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## REVIEW

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# Exercise-Associated Muscle Cramp-Doubts About the Cause

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## ABSTRACT

**Introduction:** Exercise-associated muscle cramp (EAMC) is one of the most common conditions that occur during or immediately after the exercise, with questionable etiology. **Aim:** Aim of article was to present doubts about the cause of EAMC, whether it is primarily a neurological condition or it is water and salt imbalance. **Results:** Strongest evidence supports the neuromuscular aetiology with the focus on the muscle fatigue. Muscle overload and fatigue affects the balance between the excitatory drive from muscle spindles and the inhibitory drive from the Golgi tendon organs (GTO). This results in a localized muscle cramp. Since the dehydration and electrolyte depletion are systemic abnormalities, it is not clear how these changes would result in local symptoms such as cramping of the working muscle groups. **Conclusion:** “Triad” of causes might be behind the etiology of EAMC, although the “altered neuromuscular control” theory with the “dehydration” theory is the most cogent descriptive model that explains the origin of EAMC. Treatment and prevention strategies for EAMC include: electrical cramp induction, kinesio taping and compression garments, massage therapy, electrolyte supplementation and hydration, corrective exercise, stretching, quinine, pickle juice, hyperventilation strategies.

**Keywords:** EAMC, EAMC cause, EAMC prevention.

## 1. INTRODUCTION

Exercise-associated muscle cramp (EAMC) is one of the most common conditions that occur during or immediately after the exercise (sport event) (1, 2). To date, there is some controversy about the etiology of this condition (1, 2, 3). There are two theories about the origin of EAMC. The older one is the “dehydration” and “electrolyte depletion” theory (water-salt balance), and the more recent one is the “altered neuromuscular control” theory (neurological origin) (1, 3). Prevention of EAMC is questionable, primarily due

to unclear etiology, and is mostly based on corrective exercise, massage therapy, stretching and nutritional supplements.

## 2. AIM

Aim of article was to present doubts about the origin of EAMC, whether it is primarily a neurological condition or it is water and salt imbalance and to briefly present the possibilities of EAMC prevention.

## 3. MATERIAL AND METHODS

Article has descriptive character and present narrative review of literature.

## 4. RESULTS

### 4.1. Exercise-associated muscle cramp (EAMC)–origin and prevention

Strongest evidence supports the neuromuscular aetiology with the focus on the muscle fatigue (1, 4, 5). One of the first studies showing that fatiguing muscle can develop cramping in normal healthy subjects during the exercise was reported in 1957 (6). It was also reported that those cramps are electrically active (using electromyography) and could be treated by passive stretching (6). These authors concluded that the muscle cramping is probably the motor activity that has origin in the central nervous system. It has been reported, in the laboratory-based studies (7), that muscle cramping can be induced by voluntary sustained muscle contractions, especially in a shortened position (what has recently been confirmed also by other investigators) (8, 9, 10) and electrical stimulation of the nerves supplying the motor input to muscles (10, 11). As a novel hypothesis in 1997, Schweltnus pointed out the “altered neuromuscular control” theory (12). He clearly stated that muscle fatigue might be the predecessor to the development of EAMC. Current concept states repetitive muscle exercise and overload result in the local muscle fatigue, which increases the excitatory afferent activity from muscle spindles and

decreases the inhibitory afferent activity from Golgi tendon organs (GTO) (1). The evidences come from studies in human and animal models (13, 14). Furthermore, what supports the neuromuscular aetiology is the effective immediate treatment of cramping. It is done by increasing inhibitory input to the muscle (decreasing the alpha motor neuron activity), either by stimulating the GTO afferents through stretching or by electrical stimulation of the tendon (9, 14).

In addition to aforementioned, several prospective cohort studies and case-control studies reported that serum electrolyte concentrations and hydration status are not associated with EAMC, in cramp-prone and non-cramp-prone participants (15-23). In 1986, Maughan observed 82 male marathon runners before and after a 42.2 km race, and found out that the serum electrolyte concentrations, including sodium and potassium, were not different between those suffering from cramp and those not so affected, either before or after the race (16). Moreover, a cohort of 72 runners participating in an ultra-distance road race brought us to the conclusion that serum electrolyte concentrations and hydration status are not associated with EAMC (17, 18). However, there was a significant decrease in sodium concentration in the immediate post-race values in the cramp group. Additionally, acute EAMC in iron man triathletes is not associated with a greater percent body mass loss or clinically significant differences in serum electrolyte concentrations (23). A prospective cohort study in 210 Iron man triathletes published in 2011 showed that the dehydration and altered serum electrolyte concentrations are not causes for EAMC (17). EAMC occurs in the localized muscle groups, and in the “electrolyte depletion” and “dehydration” theory there is no adequate physiological explanation of how a systematic abnormality results in localized symptoms (24, 25). Furthermore, the acute bout of EAMC is released by rest, passive stretching and sodium chloride (26).

However, there evidences in past that support the “dehydration” and “electrolyte depletion” theory. In 1904, muscle spasms (cramps) were reported for the first time in two men at the Episcopal Hospital and were presented as a new disorder due to exposure to intense heat (27). Edsall proposed that, as the pathological process, acute degeneration occurs within the muscle. In 1923 Haldane stated that he believed the cause of miners’ cramp is water poisoning (28, 29). Haldane further says, “When a man is working his blood is shunted away from the kidneys and excretion of urine stops. If the kidneys were working normally they would excrete the excess of water a save the man from cramps.” These beliefs were almost a century ago, and in that time ground breaking. Today we still have doubts about this topic.

After the preliminary note in 1904, several papers were published regarding the topic (27) and they suggested that the water-salt imbalance (loss of salt and water from the body) might be the cause for muscle cramping. Those papers were case reports or case series, mainly related to the work/physical activity done in hot and humid environmental conditions, and that actually led to the terminology “heat cramps” or “exertional heat cramps” (26). This terminology is today very questionable (1). EAMC does not only occur in hot conditions, but also in moderate to cool temperatures, and also in extreme cold (26). More recent paper from 2000 shows the linkage of heat exhaustion in a deep underground metalliferous mine

with dehydration, that leads to muscle cramping (30). In a 1 year prospective case series study, 106 cases were studied. They showed that heat cramps are associated with dehydration but not hyponatraemia. Additional fluid intake is needed to restore the balance. The mechanism for EAMC is increased sweat sodium depletion or “salty sweating”, resulting in sodium depletion, which then causes EAMC (24, 25). Pathophysiological process for this hypothesis is still not clear.

Prevention of EAMC primarily is use of dietary supplements and kinesio taping, massage therapy, corrective exercises that will lead to the improvement of the function of a particular group of muscles or the biomechanics of the organism itself, stretching exercises—most effective method; post-isometric relaxation techniques, plyometric or eccentric muscle strengthening in training programs (2, 4, 31). Supplementation with electrolytes (to achieve adequate electrolyte levels), hydration of the organism, quinine, mustard, pickle juice are listed as the nutritional form of EAMC prevention (2).

## 5. CONCLUSION

Muscle overload and fatigue affects the balance between the excitatory drive from muscle spindles and the inhibitory drive from the GTO. This results in a localized muscle cramp. Since the dehydration and electrolyte depletion are systemic abnormalities, it is not clear how these changes would result in local symptoms such as cramping of the working muscle groups “Triad” of causes might be behind the aetiology of EAMC, although the “altered neuromuscular control” theory with the “dehydration” theory is the most cogent descriptive model that explains the origin of EAMC. Corrective exercises, stretching with dietary supplements and massage therapy may be a form of prevention of the occurrence of EAMC.

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