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Exercise-Associated Muscle Cramps in Athletes: Beyond Dehydration and Electrolyte Loss — A Narrative Review

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Abstract

Introduction and Purpose

Exercise-associated muscle cramps (EAMC) are painful, involuntary contractions occurring during or shortly after physical activity. Their mechanisms remain uncertain, and dehydration or electrolyte loss alone does not explain many episodes. This review summarizes current

evidence on the epidemiology, mechanisms, risk factors, treatment, and prevention of EAMC in athletes.

Materials and Methods

A structured PubMed/MEDLINE search was performed, with the final search completed on 13 September 2026. Priority was given to studies published between 2010 and 2026, with earlier landmark publications included when relevant.

Results

EAMC are common in endurance sports and usually affect heavily recruited muscles late in prolonged exercise. Evidence does not consistently support dehydration or systemic electrolyte depletion as a universal cause. Neuromuscular fatigue and altered neuromuscular control provide a stronger framework, although EAMC are likely multifactorial. Previous EAMC are the most consistent predictor of recurrence. Gentle static stretching remains the best-supported acute treatment; routine pre-exercise stretching or electrolyte supplementation has not shown consistent preventive benefit.

Conclusion

EAMC are best viewed as a multifactorial condition shaped by neuromuscular fatigue, individual susceptibility, and exercise-specific demands. Management and prevention should therefore be individualized.

Keywords: exercise-associated muscle cramps; athletes; neuromuscular fatigue; dehydration; electrolyte balance; sports medicine

1. Introduction

Exercise-associated muscle cramps (EAMC) are painful, involuntary contractions of skeletal muscle occurring during or shortly after physical activity. They are reported across many sports, but are particularly relevant in endurance disciplines, where prolonged muscular work and fatigue create conditions in which cramping commonly occurs. Although most episodes are short-lived and resolve without lasting consequences, they can interrupt competition, impair performance, and cause considerable discomfort. Despite their frequency, the mechanisms responsible for EAMC remain uncertain [1].

Distance runners and triathletes are among the groups in whom EAMC have been studied most extensively. Cramping often develops later in competition and commonly affects heavily recruited muscle groups such as the calves, hamstrings, and quadriceps [2]. These patterns have drawn attention to the role of exercise intensity, local muscle fatigue, and individual susceptibility.

For many years, EAMC were primarily attributed to dehydration and the loss of sodium and other electrolytes through sweat. This explanation remains widely accepted in sport and continues to influence preventive strategies. However, prospective studies have not consistently shown greater dehydration, larger body mass losses, or clinically relevant differences in serum electrolyte concentrations in athletes who cramp compared with those who do not [3,4]. Experimental studies have also shown that substantial hypohydration does not necessarily increase susceptibility to electrically induced muscle cramping [5].

An alternative explanation focuses on altered neuromuscular control. According to this model, muscular fatigue alters the balance between excitatory input from muscle spindles and inhibitory feedback from Golgi tendon organs, increasing alpha motor neuron excitability and promoting sustained contraction [6]. This concept fits several clinical observations, including the tendency of EAMC to affect repeatedly loaded muscles and to occur when exercise demands exceed what the athlete is adequately prepared to tolerate. Even so, neuromuscular fatigue alone does not explain why some athletes repeatedly cramp while others exposed to similar conditions do not.

Recent evidence supports a broader interpretation. A 2026 systematic review and meta-analysis including 11,274 participants identified previous EAMC as the most consistent predictor of future episodes, while most investigated hydration, anthropometric, training, and metabolic variables showed no reproducible association [7]. This reinforces the view that EAMC are unlikely to result from a single universal mechanism and that individual susceptibility may be central to their development.

The uncertainty surrounding both mechanism and prevention has practical consequences. Athletes with recurrent cramps are still often advised to increase fluid or electrolyte intake, despite limited evidence that these measures prevent EAMC in most cases [1,7]. At the same time, interventions aimed at reducing neuromuscular fatigue or improving event-specific conditioning remain incompletely studied [1].

The aim of this narrative review is to summarize current evidence on exercise-associated muscle cramps in athletes, including their epidemiology, pathophysiology, risk factors, clinical assessment, acute management, and prevention. Particular attention is given to the limitations of the traditional dehydration and electrolyte depletion hypothesis, the evidence derived from experimental models, and the growing recognition of neuromuscular fatigue and individual susceptibility.

2. Materials and Methods

A structured literature search of PubMed/MEDLINE was performed, with the final search completed on 12 September 2026. The search focused on exercise-associated muscle cramps in athletes and physically active individuals and included terms related to EAMC, dehydration, electrolyte balance, neuromuscular fatigue, risk factors, treatment, and prevention.

Priority was given to studies published between January 2010 and September 2026, including observational and experimental studies, systematic reviews, meta-analyses, and clinically relevant narrative reviews. Earlier publications were included when they provided important mechanistic or foundational evidence. Reference lists of selected articles were also screened for additional relevant studies.

Studies addressing the epidemiology, pathophysiology, experimental assessment of cramp susceptibility, risk factors, clinical assessment, acute management, or prevention of EAMC were considered. Evidence derived directly from athletic populations was prioritized. Because of substantial heterogeneity in study populations, exercise protocols, definitions of EAMC, and outcome measures, the evidence was synthesized narratively.

3. Clinical Characteristics and Epidemiology of Exercise-Associated Muscle Cramps

Exercise-associated muscle cramps are reported across a wide range of sports, but their frequency is difficult to define precisely. Estimates vary with the population studied, exercise setting, definition of EAMC, and method used to identify cases. Studies based on self-reported history capture episodes that never require medical attention, whereas medical-tent records include mainly athletes whose symptoms are severe enough to seek assistance. Prevalence and incidence figures should therefore be interpreted in the context of how EAMC were assessed [1,2].

One of the largest studies of distance runners included more than 71,000 entrants in 21.1- and 56-km road races. Overall lifetime prevalence of EAMC was 12.8%, with previous cramping reported by 20.0% of entrants in the 56-km event and 8.5% of half-marathon runners [2]. These figures describe previous susceptibility rather than the incidence of cramping during the race itself, but they illustrate the substantial differences that can be observed between endurance populations.

The anatomical distribution is relatively characteristic. In the same cohort, the calf was the most frequently affected muscle group, accounting for 53.1% of reported episodes, followed by the hamstrings and quadriceps. Runners entering the longer event more often reported involvement of the hamstrings and quadriceps [2]. Similar observations in other endurance populations suggest that EAMC tend to involve muscles that are heavily recruited during the activity [1].

Timing during competition also follows a recognizable pattern. Almost half of reported EAMC in distance runners began during the final quarter of a race, while some developed shortly after finishing. Episodes occurring early in competition were less common. Most were classified as mild or moderate, although more severe cramps were also reported [2]. The predominance of episodes later in prolonged exercise is consistent with the increasing muscular demands accumulated over the course of an event.

Data from ultraendurance competition provide a complementary perspective. An analysis of 49,530 participants in the IRONMAN World Championship between 1989 and 2019 identified 2,863 athletes treated for EAMC, corresponding to 57.8 cases per 1,000 race participants [8]. Because these data were derived from medical records, they reflect cramps requiring medical attention rather than all episodes occurring during competition. Incidence was similar in men and women [8].

Higher frequencies have been reported when athletes are questioned directly after demanding endurance events. Comparisons between studies remain difficult, however, because definitions of cramping, follow-up periods, and the inclusion of near-cramping symptoms vary considerably [1]. Methodological differences probably account for part of the wide range of prevalence estimates reported in the literature.

Endurance running and triathlon dominate the available evidence, but EAMC have also been described in other sporting settings involving repeated or intense muscular activity [1]. Reliable

comparisons between disciplines remain limited because prospective surveillance using consistent case definitions is uncommon.

Taken together, the available data indicate that EAMC most often involve actively recruited muscle groups and frequently occur during the later stages of prolonged exercise. Their reported frequency varies substantially between studies, making study design and the method of case ascertainment important when interpreting epidemiological estimates.

4. Pathophysiology of Exercise-Associated Muscle Cramps

The pathophysiology of exercise-associated muscle cramps remains incompletely understood. Two explanations have dominated the literature: dehydration and electrolyte depletion, and altered neuromuscular control. Neither fully accounts for all episodes, although current evidence provides stronger support for mechanisms involving neuromuscular fatigue and altered motor neuron excitability [1,6].

4.1. Dehydration and Electrolyte Depletion

The traditional hypothesis proposes that prolonged sweating leads to fluid and electrolyte losses that increase neuromuscular excitability and eventually trigger cramping [6]. Its persistence is understandable, as EAMC are often reported during prolonged exercise under conditions associated with substantial sweating.

Prospective studies, however, have not consistently demonstrated greater dehydration or lower serum electrolyte concentrations in athletes who develop EAMC [3,4]. The clinical pattern is also difficult to explain by a generalized fluid or electrolyte disturbance alone. Cramps usually affect heavily recruited muscles rather than occurring diffusely, and they often appear when local muscular fatigue is greatest [1,6]. Experimental work has further shown that substantial hypohydration does not necessarily lower the threshold for electrically induced cramping [5].

This does not mean that fluid and electrolyte balance is irrelevant in every athlete. Large sweat losses and inadequate replacement remain important during prolonged exercise and may coexist with cramping. The available evidence simply does not support considering them the primary explanation for most episodes [1,6].

4.2. Altered Neuromuscular Control

The altered neuromuscular control hypothesis links EAMC more directly to muscular fatigue. Prolonged or unusually intense exercise may disturb the balance between excitatory input from muscle spindles and inhibitory input from Golgi tendon organs. This shift can increase alpha motor neuron excitability and promote sustained involuntary contraction of the fatigued muscle [6].

Several features of EAMC are compatible with this model. Cramps commonly involve muscles that have been repeatedly loaded, tend to occur later in prolonged exercise, and may develop when competition demands exceed the athlete's usual level of conditioning [1,6]. Muscle position may also be relevant, as cramping is easier to provoke when a muscle is shortened. Gentle stretching can rapidly relieve an active cramp, which is consistent with a neuromuscular mechanism, although its therapeutic effect does not by itself establish the cause of EAMC [1].

The model also has limitations. Much of the mechanistic evidence comes from experimentally induced cramps rather than spontaneous episodes during sport [1]. Fatigue is also common during competition, whereas EAMC affect only a proportion of athletes. Neuromuscular fatigue may therefore increase susceptibility without being sufficient to explain why a particular athlete develops an episode.

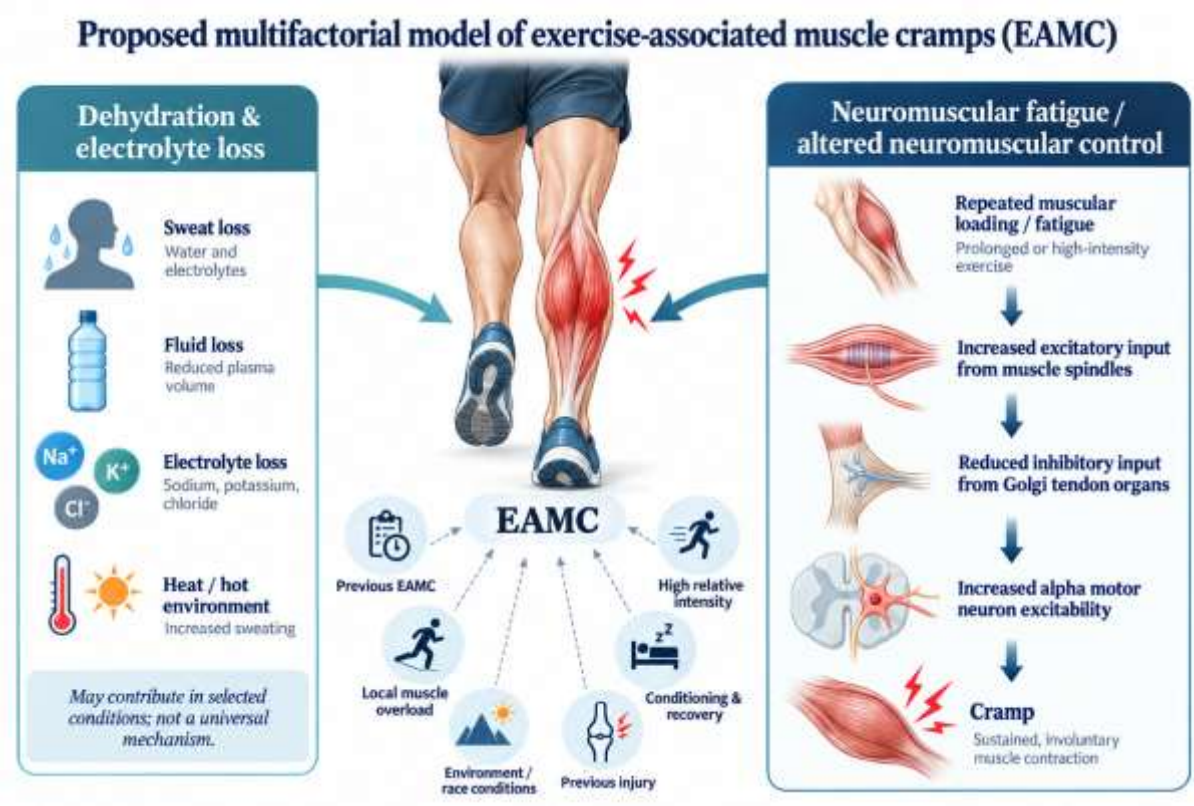
4.3. A Multifactorial Model

A broader model better reflects the variability observed in athletes. Neuromuscular fatigue may interact with individual susceptibility and with the demands placed on a particular muscle during exercise. Exercise intensity, local muscular overload, conditioning, previous injury, recovery, and other athlete-specific characteristics may influence the threshold at which cramping occurs [1].

This interpretation helps explain why no single physiological marker reliably identifies athletes who will develop EAMC. Similar exercise conditions can produce cramps in one athlete but not another, and the same athlete may cramp during one competition while remaining symptom-free during another. EAMC may therefore represent a common clinical endpoint reached through different combinations of predisposing and precipitating factors [1,6].

Altered neuromuscular control associated with fatigue currently provides the strongest mechanistic framework for many cases of EAMC, but it should not be regarded as a complete explanation. The marked variation between athletes is more consistent with several interacting influences than with a single universal cause [1].

Figure 1. Proposed multifactorial model of exercise-associated muscle cramps. Exercise-associated muscle cramps (EAMC) are likely to result from the interaction of several athlete- and exercise-specific factors rather than from a single mechanism. Dehydration and electrolyte loss may contribute under selected conditions but do not provide a universal explanation. Neuromuscular fatigue and altered neuromuscular control may increase motor neuron excitability through increased excitatory input from muscle spindles and reduced inhibitory input from Golgi tendon organs. Previous EAMC, high relative exercise intensity, local muscle overload, conditioning and recovery, previous injury, and environmental or race-related conditions may further modify individual susceptibility.



Abbreviation: EAMC, exercise-associated muscle cramps.

5. Experimental Models and Assessment of Cramp Susceptibility

Studying spontaneous EAMC under controlled conditions is difficult. Cramps are unpredictable, often occur late in prolonged exercise, and may depend on several factors that are difficult to reproduce simultaneously in the laboratory. Much of the mechanistic research has therefore relied on experimentally induced cramps. These models allow neuromuscular excitability to be studied under standardized conditions, but their relationship with spontaneous EAMC during sport is imperfect [1].

One of the most commonly used approaches is electrical stimulation of a motor nerve or muscle until a cramp is produced. Cramping is typically confirmed by a painful involuntary contraction accompanied by sustained electromyographic activity after stimulation has ended. The cramp threshold frequency (CTF) is the lowest stimulation frequency capable of inducing a cramp under a given protocol. A lower CTF is generally interpreted as greater cramp susceptibility [9].

CTF can be measured repeatedly and has been used to examine the effects of dehydration, fatigue, stretching, and other interventions [1,9]. Experimental studies using this method have challenged a simple dehydration-based explanation of EAMC. In one controlled study, substantial hypohydration accompanied by moderate electrolyte loss did not lower the threshold for electrically induced cramping [5]. This does not exclude a contribution from dehydration in individual athletes, but it shows that fluid loss alone is not sufficient to increase experimentally measured cramp susceptibility.

The relationship between fatigue and CTF is less straightforward. Neuromuscular models would predict greater cramp susceptibility after fatigue, yet laboratory findings have not always followed this pattern. In one study, localized fatiguing exercise increased rather than decreased CTF, indicating a lower propensity for electrically induced cramping immediately after the fatigue protocol [10]. Such results suggest that laboratory fatigue does not necessarily reproduce the prolonged and complex fatigue experienced during competition.

This illustrates the main limitation of experimental cramp models: their external validity. Electrical stimulation recruits motor units differently from voluntary contraction, and experiments are often performed in small muscles under highly controlled conditions. Spontaneous EAMC, by contrast, usually develop after sport-specific loading and in the presence of several physiological and environmental influences [1].

CTF should therefore be regarded as an experimental measure of cramp susceptibility rather than a diagnostic test for EAMC. There is currently no established threshold that can identify which athlete will develop a cramp during competition, and differences in the muscle tested, stimulation protocol, electrode placement, and participant characteristics make direct comparisons between studies difficult [1,9].

Experimental models remain useful for testing mechanistic hypotheses, but findings derived from them should be translated cautiously to spontaneous EAMC in athletes.

6. Risk Factors and Individual Susceptibility

Predicting which athletes will develop exercise-associated muscle cramps remains difficult. Numerous demographic, training-related, biochemical, and environmental variables have been investigated, but few have shown reproducible associations. The clearest finding is a previous history of EAMC [7].

A 2026 systematic review and meta-analysis including 11,274 participants found that athletes with previous EAMC had approximately 4.4-fold higher odds of experiencing another episode [7]. Prospective observations in Ironman triathletes point in the same direction [4], although the number of cramp events in individual studies has often been small. Previous cramping therefore appears to be a more informative marker of future risk than basic demographic or training characteristics.

Exercise intensity and fatigue may also contribute, but their role is harder to quantify. Prospective studies have associated EAMC with faster race pace, while one ultramarathon cohort also found a tendency toward greater pre-race muscle damage among athletes who subsequently cramped [4,11]. In contrast, pooled analyses have not demonstrated a consistent relationship between overall finishing time and EAMC [7]. The relevant factor may therefore be the physiological demand placed on a particular athlete rather than absolute performance itself.

Training status may be similarly context dependent. An athlete can be well trained overall but insufficiently prepared for a particular pace, terrain, duration, or pattern of muscular loading. Sudden increases in intensity or unfamiliar exercise demands could plausibly increase susceptibility through greater local fatigue, but these factors have not been established as consistent independent predictors [1,7].

Hydration and electrolyte status have received considerable attention because of their traditional role in explanations of EAMC. Available studies have not consistently shown greater body mass loss, lower serum sodium, or other evidence of greater systemic electrolyte depletion in athletes who cramp [3,4,7]. In the 2026 meta-analysis, body mass change was not associated with EAMC, while post-race potassium and magnesium concentrations were higher rather than lower in athletes who developed cramps [7].

A recent longitudinal study of Ironman triathletes found that failure to follow a planned hydration strategy was associated with approximately threefold higher odds of EAMC [12]. This finding should be interpreted cautiously because hydration behavior may also reflect pacing, gastrointestinal tolerance, race strategy, or increasing fatigue. The study also did not measure physiological hydration status, so the association does not establish that dehydration itself caused the cramps [12].

Age, body mass, height, and sex have not shown reliable associations with EAMC [7,12]. Previous musculoskeletal injury, family history, and biomechanical characteristics have also been proposed as possible contributors, but the available evidence is inconsistent [1,7]. Genetic susceptibility has been explored in smaller studies, including an association between a COL5A1 variant and a history of EAMC, but these findings remain preliminary [13].

Overall, a history of previous EAMC remains the most consistent predictor of recurrence. Beyond this, no single demographic, training, hydration, or biochemical characteristic reliably identifies athletes who will cramp [7].

Table 1. Summary of selected risk factors and current evidence for exercise-associated muscle cramps.

Potential factor	Evidence	Current interpretation
Previous EAMC	~4.4-fold higher odds of recurrence [7]	**Strongest identified predictor**
Higher relative exercise intensity / faster pace	Associations reported in prospective endurance studies [4,11]	Likely relevant in susceptible athletes

Muscle fatigue / local overload	Supported by clinical and mechanistic observations [1,6]	Plausible contributor, but difficult to quantify
Dehydration / body mass loss	No consistent association in prospective studies or meta-analysis [3,4,7]	Not a universal risk factor
Electrolyte abnormalities	Lower serum sodium, potassium, or magnesium have not consistently predicted EAMC [3,7]	Systemic electrolyte depletion alone is unlikely to explain most cases
Other individual factors	Previous injury and genetic susceptibility have been proposed, but evidence is limited [1,7,13]	Possible modifiers; insufficient for routine risk prediction

7. Clinical Assessment and Differential Diagnosis

The diagnosis of exercise-associated muscle cramps is primarily clinical. A typical episode consists of a sudden, painful, involuntary contraction of a skeletal muscle during or shortly after exercise. The affected muscle is usually visibly or palpably tense, and symptoms generally improve after reducing activity and gently stretching the muscle [1,14].

In an otherwise well athlete with an isolated, localized episode and a typical exercise-related pattern, extensive diagnostic testing is rarely necessary. Clinical assessment should focus on the timing and duration of symptoms, muscles involved, previous episodes, and whether the event differed from the athlete's usual training or competition. Recent changes in training load, exercise intensity, recovery, previous injury, environmental conditions, fluid and nutritional intake, and medication use may provide additional context [14].

A broader assessment is warranted when cramps are recurrent without a clear exercise-related pattern, unusually severe, widespread, prolonged, or occur at rest. Cramps associated with persistent weakness, sensory symptoms, fasciculations, exercise intolerance, or other neurological findings may indicate an underlying neuromuscular, metabolic, endocrine, or medication-related disorder rather than uncomplicated EAMC [14].

Systemic symptoms are particularly important. Confusion, altered consciousness, collapse, severe headache, vomiting, marked weakness, or abnormal body temperature are not typical features of localized EAMC. Exertional heat stroke should be considered when severe hyperthermia is accompanied by central nervous system dysfunction [15], while exercise-associated hyponatremia may present with headache, nausea, confusion, seizures, or altered consciousness after prolonged exercise [16]. These conditions require specific management and should not be treated as simple muscle cramping.

Exertional rhabdomyolysis is another important differential diagnosis when muscle pain is unusually severe or persists after exercise, particularly in the presence of swelling, weakness, or dark urine. In such cases, creatine kinase, renal function, urinalysis, and selected electrolyte measurements may be appropriate [17]. Laboratory investigations should otherwise be guided by the clinical presentation rather than performed routinely after an isolated EAMC episode that resolves promptly [14].

The term “heat cramps” should be used cautiously. EAMC can occur in both hot and cool environments and are not defined by elevated body temperature [1,14]. Automatically attributing exercise-related cramps to heat or electrolyte loss may therefore obscure other contributing factors.

In most athletes, a localized cramp occurring during or shortly after demanding exercise and resolving with rest or stretching can be managed as uncomplicated EAMC. Further investigation becomes more important when the pattern changes, recovery is prolonged, symptoms extend beyond the exercised muscles, or systemic features are present [14].

8. Acute Management of Exercise-Associated Muscle Cramps

The immediate management of uncomplicated EAMC is usually simple. When a cramp develops during exercise, the athlete should reduce or stop the activity and gently stretch the affected muscle. Static stretching remains the best-supported first-line intervention and is generally continued until the involuntary contraction and pain begin to subside [1]. Forceful stretching should be avoided in an already fatigued muscle.

Rest and gentle massage are often used alongside stretching, although evidence for massage as an independent treatment is limited [1]. Once symptoms resolve, return to activity should be

gradual. Immediate resumption at the previous intensity may promote recurrence when substantial local fatigue persists.

Fluid and electrolyte replacement should be considered separately from the immediate relief of the cramp. Rehydration may be appropriate after prolonged exercise, substantial sweating, or inadequate fluid intake, but oral fluids cannot rapidly correct a systemic deficit within the short period in which many EAMC episodes resolve [1]. Improvement after consuming an electrolyte-containing beverage should therefore not be taken as evidence that electrolyte depletion caused the episode.

Pickle juice is one of the best-known alternative interventions. In an experimental study, ingestion of a small volume of pickle juice shortened the duration of electrically induced muscle cramps despite producing no meaningful immediate change in plasma electrolyte concentrations [18]. A neurally mediated reflex triggered by sensory stimulation in the mouth or upper gastrointestinal tract has been proposed as a possible explanation [18]. These findings are physiologically interesting, but evidence from spontaneous EAMC during sport remains limited, and pickle juice should not be considered a standard first-line treatment [1].

Other commercially promoted anti-cramp products have similarly limited clinical support. Quinine can reduce muscle-cramp frequency in some settings, but its potential adverse effects and unfavorable risk-benefit profile preclude its routine use for EAMC [19]. Intravenous fluids are also unnecessary for uncomplicated EAMC unless another clinical indication is present [1].

For a typical localized episode, treatment therefore remains centered on reducing the provoking activity, gently stretching the affected muscle, and allowing sufficient recovery before return to exercise [1]. Persistent, widespread, or systemically accompanied cramping should instead prompt reassessment for another exercise-related or medical condition [14].

9. Prevention Strategies

Preventing exercise-associated muscle cramps remains challenging because no single intervention has shown consistent benefit across athletic populations [1]. A practical prevention strategy should therefore focus on modifiable circumstances surrounding previous episodes rather than on a universal anti-cramp protocol.

Sport-specific preparation is a reasonable starting point. Training should reflect the expected duration, intensity, terrain, and pattern of muscular loading of the target event. In athletes who repeatedly cramp in the same muscle groups, improving local strength and fatigue resistance may be useful, although controlled studies directly demonstrating a reduction in EAMC remain limited [1].

Pacing may also matter, particularly when competition intensity exceeds what the athlete has regularly tolerated in training. Reviewing when cramps occur in relation to pace, distance, terrain, and accumulated fatigue may help identify situations in which the muscular demands of an event exceed current conditioning [4,11].

Pre-exercise stretching has not been shown to reliably prevent EAMC. A recent critically appraised review found minimal evidence that pre-event stretching, flexibility, or flexibility training predicts which athletes develop EAMC during competition [20]. This should be distinguished from the role of gentle static stretching in acute treatment. Stretching may still serve mobility or other training goals, but it should not be promoted specifically as an established method of cramp prevention [1,20].

Hydration and electrolyte intake should follow the demands of the exercise rather than the assumption that recurrent cramps indicate a fluid or sodium deficit. Appropriate replacement remains important during prolonged exercise, particularly in hot conditions or in athletes with high sweat losses, but routinely increasing fluid or sodium intake has not consistently prevented EAMC [1,7].

The same caution applies to supplements marketed for cramp prevention. Magnesium is frequently promoted for muscle cramps, yet a Cochrane review found no randomized controlled trials specifically evaluating magnesium for exercise-associated muscle cramps [21]. Sodium preparations and sensory-based anti-cramp products have likewise not been shown to provide a consistently effective preventive strategy for EAMC [1]. Their use should not replace attention to training demands, pacing, and recovery.

For athletes with recurrent EAMC, the most useful approach is to look for repeatable patterns across episodes. Timing, muscle groups involved, recent training, recovery, race strategy, environmental conditions, and previous injury may reveal modifiable contributors [1,7]. Prevention is therefore likely to be most effective when tailored to the athlete and the specific exercise context rather than based on a single intervention.

10. Current Evidence Gaps and Future Directions

Despite decades of research, several aspects of EAMC remain poorly defined. Studies differ substantially in the populations examined, exercise protocols, definitions of cramping, and methods used to assess fatigue, hydration, and neuromuscular function [1,7]. Experimental studies add further uncertainty because measures such as cramp threshold frequency are useful for investigating neuromuscular excitability but do not directly reproduce spontaneous EAMC during competition [9,10]. These methodological differences make direct comparison difficult and may partly explain why proposed risk factors are not reproduced consistently.

Prevention is particularly uncertain. Many commonly recommended strategies have not been adequately tested in athletes with recurrent EAMC [1]. More informative studies would focus on athletes who repeatedly cramp under similar conditions and combine information on event-specific workload, neuromuscular function, previous injury, recovery, hydration behavior, and cramp history rather than relying mainly on isolated physiological measurements [7,12].

A central unanswered question is why some athletes repeatedly develop EAMC while others exposed to similar exercise demands do not. Previous EAMC remains the strongest identified predictor of recurrence [7], while possible contributions from genetic susceptibility and other athlete-specific characteristics remain incompletely defined [13]. Clarifying the basis of this individual susceptibility may ultimately be more useful than continuing to search for a single universal cause.

11. Conclusions

Exercise-associated muscle cramps remain a common but incompletely understood problem in athletes. Current evidence does not support dehydration or electrolyte loss as a universal explanation. Altered neuromuscular control associated with fatigue provides a stronger framework for many episodes, although the marked variation between athletes suggests that EAMC arise from several interacting factors rather than a single mechanism [1,6].

A previous history of cramping remains the most consistent predictor of recurrence [7]. In athletes with recurrent EAMC, assessment should therefore focus on the circumstances surrounding individual episodes, including exercise demands, local fatigue, conditioning, pacing, recovery, and previous injury. Acute management remains centered on reducing the provoking activity and gently stretching the affected muscle [1], while routine electrolyte

supplementation or pre-exercise stretching cannot be recommended as universally effective preventive strategies [1,20].

The substantial differences in susceptibility between athletes remain unexplained. Better prospective and intervention studies focused on recurrent EAMC are needed to identify clinically relevant risk patterns and determine which preventive strategies meaningfully reduce recurrence [7].

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Declaration of the use of generative AI and AI-assisted technologies in the writing process

During the preparation of this manuscript, the author used ChatGPT (OpenAI) to assist with language refinement, improvement of clarity and readability, and manuscript structuring and formatting. After using this tool, the author critically reviewed and edited the content as needed and takes full responsibility for the final content of the manuscript.

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