

Scientific review

## **DELAYED ONSET OF MUSCULAR SORENESS EXPERIENCED BY ATHLETES WITH ENERGY CRISIS AND A TREATMENT OPTION**

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**Abstract:** The muscle soreness experienced by athletes is associated with a lower range of motion and reduced strength and therefore, it has a negative impact on performance. Athletes often feel an indistinct aching muscular pain and the affected muscle feels stiff and tender. A contraction occurs when the actin filaments and the myosin proteins are attached to one another and come together; thereby, there is relaxation when they are uncoupled and pull apart (Cheung, Hume, 2003). For the process of uncoupling, sarcomere needs a huge amount of energy and adenosine triphosphate (ATP). In the state of an energy crisis, actin and myosin filaments become stuck in an interlocked state. This contracture knot may squeeze the capillaries and cut off the blood. Failed circulation or ischemia impairs the mitochondria which fail to produce ATP. The absence of ATP leaves the sarcomere in a shortened and contracted state, with a huge amount of proinflammatory pain-causing substance (Dommerholt, 2011). The result of that is the appearance of muscle soreness 24 to 48 hours after exercise, referred to as a delayed onset of muscle soreness (DOMS). A lack of oxygen makes oxidative stress, referring to the creation of an increased volume of reactive oxygen species (ROS). Current studies have shown that athletes may be under high oxidative stress, suggesting that is important to consume adequate amounts of antioxidants (Benardot, 2010).

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Understanding the impact of antioxidants on muscle soreness is unconcluded and will be discussed further.

**Keywords:** *muscle soreness, energy crisis, oxidative stress by exercise, free radicals, supplement therapy*

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## INTRODUCTION

The practical experience of all those engaged in intense sports show that there is often muscle soreness a shorter or longer time after training, with a varying intensity. These conditions were monitored with a decreased range of the motion that these muscles provide, as well as muscle strength, which ultimately leads to a reduction in the capacity for sports achievements. After performing training, athletes feel an undefined muscle pain with a sense of muscle tension and pain which is manifested mostly when the muscle is in action. This pain reaches its peak 24 to 48 hours after an intense workout using eccentric loads on the muscles. This state of late-onset pain is defined as a separate entity, a late appearance of muscle pain or DOMS (Cheung, Hume, 2003). DOMS typically lasts three to four days. It is practically joined with microtears of certain muscle fibers, which overall leads to abnormal muscle extensibility and the incapacity to function at full capacity. DOMS occurs in a situation of energy deficit, i.e. an insufficient creating of high-energy ATP, when the muscle overflows with acidic decay products of the metabolism, with pro inflammatory cytokines and cytokines which perform an irritation of the nerve endings and create a sensation - the sensation of pain (Dommerholt, 2011).

This pain must be separated from the occurrence of trigger point pain that is formed in the muscle fibers at non-physiological and draining functions, which leaves the consequent sensation of pain only in certain places which by palpation can be defined as painful nodules. These are places connected with contracted sarcomere that cannot relax, and as such are excluded from the normal contractile function of the mass of muscle fibers.

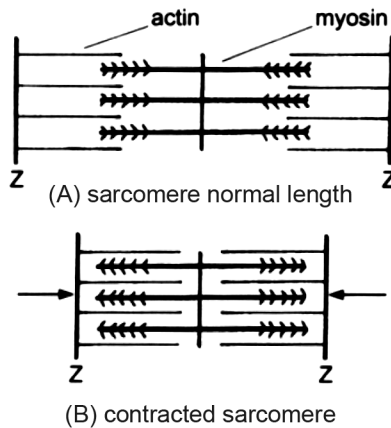
Responsible for the occurrence of DOMS are eccentric movements. These movements are defined as the condition when they are at the same time extensors and flexors around a joint, with the mission to keep the wrist in a neutral position of the load (Cheung, Hume, 2003). For example, imagine how painful the thigh muscles can be after going down a hill for a longer period when there is an eccentric muscle contraction, especially m. vastusmedialis. Such prolonged muscle activity will result in an energy deficit accompanying the formation of high-energy phosphate adenosine three-phosphate (ATP). As a result of an ATP deficit, there are primarily changes in the functioning of the basic elements of the contractile myofibrils i.e. sarcomere (Bonica, Sola, 1990).

## **Electrophysiology of muscle contractions and the mechanism for forming a muscle pain syndrome**

Thanks to the achievements and development of modern technological systems, researchers were able to visualize the structure of the very tiny muscle contractile system. By using a light and electronic microscope, it was shown that the sarcomere were composed of actin and myosin filaments, which were so arranged that in normal dormant muscles i.e. their basal tonus, actin filaments partially crept into myosin spaces (Bonica, Sola, 1990). Every myofibril consists of a chain of sarcomere which are connected to one another. Myosin fibers are dark and thick, and actin fibers are thinner and paler.

Figure 1. The strips are seen as darker transverse bars that separate the sarcomere from one another, which can show on the electronic microscope the relationship of these protein filaments in a state of contraction and relaxation.

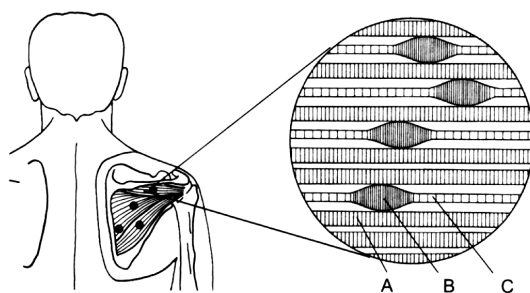
**Figure 1.** *The condition of sarcomere in a normal tonus and in a contraction*  
(From: Davies, Davies, 2005, 15)



It has been observed that in contracted muscle fibers strips Z are densely packed and that the length of the contracted sarcomere is 50% less than the relaxed fiber. Essential elements of each sarcomere are two contractile proteins, known as actin and myosin. The contraction occurs when the sarcomere actin and myosin molecules attach to one another and are in contact. For this to occur, ATP energy provided by the mitochondria is needed. This event shortens the sarcomere, which essentially also shortens the fine muscle fibers. The core of muscle contraction is the act of shortening the sarcomere. As it happens, almost one million sarcomere must be shortened in order to make a small movement. Muscle relaxation occurs when separating actin and myosin filaments, and for the act of separating actin and myosin filaments, a far greater volume of ATP is necessary (Bonica, Sola, 1990). Trigger point painful

nodules are formed when the actin fibers and myosin are stuck and cannot be separated due to a lack of ATP. This usually occurs locally in more muscle fibers in a particular muscle region. This situation with contracted sarcomere produces a painful nodule in certain regions of the muscle that makes trigger points a clinical entity of painful nodules (Dommerholt, 2011). Figure 2 shows a photograph of an electronic display of stuck sarcomere in multiple nodules in the area of the *m. infraspinatus*. The question is what the status of sarcomere in the area of nodes and outside them is, as well as the normal functional muscle fibers. On label A shown are muscle myofibrils in normal dormant muscles that are neither contracted nor relaxed. The subtle Z strips make restrictions to sarcomere, and are evenly distributed over the muscle fiber. Point B shows grouped contracted sarcomere in the area of nodules, which cannot separate their actin fibers and myosin clutch. C indicates the state of the sarcomere, which is stretched along the entire length of the muscle fiber to the locale of its attachment. In this area noticed is a greater distance between the Z strips in the entire length of the muscle fibers to the locale of the attachment.

**Figure 2.** *A diagram of trigger point pain nodules in m. infraspinatus with stretched painful muscle fibers outside* (From: Dommerholt, 2011, 24)



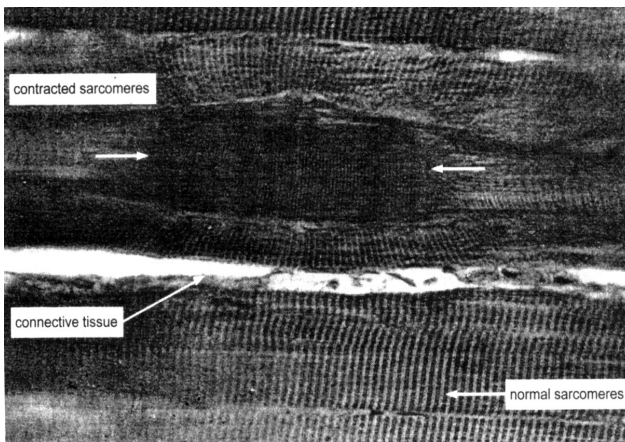
Noticed in this area was a greater distance between Z strips, which essentially reflected the continued tension and rigidity of these parts of the muscle filaments in front of and behind the painful nodules. This condition limits the elastic stretching of these muscle myofibrils and the impossibility of a full capacity of the muscle contraction, i.e. giving a muscle dysfunction with a painful feeling. The trigger point painful areas are formed in the muscle of its long and exhausting eccentric use (Dommerholt, 2011).

There are many papers on the subject of trigger point sore points. Researchers at the US National Institute of Health have developed a new technology that has enabled visualizing this area in the muscles. The scientific review of trigger points was enabled for researching by way of electrochemical methods, as electromyography could be used for

measuring the areas of electrical activity, and high and low frequencies, known as spontaneous electrical activity (SEA) (Benardot, 2010). It was thought that these points were places where the motor nerve of the muscle entered, somewhere in the middle of the muscle. However, it is now known that the electrical activity of the neuro-muscular membrane covers a wider area, and that it spreads widely in the muscle. By studying these painful trigger points and such changed structures of the sarcomere, many things were discovered. Researchers have found that trigger points may appear with a color Doppler ultrasound, when stimulated with vibration sono electro-stimulators (Figure 3) (Davies, Davies, 2005).

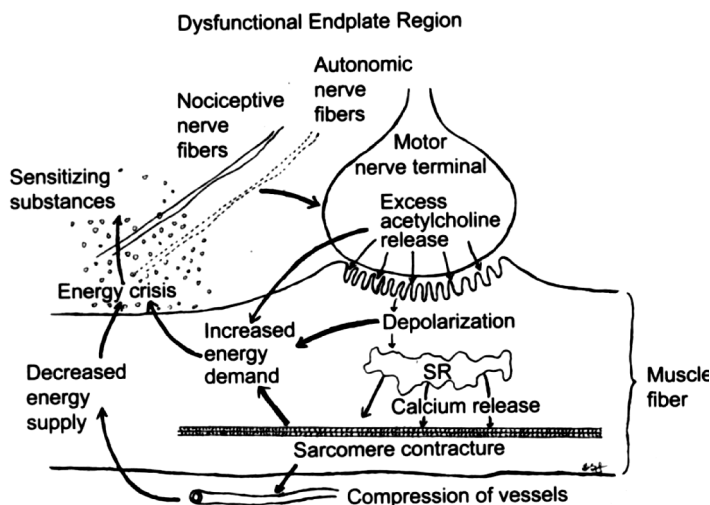
These places can be biopsies on animals and cadavers and thus we can study their electro-chemical alterations. They are an unusual mix of biochemical markers presented as an appropriate environment with an increased concentration of acidic decay products of metabolism, proinflammatory mediators and mediators that cause pain. Thus, in recent years a scientific presentation and explanation of muscle pain syndromes with physiological, chemical and cytohistology findings was given, with detailed structural images of muscle elements. It has been proved that nodules of contracted fibrils can make a compression on the surrounding capillaries in the regions around the node alterations of the muscle fibers, which sometimes completely cuts off the circulation in that region or diverts it. The result is the appearance of ischemia and oxygen deficiency in these regions. The lack of oxygen leads to a local dysfunction of mitochondria that cannot create the required amounts pursuant to the requirements of ATP. It all creates an image of oxidative stress with the formation of free oxygen radicals (ROS) (Benardot, 2010).

**Figure 3.** *A Doppler color scan of trigger point painful nodes in a muscle*  
(From: Davies, Davies, 2005, 26)



ATP has the function of contracting and relaxing myosin and actin fibers (Figure 4). When a nerve impulse is conducted to the muscle fibers and the neuro muscular plate, it empties out the deposits of calcium from the endoplasmic reticulum which results in the emptying out of acetylcholine (ACh) from vesicular enlargement to the nerve endings (Dommerholt, 2011). Calcium, thus freed, carries out the opening of the heads of actin which are then available to myosin that they can connect to, and thus start the contraction of the sarcomere. Sarcomere remain contracted until the energy of ATP no longer conditions the draining of actin and myosin. ATP is required to again take over Ca ions back into a sarcoplasmic reticulum, thus breaking the stimulus for the duration of the contraction for relaxation to take place. This mechanism reflects a significant impact of the energy crisis in the exhausted muscles, when calcium due to the inability to get back into the endoplasmic reticulum, permanently holds the connected actin and myosin fibers, i.e. the sarcomere are in constant contraction. Further studies have found that the lack of oxygen in the muscles occurs by releasing the protein “Calcitonin gene-related peptide” (CGRP), which enhances the release of ACh and thus prolongs and intensifies the contraction which in turn enhances the status of ischemia and the pain syndrome (Benardot, 2010).

**Figure 4.** *Neuro-muscle links and the significance of the calcium ion, as well as CGRP for an extended muscle contraction*  
(From: Davies, Davies, 2005, 27)



A CGRP peptide also stimulates the inhibition of the Ach enzyme that breaks down acetylcholine, extending the operating time of the discharged acetylcholine on the postsynaptic membrane. All these circumstances in

reality enhance the sarcomere contraction in the situation of a lack of oxygen and oxidative stress in certain muscle groups. Until this cycle is extended, there is a secretion inflammatory mediators and mediators that create the sensation of pain. All these mediators stimulate nerve endings and their nociceptors that give the local sensation of pain and edema, also sending signals of pain to the sensitive part of the cortex where the same sensations are processed in terms of individual perception of the quality and intensity of pain.

### **Oxidative stress**

Oxidative stress is presented as a condition of forming toxic reactive oxygen species (ROS) at increased volume in the working muscles, in a situation where there are no adequate amounts of antioxidants (Cheung, Hume, 2003). Such an unbalanced maintenance between (ROS) and antioxidants is bound to create resulting cell damage. Namely, by their penetration into the cells they carry out their own degradation and cell death. Our body performs an inhibition, i.e. deactivates ROS, creating antioxidants that are mainly certain groups of vitamins and minerals. Also studied and defined were the functions of essential antioxidants in the biochemical processes at the cell level.

*Vitamin C* at a dosage of 90 mg/day eliminates an oxidation reaction in leukocytes, lungs, gastric mucosa by reducing the lipid peroxidation of cells (Brayer, Goldfarb, 2006).

*Vitamin E* at a dosage of 15 mg/day globally impacts the preventive oxidation in cells (Maghan, 1999).

*Selenium* at a dosage of 55 mg/day achieves its antioxidant role through the creation of selenoproteins, which are defensive oxidative enzyme as they form a defense against oxidation and the creation of oxygen radicals. A daily intake of Se is based on the input that is required to form a selenoprotein with glutathione peroxidase (Maghan, 1999).

Beta-carotene at a dose of 700 to 900 mcg/day is sufficient to create retinol, i.e. Vitamin A. Beta carotene itself becomes an important biomarker for the use of fresh fruit, vegetables which through this biomarker realizes its antioxidant role. There is no doubt that the body through minerals and vitamins inhibits and neutralizes toxic oxygen radicals. Minerals exercise this role by regulating the enzymatic activity that reduces the generation of ROS. Vitamins achieve their effect by accepting the already created ROS from the cell environment and eliminating them, thereby also eliminating their harmful effects. Somewhat different are more recent views with taking vitamin E from previous ones. Earlier it was claimed that vitamin E as a fat soluble vitamin reduces the generated ROS radicals with their removal from the cellular environment. However, according to newer theories, Vitamin E supplementation by itself can destabilize the overall balance between oxidative



radicals and antioxidants, that is, slow down the whole antioxidant role of the body, if Vitamin E is taken arbitrarily. Therefore, the overall defense and prevention of damage from ROS is minimized (Benardot, 2010).

The proposed strategy is that the prevention of cell damage in the imbalance between oxidants and antioxidants is done with a regular intake of the cocktail of antioxidants from food, rather than as individual vitamin supplements. It is necessary to ensure that there is a continuous unchanged balance between ROS and antioxidants.

In special situations when ROS is generated in enormous amounts (intensive training, exhausting competitions) the body should be provided with an increased intake of antioxidative cocktails, which therefore provide a greater defense against toxic ROS radicals. The imbalance between ROS and antioxidants in addition to the pain syndrome produces the inflammatory syndrome accompanied by pain in the muscles and muscle dysfunction. Also, it can affect a decline in immune system function. An extremely high level of Vitamin E can create an inadequate function of the immune system. On the other hand, an inadequate intake of vitamins along with Fe, Se, Zn, Cu, Ca, Mg can also create immune problems (Shepard, Shek, 1998).

All this information leads to the conclusion that the balance between ROS and toxic antioxidants is very important, due to the enormous volume of ROS in different intense engagements of sports or conditions. Maintaining the balance of antioxidants is more important than giving priority to one, two or three of them, expecting that they can accomplish a more powerful biochemical effect of deactivating oxygen radicals.

### **Diet intake of supplements in athletes and DOMS in intense training sessions**

There is limited information that certain nutrients affect the occurrence of DOMS, and there are a number of studies under way now in this field. The nutrients evaluated include the following: alpha carnitine, vitamins C, E, D, flavonoids, long branched-chain amino acids, omega-3 fatty acids and certain proteins, all individually or in target combinations.

With fish oil, which is very rich in the content of Omega-3 fatty acids, with eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), it has been proved that all these entities in fish oil reduce the inflammatory status in the rheumatoid syndrome. Some studies based on this observation, prove that there is an intense buffering of the inflammatory response with fish oil (Tartibian et al., 2009). It achieves this effect with all three factual components and with inflammation that is created with intense physical activity training. A review on these observations confirms them with a number of established studies in this direction. However, the food taken was not tested, or the period



and the time compared to before and after the upcoming intense physical activity in sports. A review of these studies can be summarized to say that a nutrient deficit significantly affects various factors that influence poor muscle function (Benardot, 2010).

### **Some of the signs of muscle cell decay present in long-term oxidative stress**

*A disruption of the cytoskeleton* is present, but for this occurrence there is no proof which nutrient is responsible.

With *magnetic resonance imaging (MRI)* of a confirmed tissue disruption, it was shown that it can occur due to an insufficient intake of L-carnitine. Its intake should be 2 gr/day in order to remove this undesirable effect.

*Muscle strength* is weakened due to an insufficient intake of Vitamin C. This condition cannot be eradicated by taking in 500 mg of Vitamin C, together with an intake of 1200 IU Vitamin E.

*Interleukin 6 (IL-6) and C-reactive protein* as inflammatory markers are a sign of uncontrolled muscle inflammation. This condition can be controlled by an intake of a mixture of tocopherol, DHA, and flavonoids in combination.

*A late occurrence of DOMS* has been shown to occur as an indicator of the intake of antioxidants and an enormous production of ROS. This syndrome can be eradicated by taking Vitamin E 400 IU/day in a combination with Vitamin C, a cocktail of amino acids with branched chains to the amount of 5 gr/day, with L-carnitine at 2 gr/day (only for women).

*Creatine kinase (CK) and lactate dehydrogenase (LDH)* are the signs of cell deterioration in oxidative stress and other negative influences. This condition can be removed by taking Vitamin E (400 – 1200 IU), Vitamin C 1-3 gr/day, and L-carnitine 2gr/day.

*The range of motion*, as a test of impaired muscle function and status of supplementation with antioxidants, has not been researched.

*3-methyl histidine (3-MH)* as a humoral marker of muscle cell damage can be used as a test of deteriorating muscles in an insufficient anti-inflammatory supplementation. This state can be preventatively removed by taking beta-hydroxy and beta methylbutyric acid - 1.5 do 3 gr/day (Benardot, 2010).

### **Omega 3-fatty acids**

These fatty acids can reduce the occurrence and severity of the manifestation of DOMS by over two times. It performs an antioxidant and

anti-inflammatory function, and carries out its function by competing with arachidonic acid for creating eicosanoids which stimulate the formation of anti-inflammatory mediators for prostaglandin and thromboxane (Tartibian et al., 2009). It has been shown that fatty acids become part of cell membranes, thereby changing the release of inflammatory agents, including two series of prostaglandins, thromboxane and prostacyclin. It is possible that this anti-inflammatory effect reduces the occurrence or severity of the manifestation of the DOMS syndrome in persons exposed to intense eccentric muscle loads.

Studies have examined 27 males, with median age 33.4 years, assessing the occurrence of pain in the muscles after intense eccentric muscle loads. The individuals took fish oil capsules a day, which contained 324 mg of (EPA) acid, and 216 mg (DHA) acid. They also took Vitamin E (alpha thocopheryl acetate) 100 IU / day, in addition to omega-3 fatty acids. Supplementation was carried out 30 days before the intensive training and two days after training. It was found that people who consumed omega-3 fatty acids had better control of the emergence of pain than the muscle groups which were eccentrically-loaded. The pain after training was evaluated after 24 and 48 hours after training. The pain was significantly lower in the treated group after 48 hours of training, but did not differ in the pain score in the treated and untreated group 24 hours after training.

Another parameter that reflected the emergence of the inflammatory syndrome in intensive eccentric load is measuring the volume of the thigh, which should assess the possible occurrence of edema in the muscles that have been subjected to the above-mentioned loads. It was found that there was a significantly smaller thigh volume in the group that was treated in the manner mentioned above, omega-3 fatty acids and vitamin E, and therefore a lesser display of edema as a marker of muscle inflammation. Vitamin E, according to the study, would have the additional role of an antioxidant that reduces the formation of toxic radicals ROS, and consequently, contributes to a reduction of occurrence of DOMS.

A second study assessed the efficacy of omega-3 fatty acids on the formation of DOMS in 10 untrained patients. These were men between the ages of 22.7 years and six untrained women, average age 24.5 years. The individuals were organized into three groups and treated differently with supplements. Beyond that, evaluated was the effect of supplements on the incidence of DOMS. One group received fish oil capsules and the other group isoflavonoids in combination isolated from soy whey as non-omega-3 fatty acids. The third group was the placebo group. All three groups were supplemented 30 days before and one week after training. After training in 2, 4, and 7-day intervals, evaluated was intensity pain syndrome in all three groups. The individuals also received Vitamin E to help the appearance of fat oxidation. All of them had intensive training with eccentric movements of

twisting and bending, which provided intensive non-physiological loads for certain muscle groups (Benardot, 2010).

The conclusions of these studies are that in the group which took fish oil capsules the concentrations of EPA and DHA were significantly higher, but there was no difference in the occurrence of pending DOMS pain syndrome between the first two groups.

This study suggests that there may be a slight difference in controlling the occurrence of DOMS with dissimilar antioxidants and anti-inflammatory funds. It is important to take a specific mix of nutrients long enough for its effect to be evident.

### **Vitamin D**

The consumption of fish from the cold northern seas adds to the diet of athletes a rich source of EPA and DHA acids. They are also a great source of vitamin D. Thus treated patients had a good control of the occurrence of painful DOMS syndrome. Omega-3 fatty acids with EPA and DHA acids as well as Vitamin D are responsible for this effect (Houston, Cesari, 2007).

In Australia, studies have been made examining the connection between the real levels of vitamin D and the existence of chronic muscle pain in humans. Tested was a group of people who had muscle pain and another pain-free one compared with the humoral levels of vitamin D. It was found that the group that had pain had significantly lower levels of vitamin D, compared to the group without pain whose levels of vitamin D were satisfactory. None of the patients with a normal Vitamin D concentration had a pain syndrome (Tartibian et al., 2009).

The next phenomenon may be important, and it is related to the decrease in the concentration of Vitamin D in circulation and reducing of muscle strength. Another study looked at the assessment of muscle strength in a situation where the concentration of Vitamin D was deficient, insufficient compared to a normal concentration. The study group consisted of 976 persons. The parameter estimation of muscular strength was followed by a dynamometer that measured the strength of the fist grip. The study found that in the group with good levels of a Vitamin D status, there was a higher score than in the muscular strength in groups with deficit and insufficient concentrations. The occurrence of DOMS reduced muscle strength, and Vitamin D in this situation has the ability of easing the symptoms (Benardot, 2010).

It is thus very important to realize who needs to measure the level of Vitamin D. Of course, there are athletes who train indoors out of direct sunlight (gymnasts, figure skating on ice) that have an insufficient conversion of pro Vitamin D into Vitamin D. In this group, due to all the previous, it may be wise to do use Vitamin D supplements, which should reduce the occurrence of DOMS (Houston, Cesari, 2007).

## **Vitamin E**

Studies that have been researching the role of Vitamin E in the prevention and treatment of DOMS cannot give any clear conclusions. Mainly, Vitamin E is taken with Omega-3 fatty acids. It is known that Vitamin E is a strong antioxidant that acts to inhibit the cellular damage resulting from the influence of free oxygen radicals, as well as excessive muscle loads. The study which followed the anti-inflammatory role of Vitamin E examined female students that were involved in sports activities within university teams. They were divided into two groups. One group was given Vitamin E, tocopherol at a dose of 800 IU once a day, 14 days before the competition performances and seven days after the event. The other group was a placebo group. After eccentric regimen, tested was the occurrence of the pain syndrome 2, 4, and 7 days and the same pain was measured by a score that presents the strength of the pain. The group which took Vitamin E had a significantly lower score in the exercising of DOMS from the placebo group. Also, the treated group had significantly fewer performances of oxidative damage from the placebo group. Despite these findings, it can be said that Vitamin E has a significant anti-inflammatory role (Cheung, 2003).

In conclusion, we can say that athletes should access treating the antioxidant changes with Vitamin E with some caution, as it can block the antioxidant properties of the organism. However, if an athlete does not consume food that has enough of this vitamin, small or moderately-dose intakes can reduce DOMS performances.

## **Vitamin C**

The effect of Vitamin C on DOMS is still unclear. One study followed 18 men who had no training (age 23 years). The individuals were organized into groups that took 3 g Vitamin C, as well as a placebo group. The time of the intake of Vitamin C was two weeks before the show, and four days after training with eccentric movements. The score of pain syndrome in the treated group was lower compared to the placebo group, which was evaluated after 24 hours. Another study followed the effect of vitamin C on 24 untrained males and females. There was a treatment group which took vitamin C 3 g per day, three days before the appearance of training and five days after that. In doing so, there was no difference in the findings of recent pain syndrome between the treated and placebo groups (Brayer, Goldfarb, 2006).

In conclusion, it can be said that a factor of crucial importance for the effectiveness of treatment with Vitamin C and DOM performance is the length of the treatment. It should be borne in mind that 3000 mg of vitamin C is a very large dose, six times higher than usual. In addition, account should

be taken of the potential bad effects of this treatment (such as kidney stones and a decrease of cellular sensitivity to vitamins).

### **Essential amino acids/branched-chain amino acids (BCAA)**

Essential amino acids cannot be created in the body, they must be taken in by food. They may be the oxidized in skeletal muscles, and this oxidation is accelerating by exercise training, which makes it logical that these amino acids are taken as supplements during intensive training to make up for their loss. Without an adequate compensation of these amino acids, a significant inhibition of muscle proteins can occur (Davies, Davies, 2005).

A study which assessed the taking of BCAA supplementation and their effect on the occurrence of DOMS has shown that after the consumption of amino acids of this solution in a dose of 5 g, and 15 minutes prior to training performance, there were positive effects on the function and muscle pain states. The results showed that in women competitors, the score of pain performance was significantly lower in the treated group than the untreated. The assessment of pain score was carried out 24, 48 and 72 hours after training. The reason why men failed to show a better effect on the performance of pain syndrome type DOMS in the treated group than the placebo group indicated that a fixed dose of 5 g seems insufficient for men who have a higher percentage of muscle mass.

In conclusion, we can say that taking fixed-dose BCAA essential amino acids is not sufficient to create any benefits. Perhaps the dose for men can be effectively useful if 10 grams is taken in the same mode. This amino acid composition can effectively add carbohydrates and so it can make a cocktail that can be taken before and after the execution of the training (Davies, Davies, 2005).

### **Taking protein**

Training often leads to muscle damage, especially if accompanied with an eccentric engaging muscles and muscle groups. This muscle damage is associated with the disintegration of muscle proteins and a complete emptying of the glycogen deposits in the muscles. These findings indicate that there is a need to supplement protein, especially during the beginning of the new training program involving a large volume of strength training. The maximum rate of protein compensation shows that 1.5 g/kg body weight of TT protein is sufficient for the competitors, i.e. it is twice as high as in non-athletes (Dommerholt, 2011). Only minor changes in the choice of food can satisfy these needs, bearing in mind that in practice, many people – both athletes and non-athletes include extensive protein in their diets. A study of female students undergoing regular sports activities has shown that a regular

intake of protein and casein whey protein as a supplement has produced an improvement of the results of achievements in sport and the improvement in the composition of the body. Chocolate milk has been shown to be a highly effective drink for quick recovery from intense training, perhaps because the combination of carbohydrates and protein enables a faster recovery of normal muscle cells (Benardot, 2010).

The deduction is that even a small amount of high quality protein precisely measured may have an impact on reducing DOMS performance and improved results in sports. In general, the study indicates that it is very important to establish a permanent supply of proteins divided into smaller quantities (30 g) rather than taking a greater amount of protein at once.

### **Hydration**

Long-time experience has shown that dehydration is associated with a higher performance muscle pain syndrome. We studied the record score of pain syndrome in well-hydrated and dehydrated individuals before the appearance of physical fatigue. DOMS was evident in both groups, but the muscle pain in the dehydrated group was 44% higher than in a well hydrated group (Clary, Swinney, 2005).

The conclusion is that there are several reasons why athletes should be well hydrated. One reason is a lesser occurrence of pain syndrome in the muscles. Another reason is that the existence of a large pool of extracellular and intracellular liquids reduces the concentration of oxygen free radicals, which are diluted into the assay and therefore have less impact on the cell muscle damage.

Alcohol consumption among athletes, even on a sociological scale in sports or general life events, is reflected in the results achieved by the athletes. Although alcohol is a potential source of calories as it compensates for the 7 cal gr, it is regarded as anti-nutrition as it inhibits the conversion of Vitamin B according to its active co-enzymes involved in the generation of energy from carbohydrates, proteins and fats. Studies have shown that alcohol consumption, depending on the volume and frequency of its administration, can have a negative impact on cardiovascular function, energy metabolism, muscle damage and muscle recovery, the thermoregulatory mechanism and the neuromuscular function - and they all can very much affect the results in sporting achievements. It should be noted that the effects of alcohol consumption can last for a long time, for hours and days after taking (Benardot, 2010).

Alcohol shows the impact on results in sports via the changes of glycogen metabolism and the formation of its deposits by varying hydration and thermoregulation. The consumption of alcohol dramatically inhibits the formation of glycogen deposits in the liver cells and muscles, when compared

with a group that does not consume alcohol. In doing so, taking only 4% alcohol contributes to the extension of the recovery time. Alcohol is a diuretic because it supports a significant loss of fluid through urine.

In conclusion, we can say that the consumption of alcohol in athletes and on a sociological scale, significantly impacts the affect of dehydration depleted glycogen deposits, as both processes contribute to an inflammation of muscles and their dysfunctions.

## **CONCLUSION**

Our current understanding of the impact of nutrients on muscle pain syndrome can be summarized in five points:

- There is limited evidence of the impact of Vitamin C when taken independently, as well as when it is taken in combination with Vitamin E. There is also limited information about the importance of taking flavonoids when taken with tocopherol.
- Nutritional supplements are not beneficial to the elimination of muscle damage, but can be useful for reducing certain signs and symptoms of muscle load, including pain.
- If supplements are taken, they must be taken for days or weeks before the performance training and physical stress load with the expectation that there will be results. The optimum pretreatment period is unknown and varies for each nutrient.
- The optimal dose of nutrients individually which should reduce muscle damage and pain syndrome is unknown, as they have not been treated with different doses. Several studies have tested food intake to estimate the access to nutrients, whereupon a total deficit of nutrients could be ascertained.



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