leading to exercise-associated hyponatremia

VP – arginine vasopressin; RAAS – renin–angiotensin–aldosterone system.

4. Risk Factors

Exercise-associated hyponatremia results from a complex interaction between physiological, environmental, and behavioral factors. Although excessive hypotonic fluid intake remains the principal cause, multiple variables may further increase the likelihood of developing EAH during prolonged endurance exercise [1, 3, 9, 18].

Prolonged exercise duration is one of the strongest predictors of EAH. Athletes participating in events lasting more than four hours are consistently at greater risk, primarily because longer exercise provides more opportunities for excessive fluid consumption and prolonged non-osmotic arginine vasopressin secretion. Slower race pace has similarly been associated with an increased incidence of EAH, particularly among recreational endurance athletes [8, 9, 11, 20].

Several individual characteristics have also been associated with an increased risk of EAH. Lower body mass may increase susceptibility to dilutional hyponatremia because a given volume of ingested fluid represents a larger proportion of total body water. Earlier studies suggested that female athletes were at greater risk; however, more recent evidence indicates that this association is largely explained by differences in body size, race duration, and hydration practices rather than sex itself [3, 6, 11, 18].

Environmental conditions and medication use may further contribute to the development of EAH. High ambient temperatures encourage greater fluid intake, while non-steroidal anti-inflammatory drugs (NSAIDs) may impair renal free water excretion by potentiating the effects of arginine vasopressin. Consequently, athletes exercising in hot conditions while consuming excessive amounts of hypotonic fluids should be considered at particularly high risk of developing EAH [1, 5, 7, 18].

Current evidence suggests that behavioral factors remain the most important modifiable determinants of EAH. Drinking beyond thirst and adhering to predetermined hydration schedules have consistently been associated with an increased incidence of hyponatremia. Therefore, individualized hydration strategies based on physiological thirst are currently recommended for most endurance athletes [1, 5, 18, 22].

Risk factors	Mechanism contributing to EAH
Excessive hypotonic fluid intake	Dilution of serum sodium due to water overload
Prolonged exercise duration	Increased risk of overhydration and persistent AVP secretion
Slow race pace	Longer exposure to fluid consumption during the event
High ambient temperature	Greater sweat loss and fluid intake
Female sex*	Frequently associated with lower body mass and longer exercise duration
Low body mass	Smaller total body water volume
NSAID use	Potential of AVP action and reduced renal free water excretion
Inexperienced athlete	Inappropriate hydration strategies and overdrinking

Table 1. Major risk factors for exercise-associated hyponatremia

EAH – exercise-associated hyponatremia; AVP – arginine vasopressin;

NSAIDs – non-steroidal anti-inflammatory drugs

Collectively, these findings indicate that behavioral factors, particularly excessive fluid intake, remain the most important modifiable determinants of EAH. Recognition of high-risk athletes may facilitate early preventive interventions and reduce the incidence of this potentially life-threatening condition [1, 3, 18].

5. Clinical Presentation and Diagnosis

The clinical presentation of exercise-associated hyponatremia (EAH) ranges from mild, nonspecific symptoms to severe neurological impairment. Early manifestations include nausea, headache, dizziness, fatigue, and generalized weakness, which may be difficult to distinguish from dehydration or normal post-exercise fatigue. Consequently, EAH is frequently underrecognized during endurance events, particularly in field settings where laboratory testing is unavailable [1, 3, 6, 12].

As serum sodium concentration decreases, neurological symptoms become increasingly prominent due to the development of cerebral edema. Athletes may present with vomiting, confusion, agitation, altered mental status, gait instability, or seizures. Severe EAH represents

a medical emergency because progressive cerebral edema may lead to respiratory failure, coma, brain herniation, and death if appropriate treatment is delayed [1, 3, 6, 19].

The diagnosis of EAH should be suspected in any athlete who develops neurological symptoms during or within 24 hours after prolonged endurance exercise, particularly when there is a history of excessive fluid intake or weight gain during competition. Although measurement of serum sodium concentration remains the diagnostic gold standard, treatment decisions should not be delayed in critically ill patients when EAH is strongly suspected [1, 2, 6, 25].

Early recognition of EAH relies on careful assessment of symptoms, hydration history, exercise duration, and body weight changes whenever available. Combining clinical findings with point-of-care serum sodium measurement, when accessible, enables rapid diagnosis and facilitates appropriate treatment in both prehospital and hospital settings. Point-of-care blood analyzers, when available during endurance events, may facilitate early diagnosis and appropriate treatment decisions. Because the initial symptoms of EAH frequently mimic dehydration, assessment of hydration history and body weight changes may provide valuable diagnostic clues [1, 2, 6, 15].

6. Differential Diagnosis

The differential diagnosis of exercise-associated hyponatremia (EAH) includes several exercise-related conditions that may present with similar clinical manifestations but require substantially different management. The most important conditions include dehydration, exertional heat illness, exercise-associated collapse, and exercise-associated muscle cramps. Because inappropriate treatment may significantly worsen patient outcomes, rapid differentiation between these conditions is essential, particularly in prehospital settings where laboratory testing is often unavailable [1, 2, 14, 15].

Dehydration and EAH frequently share nonspecific symptoms such as fatigue, dizziness, nausea, and reduced exercise performance. However, athletes with dehydration typically demonstrate body weight loss, thirst, tachycardia, and signs of hypovolemia, whereas patients with EAH often have stable or increased body weight due to excessive fluid intake. Administration of isotonic or hypotonic fluids is appropriate in dehydration but may aggravate cerebral edema in symptomatic EAH [1, 3, 15, 19, 24].

Exertional heat stroke should also be considered in athletes presenting with altered mental status after prolonged exercise. Unlike EAH, heat stroke is characterized by marked hyperthermia, central nervous system dysfunction, and multiorgan injury requiring immediate cooling. Although both conditions may coexist, recognition of hyponatremia is crucial because fluid management differs considerably. Whenever available, point-of-care serum sodium measurement can facilitate rapid differentiation and guide appropriate treatment [1, 2, 14, 25].

In clinical practice, a careful history regarding fluid intake, exercise duration, body weight changes, and medication use, combined with neurological assessment and serum sodium measurement when available, provides the most reliable approach to distinguishing EAH from other exercise-related disorders. Early recognition of EAH is essential to avoid inappropriate fluid administration and reduce the risk of severe neurological complications [1, 2, 3, 25].

7. Management

Management of exercise-associated hyponatremia depends primarily on symptom severity rather than serum sodium concentration alone. The initial assessment should include evaluation of neurological status, hydration history, vital signs, and, whenever available, measurement of serum sodium concentration. Athletes presenting with mild symptoms and preserved mental status may often be managed conservatively, whereas severe neurological manifestations require immediate intervention [1, 2, 3, 25].

Athletes with mild EAH, presenting with symptoms such as nausea, dizziness, or headache without neurological impairment, should discontinue fluid intake and be observed closely. Oral intake should be restricted until spontaneous diuresis occurs, allowing excess free water to be excreted. In most cases, symptoms resolve without the need for intravenous fluid therapy. Routine administration of isotonic fluids is not recommended, as it may worsen hyponatremia in athletes with water overload [1, 2, 5, 14].

Severe EAH, characterized by altered mental status, seizures, coma, or signs of cerebral edema, constitutes a medical emergency. Current consensus guidelines recommend administration of a 100-mL bolus of 3% hypertonic saline over approximately 10 minutes, which may be repeated up to two additional times in patients with persistent severe neurological symptoms.. Early treatment is associated with rapid neurological improvement and may prevent progression to

brain herniation or death. Importantly, initiation of hypertonic saline therapy should not be delayed in patients with a strong clinical suspicion of severe EAH, even if immediate laboratory confirmation is unavailable [1, 2, 19, 23].

Hypertonic saline rapidly increases plasma osmolality, promoting the movement of water from the intracellular to the extracellular compartment, thereby reducing cerebral edema and intracranial pressure [4, 5, 13].

The prognosis of exercise-associated hyponatremia largely depends on the severity of neurological involvement and the timing of appropriate treatment. Most athletes with mild EAH recover completely following fluid restriction and spontaneous diuresis. In contrast, delayed recognition of severe symptomatic EAH may result in irreversible neurological injury, brain herniation, or death. Prompt administration of hypertonic saline is therefore the most important determinant of a favorable clinical outcome [1,2,19,25].

Clinical finding	Recommended management
Mild EAH	Fluid restriction and observation
Severe EAH	Immediate IV 3% hypertonic saline
Hypotonic/isotonic fluids	Avoid until EAH is excluded
Hospital management	Neurological and serum sodium monitoring
Prevention of recurrence	Education and individualized hydration

Table 2. Practical management of exercise-associated hyponatremia according to clinical presentation.

EAH – exercise-associated hyponatremia; IV – intravenous

Hospital management should include continuous neurological monitoring, repeated assessment of serum sodium concentration, and careful correction of hyponatremia to avoid complications associated with overly rapid sodium normalization. Additional supportive treatment should be individualized according to the patient's clinical condition and the presence of concomitant exercise-related illnesses [2, 6, 24, 25].

Prehospital providers, sports medicine physicians, and event medical teams play a critical role in the early recognition and management of EAH. Prompt identification of high-risk athletes, avoidance of unnecessary fluid administration, and rapid initiation of appropriate therapy

remain the cornerstone of successful management and contribute to reducing morbidity and mortality associated with this condition [1, 2, 14].

8. Prevention

Prevention of exercise-associated hyponatremia primarily focuses on avoiding excessive fluid intake during prolonged endurance exercise. Current recommendations discourage predetermined hydration schedules and instead promote individualized fluid replacement based on physiological thirst. This approach has been shown to reduce the risk of overhydration while maintaining adequate hydration status in most athletes [1, 5, 18, 22].

Education of athletes, coaches, race organizers, and medical personnel plays a crucial role in preventing EAH. Participants should be informed about the potential dangers of overdrinking, early symptoms of hyponatremia, and the importance of recognizing that excessive fluid intake may be more harmful than mild dehydration during endurance events. Event organizers should also ensure that hydration recommendations are consistent with current evidence-based guidelines [1, 2, 14, 18].

Preventive strategies should be individualized according to exercise duration, environmental conditions, sweat rate, and previous athletic experience. Monitoring body weight before and after prolonged events may help identify athletes at risk of overhydration, while avoiding unnecessary use of non-steroidal anti-inflammatory drugs (NSAIDs) may further reduce the likelihood of developing EAH. Current evidence also suggests that sodium supplementation alone does not prevent exercise-associated hyponatremia when excessive fluid intake persists and therefore should not replace individualized hydration strategies based on physiological thirst [1, 5, 17, 18].

Although EAH cannot be completely eliminated, implementation of appropriate hydration strategies together with improved education and early recognition may substantially decrease its incidence and reduce the risk of severe neurological complications [1, 3, 18].

9. Discussion

Exercise-associated hyponatremia remains an important challenge in sports medicine despite substantial advances in understanding its pathophysiology and prevention. The findings summarized in this review consistently indicate that excessive hypotonic fluid intake combined with persistent arginine vasopressin secretion represents the principal mechanism responsible for the development of EAH. These observations have fundamentally changed hydration recommendations over the past two decades, shifting clinical practice from aggressive fluid replacement toward individualized hydration strategies based on physiological thirst.

From a clinical perspective, early recognition of EAH remains essential because its initial manifestations frequently resemble dehydration or exertional heat illness. Misdiagnosis may result in inappropriate fluid administration, potentially worsening cerebral edema and neurological injury. Consequently, healthcare professionals responsible for medical care during endurance events should be familiar with the current diagnostic criteria and evidence-based treatment recommendations, particularly the prompt use of intravenous hypertonic saline in athletes presenting with severe neurological symptoms.

Although current recommendations are supported by growing evidence, several aspects of EAH remain incompletely understood. The relative contribution of sodium loss through sweat, individual susceptibility, environmental factors, and hormonal responses may vary considerably between athletes. Furthermore, the optimal hydration strategy may differ according to exercise duration, climate, and competition characteristics, highlighting the need for individualized preventive approaches rather than universal hydration protocols.

Future research should focus on improving risk stratification, identifying athletes particularly susceptible to EAH, and optimizing preventive strategies for different endurance disciplines. Further prospective studies are also required to evaluate hydration practices, educational interventions, and field-based diagnostic tools capable of facilitating early recognition and treatment during endurance events.

10. Conclusions

Exercise-associated hyponatremia remains one of the most important and potentially life-threatening medical complications encountered during prolonged endurance exercise. Although its pathophysiology is complex, current evidence indicates that excessive hypotonic fluid intake combined with impaired renal free water excretion represents the principal mechanism responsible for its development. Early recognition of clinical symptoms, prompt differentiation from other exercise-related conditions, and appropriate management are essential to prevent severe neurological complications.

Current evidence supports individualized hydration strategies based on physiological thirst rather than predetermined drinking schedules. Education of athletes, coaches, and healthcare professionals remains fundamental for reducing the incidence of EAH and improving patient outcomes. Continued research is needed to further optimize prevention strategies and refine clinical management in different endurance sports. As participation in endurance sports continues to increase worldwide, improved awareness of EAH among athletes and healthcare professionals will remain essential for minimizing preventable complications and optimizing athlete safety [1, 3, 4, 18, 25].

Disclosure

Author Contributions

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Formal analysis: PZ, AZ, MŁ

Investigation: AGS, HL

Methodology: IF, MS

Resources: IF, AZY

Supervision: PZ, AM, AGS

Visualization: AT, MS

Writing - rough preparation: PZ, HL, AZY, MŁ

Writing - review & editing: AZ, GS, IF, AT

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Bibliography

1. Bennett BL, Hew-Butler T, Rosner MH, Myers T, Lipman GS. Wilderness Medical Society clinical practice guidelines for the management of exercise-associated hyponatremia: 2019 update. *Wilderness Environ Med.* 2020;31(1):50-62. doi:10.1016/j.wem.2019.11.003.
2. Hew-Butler T, Rosner MH, Fowkes-Godek S, Dugas JP, Hoffman MD, Lewis DP, et al. Statement of the Third International Exercise-Associated Hyponatremia Consensus Development Conference, Carlsbad, California, 2015. *Clin J Sport Med.* 2015;25(4):303-320.
3. Rosner MH. Exercise-associated hyponatremia. *Trans Am Clin Climatol Assoc.* 2019;130:76-87.
4. Altieri B, Aini I, Cannavale G, Magnelli C, Mancini C, Zamponi V, et al. Pathophysiology and treatment of exercise-associated hyponatremia. *J Endocrinol Invest.* 2025. doi:10.1007/s40618-025-02673-7.

5. Hew-Butler T, Loi V, Pani A, Rosner MH. Exercise-associated hyponatremia: 2017 update. *Front Med (Lausanne)*. 2017;4:21. doi:10.3389/fmed.2017.00021.
6. Rosner MH, Kirven J. Exercise-associated hyponatremia. *Clin J Am Soc Nephrol*. 2007;2(1):151-161. doi:10.2215/CJN.02730806.
7. Rogers IR, Hew-Butler T. Exercise-associated hyponatremia: overzealous fluid consumption. *Wilderness Environ Med*. 2009;20(2):139-143. doi:10.1580/08-WEME-CON-231R2.1.
8. Almond CS, Shin AY, Fortescue EB, Mannix RC, Wypij D, Binstadt BA, et al. Hyponatremia among runners in the Boston Marathon. *N Engl J Med*. 2005;352(15):1550-1556. doi:10.1056/NEJMoa043901.
9. Noakes TD, Sharwood K, Speedy D, Hew T, Reid S, Dugas J, et al. Three independent biological mechanisms cause exercise-associated hyponatremia: evidence from 2,135 weighed competitive athletic performances. *Proc Natl Acad Sci U S A*. 2005;102(51):18550-18555. doi:10.1073/pnas.0509096102.
10. Speedy DB, Noakes TD, Schneider C. Exercise-associated hyponatremia: a review. *Emerg Med (Fremantle)*. 2001;13(1):17-27. doi:10.1046/j.1442-2026.2001.00173.x.
11. Klingert M, Nikolaidis PT, Weiss K, Thuany M, Chlábková D, Knechtle B. Exercise-associated hyponatremia in marathon runners. *J Clin Med*. 2022;11(22):6775. doi:10.3390/jcm11226775.
12. Johnson KB, Connolly CP, Cho SP, Miller TK, Sallis RE, Hiller WDB. Clinical presentation of exercise-associated hyponatremia in male and female IRONMAN triathletes over three decades. *Scand J Med Sci Sports*. 2023;33:1841-1849. doi:10.1111/sms.14401.
13. Hew-Butler T. Exercise-associated hyponatremia. *Front Horm Res*. 2019;52:178-189. doi:10.1159/000493247.
14. Jonas CE, Arnold MJ. Exercise-associated hyponatremia: updated guidelines from the Wilderness Medical Society. *Am Fam Physician*. 2021;103(4):252-253.
15. Buck E, McAllister R, Schroeder JD. Exercise-associated hyponatremia. In: *StatPearls [Internet]*. Treasure Island (FL): StatPearls Publishing; 2023.
16. Noakes TD. Hyponatremia in distance runners: fluid and sodium balance during exercise. *Curr Sports Med Rep*. 2002;1(4):197-207. doi:10.1249/00149619-200208000-00003.
17. Hew-Butler TD, Sharwood K, Collins M, Speedy D, Noakes T. Sodium supplementation is not required to maintain serum sodium concentrations during an

- Ironman triathlon. Br J Sports Med. 2006;40(3):255-259. doi:10.1136/bjsm.2005.022418.
18. Seal AD, Kavouras SA. A review of risk factors and prevention strategies for exercise-associated hyponatremia. Auton Neurosci. 2022;238:102930. doi:10.1016/j.autneu.2021.102930.
 19. Ayus JC, Varon J, Arieff AI. Hyponatremia, cerebral edema, and noncardiogenic pulmonary edema in marathon runners. Ann Intern Med. 2000;132(9):711-714. doi:10.7326/0003-4819-132-9-200005020-00005.
 20. Hoffman MD, Hew-Butler T, Stuempfle KJ. Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners. Med Sci Sports Exerc. 2013;45(4):784-791. doi:10.1249/MSS.0b013e31827985a8.
 21. Hoffman MD, Fogard K, Winger J, Hew-Butler T, Stuempfle KJ. Characteristics of 161-km ultramarathon finishers developing exercise-associated hyponatremia. Res Sports Med. 2013;21(2):164-175.
 22. Winger JM, Dugas JP, Dugas LR. Beliefs about hydration and physiology drive drinking behaviours in runners. Br J Sports Med. 2011;45(8):646-649.
 23. Rogers IR, Hook G, Stuempfle KJ, Hoffman MD, Hew-Butler T. An intervention study of oral versus intravenous hypertonic saline administration in ultramarathon runners with exercise-associated hyponatremia: a preliminary randomized trial. Clin J Sport Med. 2011;21(3):200-203. doi:10.1097/JSM.0b013e31821a6450.
 24. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342(21):1581-1589. doi:10.1056/NEJM200005253422107.
 25. Verbalis JG, Goldsmith SR, Greenberg A, Korzelius C, Schrier RW, Sterns RH, et al. Diagnosis, evaluation, and treatment of hyponatremia: expert panel recommendations. Am J Med. 2013;126(10 Suppl 1):S1-S42. doi:10.1016/j.amjmed.2013.07.006.