

A NARRATIVE REVIEW OF EXERCISE-ASSOCIATED MUSCLE CRAMPS: FACTORS THAT CONTRIBUTE TO NEUROMUSCULAR FATIGUE AND MANAGEMENT IMPLICATIONS

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ABSTRACT: Although exercise-associated muscle cramps (EAMC) are highly prevalent among athletic populations, the etiology and most effective management strategies are still unclear. The aims of this narrative review are 3-fold: (1) briefly summarize the evidence regarding EAMC etiology; (2) describe the risk factors and possible physiological mechanisms associated with neuromuscular fatigue and EAMC; and (3) report the current evidence regarding prevention of, and treatment for, EAMC. Based on the findings of several large prospective and experimental investigations, the available evidence indicates that EAMC is multifactorial in nature and stems from an imbalance between excitatory drive from muscle spindles and inhibitory drive from Golgi tendon organs to the alpha motor neurons rather than dehydration or electrolyte deficits. This imbalance is believed to stem from neuromuscular overload and fatigue. In concert with these findings, the most successful treatment for an acute bout of EAMC is stretching, whereas auspicious methods of prevention include efforts that delay exercise-induced fatigue.

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In this review we focus on exercise-associated muscle cramps (EAMC), which have been defined as painful, spasmodic, and involuntary contractions of skeletal muscle that occur during or immediately after exercise and have no underlying metabolic, neurological, or endocrine pathology.¹ Nocturnal cramps or cramps associated with metabolic abnormalities are not considered EAMC and are beyond the scope of this review. Exercise-associated muscle cramps have also been called heat cramps; however, this term has lost favor, as cramping has been documented during exercise in cooler conditions.^{2,3}

The clinical presentation of EAMC includes acute pain, stiffness, and bulging or knotting of the muscle. Exercise-associated muscle cramps often last from 1 to 3 minutes and generally occur in multi-joint muscle groups when contracting in a shortened position (e.g., quadriceps, hamstrings, triceps surae).^{4–6} The severity of EAMC can range from

mild discomfort, with limited effects on physical performance, to extreme pain and debilitation.⁷

EAMC are among the most common conditions that require medical intervention, either during or immediately upon completion of athletic events, representing up to two-thirds of complaints reported during endurance-related competitions.^{5,8} In a 12-year summary report of marathon medical issues, cramping accounted for 6.1% of medical encounters, with 1.2 cases of EAMC per 1,000 participants.⁹ Most recently, a prospective study of 26,354 ultramarathon runners revealed that 1 in every 526 race starters developed EAMC.¹

The 2 main theories behind EAMC, the dehydration and electrolyte imbalance theory and the altered neuromuscular control theory, have been reviewed elsewhere, and the strongest evidence supports a neuromuscular etiology.^{7,10,11} Briefly, the serum electrolyte and dehydration theory postulates that the extracellular fluid compartment becomes increasingly contracted due to sweating, leading to a loss of interstitial volume. In addition, excessive sweating can lead to concomitant sodium, calcium, magnesium, chloride, and potassium deficits.^{12–14} It follows that a mechanical deformation of nerve endings and an increase in the surrounding ionic and neurotransmitter concentrations leads to hyperexcitable motor nerve terminals and spontaneous discharge.¹⁵

Although some researchers have reported an association between EAMC and electrolyte deficits,^{16–19} those investigations have some methodological limitations. First, the studies were all observational, where causation may not be inferred due to potential confounding factors. Second, during the days of testing, no study participants had EAMC, despite having either serum or sweat electrolyte losses.^{16–19} Third, sample sizes were small in each of those investigations, limiting power and external validity. Finally, adjustments were not made for known EAMC correlates, such as participant fitness level, exercise intensity, fatigue, previous injury, and acclimatization, in many of those studies. In addition, several prospective cohort studies and case-control studies reported no

Abbreviations: ACSM, American College of Sports Medicine; BMI, body mass index; CTF, cramp threshold frequency; EAMC, exercise-associated muscle cramps; EIMC, electrically induced muscle cramps; GTO, Golgi tendon organ; KT, kinesio taping

Key words: dehydration; electrolyte deficit; muscle fatigue; muscular cramps; neuromuscular control

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differences in hydration status or plasma concentrations of electrolytes between cramp-prone and non-cramp-prone participants.^{1,3,8,20–25}

The plausibility of hydration status and electrolyte concentrations causing EAMC also seems questionable, as it is often relieved by stretching of the affected muscles,^{20,26} or by activation of the Golgi tendon organs (GTOs).²⁷ These treatments would certainly be incongruous for a condition mediated by electrolyte deficit and dehydration. Likewise, the systemic nature of electrolyte deficits and dehydration does not explain the fairly consistent presentation of cramping only within working muscles.

The “altered neuromuscular control theory,” popularized by Schweltnus, posits that EAMC result from altered reflex control mechanisms in response to neuromuscular fatigue.¹⁰ Specifically, muscle overload and fatigue engender an imbalance of the excitatory drive from muscle spindles and the inhibitory drive from GTOs. The result is an increase in excitatory drive to the alpha motor neuron, which ultimately produces a localized cramp. It has been observed that runners who develop EAMC almost exclusively report a subjective feeling of muscle fatigue before onset of EAMC.²⁸ In addition, EAMC has been shown to occur in athletes toward the end of games or races.^{3,25,28} The theory also explains the increased baseline electromyographic (EMG) activity recorded between bouts of cramping in athletes who are experiencing fatiguing exercise.²⁴ This hypothesis has experimental support, as skeletal muscle fatigue has been shown to reduce inhibitory input to alpha motor neurons from the GTOs and to increase excitatory input from the muscle spindles in animal models.^{27,29,30} It has also been shown that, when skeletal muscle contracts in a shortened position, there is depressed signaling from the GTOs, which explains why stretching is the best-known and most effective treatment for acute EAMC.^{7,27,31} Along these same lines, electrical stimulation of tendon afferents has been shown to successfully relieve muscle cramping.²⁷ Specifically, Khan and Burne²⁷ induced cramps in the gastrocnemius of subjects using maximum voluntary contraction while the muscle was in a shortened position. In all cramping subjects, reflex inhibition was observed after Achilles tendon electrical stimulation, with all subjects reporting relief of the cramp. This finding lends further support to the theory that abnormal spinal reflex activity is associated with muscle cramping.

In summary, support for the dehydration and electrolyte theory is based on low-level evidence in the form of case series, case-control data, and anecdotal observations. These observations have not been supported in recent prospective cohort

investigations, where athletes have experienced EAMC when fully hydrated and sufficiently supplemented with electrolytes. In addition, evidence from human and animal models has demonstrated that altered spinal reflex activity during fatigue is associated with skeletal muscle cramping. Finally, stretching has been shown to restore the balance between excitatory and inhibitory impulses to the alpha motor neuron and is the most effective remedy for acute EAMC, offering further support to the altered neuromuscular control theory.

To date, the phenomenon of neuromuscular fatigue is poorly understood. By summarizing recent EAMC investigations that explored possible risk factors and management strategies, it is our hope to further elucidate the mechanisms behind neuromuscular fatigue and the imbalance of inhibitory and excitatory drives that elicits EAMC. Accordingly, in this review we report the current evidence regarding neuromuscular fatigue risk factors and prevention and treatment methods.

ALTERED NEUROMUSCULAR CONTROL RISK FACTORS

History of EAMC. EAMC susceptibility varies widely among individuals; some routinely develop EAMC and others, despite being similarly matched for conditioning duration and intensity, demonstrate cramp resistance (Fig. 1).^{8,23} Along these lines, Miller *et al.*³² reported that cramp susceptibility is correlated with an individual cramp threshold frequency (CTF), defined as the minimum electrical stimulation required to evoke a muscle cramp.³² They reported significantly ($P < 0.001$) lower CTF in participants with a positive cramp history than in those with no history of cramping.

Several recent observational investigations have reported that athletes with a history of EAMC were more likely to cramp during or shortly after exercise than those who had no history of EAMC.^{8,20,25} Schweltnus *et al.*⁸ reported that triathletes who experienced EAMC while participating in an Ironman event, or in the 6 hours after the race, had a significantly higher reported history of EAMC compared with the triathletes who did not cramp ($P < 0.001$). More specifically, a history of cramping in the previous 10 events, was strongly associated with EAMC. A prospective, longitudinal investigation of 103 male rugby players described significant differences in the history of cramps among players who experienced EAMC throughout the season compared with those who did not ($P = 0.004$).²⁵

Family History and Genetics. The findings regarding family history of cramping and EAMC are somewhat equivocal. A recent, prospective study and a case-control study reported that a positive family history of EAMC was not associated with the

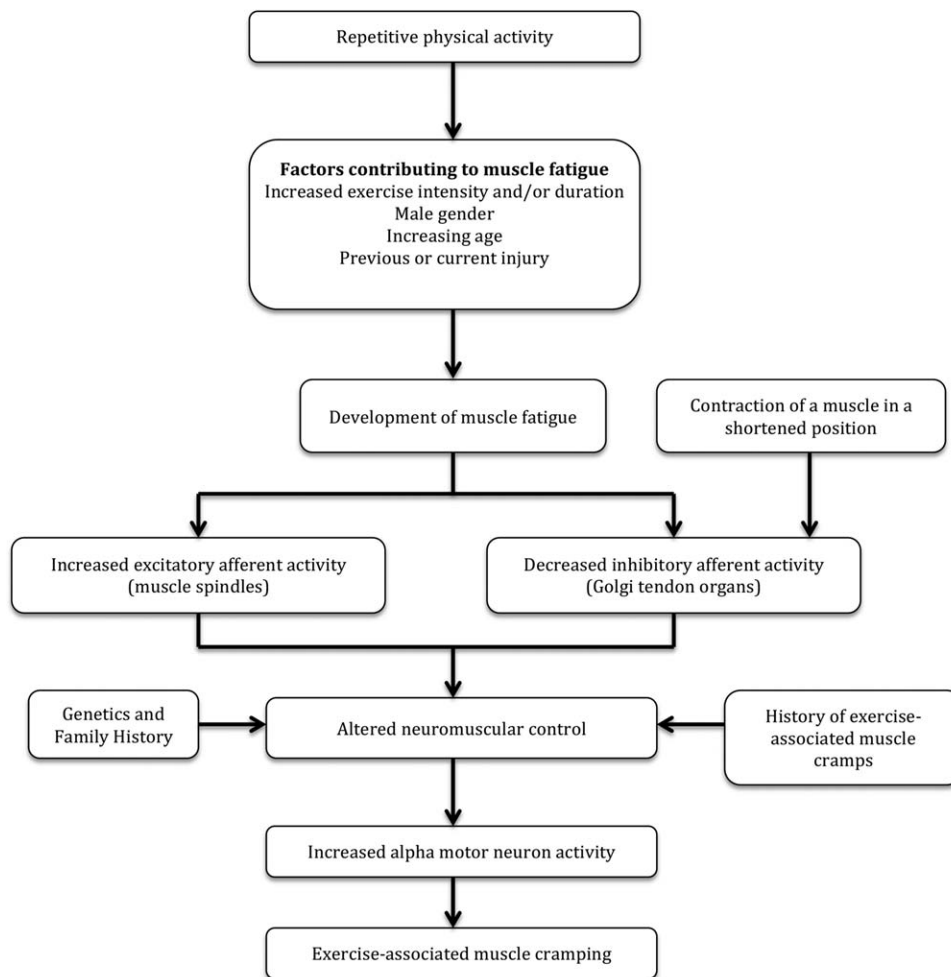


FIGURE 1. Factors associated with exercise-associated muscle cramps.

risk of developing EAMC during endurance events.^{8,20} Conversely, a cross-sectional study of 1,300 marathoners reported an association between family history of EAMC and a history of EAMC.²⁸ This study was subsequently supported in a case-control investigation in which cramp-prone triathletes were shown to be significantly more likely to have a family history of EAMC when compared with non-cramp-prone triathletes ($P < 0.001$).²³ Interestingly, those researchers also reported that there was a significant family history of EAMC on the male side of the family, suggesting a possible genetic component of EAMC.

Despite equivocal findings regarding an association between family history and EAMC, a recent case-control investigation reported that a structural component of the extracellular matrix of soft tissue is associated with EAMC.³³ Specifically, the researchers discovered that the *COL5A1* rs12722 CC genotype, a polymorphism associated with structural and mechanical properties of collagen fibrils and musculoskeletal soft tissue injury, was

significantly associated with a history of EAMC ($P = 0.034$). Thus, the extracellular matrix of muscle tissue, which has also been implicated in delayed-onset muscle soreness, may play a role in facilitating EAMC.

Gender. Gender-related differences have been shown to be associated with EAMC.^{1,23} The SAFER II study, an investigation of medical injuries over the course of an ultramarathon race, reported that male runners had a higher incidence of EAMC than female runners ($P = 0.0482$).¹ Shang *et al.*²³ discovered that there were significantly more men ($P = 0.011$) than women among triathletes who were grouped based a self-reported history of EAMC. A plausible explanation for this finding is that women have been found to be less fatigable than men when exercising at similar relative intensities.³⁴ Although gender-related mechanisms of fatigue are not fully understood, it has been proposed that the skeletal muscles of men possess a greater proportional area of fast-twitch (type II)

fibers in muscles of locomotion than women.^{35,36} As type II fibers are known to be more fatigable compared with type I fibers due to lower capillary and mitochondrial densities, this may explain the relationship between gender and EAMC incidence. In addition, during moderate- to high-intensity endurance exercise, women have been shown to oxidize more fat and less carbohydrate than men at the same relative activity intensity.^{36,37} The capacity to utilize lipid for fuel during endurance events may also explain the gender differences in fatigability and, ultimately, EAMC in the late stages of endurance events.

Age. There is limited evidence to indicate a relationship between older age and EAMC in endurance and team sport competitions.^{1,25} The SAFER II investigation showed that runners >50 years of age also had a greater incidence of EAMC than those in the 41–50-year category ($P = 0.0007$).¹ A prospective study of rugby players showed that athletes in the senior division of competition ($n = 59$, mean age 21.1 ± 4.1 years) had a significantly greater risk of EAMC ($P < 0.001$) than those in the younger division ($n = 44$, 15.8 ± 0.9 years). Conversely, several large, prospective and case-control investigations have reported no association between EAMC and age.^{8,20,23,24}

Body Size. Several studies have investigated increased body mass index (BMI) and body weight as independent risk factors for EAMC. A recent prospective cohort investigation of triathletes showed no significant differences in BMI or body weight between athletes who cramped during an event or up to 6 hours after the event and those who did not.⁸ In support of these findings, a cohort study of ultradistance runners showed no significant between-group differences in body weight between runners who cramped and those who did not.²⁰ In a case-control study involving Ironman triathletes with a history of cramps ($n = 216$) and triathletes with no history of cramps ($n = 217$), no significant between-group differences were found for BMI ($P = 0.493$).²³ Although elevated body mass and BMI are known to have performance implications in endurance events,³⁸ based on current evidence, they do not appear to be associated with EAMC.

The evidence regarding any association between height and EAMC is limited and equivocal. Schwellnus *et al.*⁸ found no significant differences in height between triathletes who cramped during an Ironman event and those who did not ($P = 0.882$). In contrast, Shang *et al.*²³ reported that triathletes with a self-reported history of EAMC were taller when compared with those having no EAMC history ($P = 0.003$). Interestingly, in

that case-control study, the difference in height remained significant, even after adjustment for gender ($P = 0.007$). The authors speculated that the differences in the biomechanics of the lower leg in taller athletes may alter running economy and, ultimately, contribute to premature fatigue and EAMC.

Exercise Intensity and Duration. Exercise intensity and duration have been the most extensively investigated risk factors related to fatigue and EAMC. There has been a consistent association between EAMC and prolonged activity performed at vigorous intensities.^{3,8,23,25,28} Schwabe *et al.*¹ discovered that those in the sub-6-min/km category had a higher incidence of EAMC when compared with those in the 6–7-min/km category ($P = 0.0066$), but no difference in EAMC incidence when compared with runners in the >7-min/km category ($P = 0.0921$).¹ Shang *et al.*²³ found that triathletes categorized as cramp-prone had faster overall finishing times ($P < 0.043$) compared with those categorized as non-crampers. It should be noted that the 2 groups were similarly matched for previous performance, training duration, intensity, and frequency before the event. Those findings echo results from a recent prospective study of 210 triathletes.⁸ Athletes were grouped as crampers or non-crampers, based on the incidence of EAMC during the event. Although the triathletes were matched for pre-event training times and training volume, it was found that faster overall finishing time was a significant independent risk factor for EAMC ($P = 0.01$).

Playing in an advanced-level rugby league has also been shown to be a predictor of EAMC.²⁵ This finding may be attributed to the fact that the length of games in the advanced league are longer than in lower competition levels and that EAMC almost exclusively developed in the final 10 minutes of the games. Moreover, the season length in the junior competition spans 9 weeks, whereas the season length for senior players spans 26 weeks. The study reported that, as the number of games increased, there was a slightly higher ($P = 0.051$) EAMC incidence, suggesting there may be a positive relationship between EAMC and the number of games played during a season.

Previous or Current Injury. Shang *et al.*²³ found that cramp-prone athletes were more likely to have a history of tendon and/or ligament injury ($P < 0.022$) when compared with non-cramping athletes. They speculated that soft tissue injury could trigger an increase in reflex alpha motor activity, resulting in EAMC. Another plausible explanation is that previously injured areas may be vulnerable

to development of premature fatigue due to weakness of the localized muscles.

A recent prospective investigation showed an association between lower back pain, which was severe enough to miss playing time, and EAMC in the calf muscles of rugby players.²⁵ This was the first study to reveal such a relationship; however, the researchers reasoned that low back pain results in altered neural transmission in the nerves supplying the lower limb, resulting in calf cramps. Although several biopsychosocial factors are known to cause back pain,^{39,40} and this study did not identify the source of the low back pain, further investigation is warranted to reveal the exact nature of the relationship between low back pain and EAMC.

In summary, several observational studies have addressed potential risk factors for EAMC. The strongest evidence indicates that a previous history of EAMC, male gender, and prolonged and relatively vigorous exercise are the best predictors of EAMC in endurance and team sport events. These results, however, should be interpreted with caution, as they are based on observational studies where causation cannot be inferred. In addition, although neuromuscular fatigue may be central to the etiology of EAMC, it is important to note that muscle fatigue is a vague and poorly understood condition. Moreover, it is still unclear why certain athletes are more susceptible to fatigue and EAMC. Toward this end, well-designed experimental studies are needed to elucidate the precise mechanisms that may trigger fatigue and subsequently lead to decreases in GTO activity and increases in muscle spindle activity.

Notwithstanding this lack of detailed study, the findings of this exploratory review of EAMC correlates should provide practitioners and researchers with some preliminary data for which to screen athletes and guide further EAMC research.

TREATMENT AND PREVENTION

In practice, management strategies for EAMC are diverse, likely owing to the uncertainty of EAMC etiology. In this section we summarize the investigations that have reported on the efficacy of treatment and prevention strategies for EAMC.

Electrical Cramp Induction. Over a 6-week time frame, Behringer *et al.*⁴¹ applied electrical muscle stimulation twice weekly to both calf muscles of intervention participants ($n = 10$). The stimulation was applied to 1 calf in a shortened position, which elicited cramping, while the contralateral calf was stimulated with the ankle in a neutral position, which hindered muscle cramping. The researchers measured CTF for both legs of the intervention participants, as well as non-treated

control participants ($n = 5$) at 3 and 6 weeks. The CTF of the calf muscles receiving stimulation in a shortened position was increased significantly at 3 ($P < 0.001$) and 6 ($P < 0.001$) weeks, whereas no significant changes were noted at either assessment point in the contralateral legs of the intervention participants or in the calf muscles of participants in the control group. In a follow-up study, Behringer *et al.*⁴² found that 2 bouts of electrically induced muscle cramps (EIMC), induced 1-week apart in the gastrocnemius of an intervention group ($n = 8$), raised the CTF significantly for 24 hours in each of the 2 bouts ($P < 0.05$). The CTF remained elevated for 1 week in the EIMC participants, although this finding was not considered statistically significant after 24 hours. Control group participants ($n = 5$) received no EIMC and experienced no change in their CTF at any assessment point. The authors of these studies proposed that application of EIMC may rebalance the excitatory and inhibitory inputs to the alpha motor neurons by eliciting adaptations in the central nervous system. Although inducing painful skeletal muscle cramps may not seem like an acceptable preventive measure for the majority of individuals, athletes who routinely experience EAMC during vigorous training and competition may view electrical induction as a worthwhile performance-enhancing solution to EAMC.

Kinesio Taping and Compression Garments. The use of lower leg compression stockings and kinesio taping (KT) have increased in popularity in recent years. Although little empirical evidence supports the use of these training aids for prevention of EAMC, compression and KT are thought to attenuate soft-tissue vibrations upon ground impact, thereby improving muscle activation, which may offset fatigue-induced changes in running mechanics.^{43,44} A small, prospective cohort study explored the effects of KT on calf pain and incidence of EAMC.⁴⁵ The researchers applied the KT to the triceps surae muscles of 6 triathletes before racing. Athletes were asked after the event about EAMC incidence, and none of them had cramping in the triceps surae group. Interestingly, 2 athletes had quadriceps EAMC during the events. The researchers proposed that application of tape creates convolutions in the skin of the athlete, which engenders an increase in local blood flow and reduces pressure on mechanoreceptors, ultimately decreasing the incidence of EAMC.

Massage Therapy. It has been proposed that the mechanical pressure during massage alters neural excitability, and these neural changes may reduce the potential for cramping.^{46,47} Sefton *et al.*⁴⁸ discovered a reduction in the Hoffman (H)-reflex,

which was used to measure the excitability of the motor neuron pool in study participants who received a 1-hour full-body massage. Behm *et al.*⁴⁶ found that massage decreased spinal reflex excitability, with significant reductions in subjects who received 30 seconds of tapotement (a percussive massage stroke). Despite these neural changes induced by massage, experimental evidence is still needed to determine whether it actually facilitates a balance between excitatory impulses from muscle spindles and inhibitory impulses from GTOs. From a performance perspective, given the potential to reduce spinal level reflex excitability, it is unclear whether massage performed immediately before an event would result in performance decrements. As such, further investigation is warranted to determine whether treatment variables, such as the relative timing of massage, depths of pressure, speed of stroke, and type of massage stroke, influence EAMC without negatively impacting performance.

Electrolyte Supplementation and Hydration. Despite the lack of empirical evidence in support of the dehydration–electrolyte theory, salt tablets and magnesium supplementation are commonly used among athletes as a means to treat and prevent EAMC.⁴⁹ Given the lack of substantive data linking electrolyte deficits and dehydration with EAMC, it is unlikely that salt tablets or magnesium supplementation would be effective. It is, however, sensible for practitioners to encourage athletes and exercise participants to follow hydration and electrolyte supplementation guidelines to prevent heat-related illness. Accordingly, health professionals should recommend fluid replacement during and after physical activity for athletes and exercise participants. The American College of Sports Medicine (ACSM) position on fluid replacement is that athletes consume a volume of fluid that prevents more than a 2% body weight loss from perspiration.⁵⁰ The ACSM also acknowledges the interpersonal variability in sweating rates and sweat electrolyte concentrations, so that fluid replacement may best be determined by measuring body weight before and after exercise.

Corrective Exercise. To date, the performance of corrective exercises engineered to improve biomechanics, muscular imbalances, and postural issues has very little scientific basis in treatment and prevention of EAMC. There is some evidence showing that EAMC is correlated with tendon injuries.²³ If this is the case, incorporation of eccentric exercise, a treatment used for tendinopathies,^{51,52} may be a feasible remedial choice for cramp-prone athletes. In a similar fashion, plyometric activity may have a place in prevention of EAMC. This training method is characterized by a rapid eccentric mus-

cle action immediately followed by a rapid shortening of the same muscle, and may improve neuromuscular function. Accordingly, future study within the context of EAMC is needed to determine the optimal frequency, intensity, and duration of these training practices. Although neuromuscular re-education and attempts to normalize muscular imbalances would seem to be reasonable approaches to preventing cramps, there is a paucity of data to support these methods. There is very limited evidence in this area, although 1 case study investigated the effects of strengthening and neuromuscular re-education of the gluteus maximus muscle in a triathlete prone to EAMC of the hamstrings.⁵³ Given the agonistic relationship of the hamstrings and the gluteus maximus muscles, the researchers reasoned that weakness of the gluteus maximus could increase the relative contribution of the hamstring muscles during running and lead to overuse, premature fatigue, and ultimately EAMC. After several weeks of progressive gluteus maximus strengthening and neuromuscular training, the subject completed 3 triathlons without hamstring cramping.

Stretching. Although Schweltnus *et al.*⁸ found no association between history of stretching and development of EAMC ($P = 0.43$), it appears that stretching is the most effective treatment in relieving acute fatigue-induced muscular cramping.^{6,20,26} The literature has consistently shown that shortened muscles that span 2 joints are more prone to cramping.^{4–6} In view of this, stretching seems to be a plausible treatment. Although the precise mechanism is unclear, it has been suggested that passive stretching may increase tension in the GTO, resulting in increased afferent reflex inhibition to the alpha motor neuron.^{27,30,54}

Quinine. The use of 200–300 mg/day of quinine has been shown to reduce the incidence of nocturnal and idiopathic muscular cramps.^{55,56} Quinine has been associated with thrombocytopenia⁵⁷; consequently, quinine supplementation for muscle cramping is no longer allowed in the United States. Furthermore, there are no published data to suggest that quinine is useful for EAMC.

Pickle Juice. Consumption of small amounts of pickle juice is another common treatment for EAMC.^{49,58} Pickle juice contains high concentrations of salt along with acetic acid, which is thought to trigger a reflex that increases inhibitory neurotransmitter activity in cramping muscles.^{58,59} One case report indicated that drinking 30–60 ml of pickle juice relieved EAMC within 30–35 seconds after consumption by restoring electrolyte balance.⁵⁸ Miller *et al.*⁵⁹ compared the effects of

consumption of 1 ml/kg of body weight of pickle juice to a similar volume of de-ionized water immediately after cramp induction in the flexor hallucis brevis muscles of 12 hypohydrated (3%) men. The researchers reported that cramp duration was 49.1 ± 14.6 seconds shorter after pickle juice ingestion compared with the water condition ($P < 0.05$). In addition, when comparing the 2 conditions, no significant differences in plasma composition were found for up to 5 minutes after consumption. The authors reported the amount of ingested pickle juice had a negligible effect on extracellular fluid electrolyte concentrations and hypothesized that the decrease in cramp duration was attributed to an inhibition of the oropharyngeal reflex, which reduces alpha motor neuron activity of cramping muscles throughout the body. In support of these data, Miller *et al.*⁶⁰ found no significant changes in plasma electrolyte concentrations within 1 minute of consumption of 1 ml/kg body mass of pickle juice among 9 euhydrated men. No changes in plasma sodium, magnesium, calcium concentrations, plasma osmolality, or plasma volume 60 minutes after consumption were reported ($P \geq 0.05$). The authors ultimately concluded that, if EAMC are triggered by electrolyte deficits due to sweating, small volumes of pickle juice are unlikely to restore any deficit induced by exercise.

Hyperventilation Strategies. A recent case series consisting of 3 participants assessed hyperventilation as a treatment for EAMC.⁶¹ The researchers hypothesized that hypoventilation with resulting respiratory acidosis may be a contributing factor to muscular cramping. One participant was instructed to hyperventilate (20–30 deep breaths/min) while experiencing a cramp. The investigators reported complete resolution of cramps within 1 minute of the breathing technique on 5 separate occasions. Another participant in the series performed the hyperventilation technique after developing EAMC two-thirds of the way through a 100-mile mountain bike ride. The subject reported complete resolution of symptoms without recurrence.

In summary, we found several promising strategies for EAMC management, including neuromuscular re-education of weak muscles, hyperventilation, and KT for management of EAMC. It is worth noting that the studies describing these approaches were all observational studies. Well-controlled, high-quality trials are needed to fully determine treatment efficacy. Although massage therapy is certainly a plausible EAMC management strategy, no conclusive evidence exists to support its effectiveness for EAMC. In well-controlled laboratory studies, consumption of small amounts of pickle juice has been shown to reduce the dura-

tion of EAMC. Electrical cramp induction has been shown to increase CTF in 2 laboratory-based studies. Although this strategy may prove to be a viable treatment option for athletes who experience repeated EAMC during competition, it would likely be viewed as impractical for the large majority of those with EAMC. Finally, stretching appears to be the most effective method for alleviating acute bouts of EAMC.

LIMITATIONS AND FUTURE DIRECTIONS

There are some limitations to our review. First, the review may be subject to selection bias, as no systematic method was used to select relevant articles. Second, it is limited by the information provided in the included primary studies. With respect to treatment and prevention, many studies had small sample sizes or were of a case study or case series design, and were not powered for multiple statistical tests, thus limiting the ability to detect clinically meaningful improvements. In addition, among studies that investigated treatment or prevention methods, few used multiple measurement points to evaluate the success of these methods. For the effects of treatments to be fully explored, regular assessment across a range of times and among varying EAMC populations is needed.

With regard to the mechanisms behind EAMC, many studies relied on self-report questionnaires to determine EAMC status, meaning that recall bias is an inherent limitation. The available evidence supports the altered neuromuscular control theory, in which fatigue is a central component. However, it is important to recognize that fatigue is a multidimensional construct that remains relatively understudied in EAMC research. In addition, there is a lack of understanding of the origins of fatigue in different physical tasks of differing durations and intensities. Although current EAMC studies have shed some light on the physical components of fatigue, such as relative exercise intensity and duration and gender-based differences in fatigability, it is unknown how other independent contributors of fatigue, such as sleep quality and anxiety, are involved in the genesis of EAMC. As such, there is a need for more high-quality trials investigating skeletal muscle fatigability and the mechanisms that may promote effective approaches to delay the onset of muscle fatigue. Interestingly, the use of various periodization models has gone unexplored relative to preventing EAMC. Periodization is a concept that involves systematic variation in program design, largely to promote long-term training and performance improvements, and to prevent overtraining. As fatigue appears to be the most salient factor of

EAMC, it is not unreasonable to assume that periodically varying exercise volume and training intensity may offset EAMC. Along these same lines, it would be worthwhile to investigate whether any correlation exists between overtraining syndrome and the incidence of EAMC.

Despite strong support for the altered neuromuscular control theory, the available evidence does not exclude the possibility that electrolyte disturbances and dehydration play a role in the etiology of EAMC among certain individuals. It may be the case that certain people are more sensitive to hydration and electrolyte disturbances. Further investigation is warranted to determine the relevance of these interpersonal differences.

CONCLUSIONS

The development of EAMC is more common in the later stages of athletic events and is more prevalent among athletes who exercise at relatively vigorous intensities. In addition, several large prospective cohort investigations have shown no association between hydration status, electrolyte concentrations, and EAMC. Based on these considerations, the altered neuromuscular control theory is the most cogent descriptive model that explains the origins of EAMC. Although the available evidence supports a neuromuscular foundation that stems from fatigue, further research is needed to elucidate the specific risk factors that contribute to neuromuscular fatigue and underlie EAMC. Owing to the limited understanding of fatigability and the involved mechanisms, the most effective prevention methods are unknown. Strong evidence indicates that stretching is the most effective method for treating acute muscle cramps.

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