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# Delayed onset muscle soreness (DOMS) management: present state of the art

VALENTINA CONTRÒ, ESAMUELA PIERETTA MANCUSO, PATRIZIA PROIA

### Abstract

This paper reviews existing theories explaining the delayed onset muscle soreness (DOMS) phenomenon. The authors describe the main symptoms of DOMS and their relation to different kinds of training strategies, and discuss the most popular muscle pain reduction interventions such as stretching, diet, dietary supplements, nonsteroidal anti-inflammatory drugs (NSAIDs), cryotherapy and cold water immersion, concurrent training, and the repeated bout effect.

**KEYWORDS:** delayed onset muscle soreness, DOMS treatment, stretching, cryotherapy.

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Corresponding author: [valentina.contro@unipa.it](mailto:valentina.contro@unipa.it)

*University of Palermo, Department of Psychological, Pedagogical and Educational Sciences, Palermo, Italy*

### What is already known on this topic?

Delayed onset muscle soreness (DOMS) occurs with repeated exposure to eccentric muscle contractions or to unaccustomed exercise. It continues to increase after exercise, reaching the peak between 24 and 48 hours after exercise. The mechanisms of DOMS have been widely discussed, but no clear explanation for its incidence has been offered as yet. There are many methods of DOMS treatment, however, none of them is really convincing.

### Introduction

It is commonly accepted that delayed onset muscle soreness (DOMS) occurs with repeated exposure to eccentric muscle contractions or to unaccustomed exercise [1, 2]. Generally, DOMS continues to increase after exercise with peaks between 24 and 48 hours after exercise. Although the exact etiology of DOMS remains unclear, several studies suggest that it is triggered by a variety of biochemical changes and inflammatory responses after muscle damage, rather than by a single harmful event [3, 4].

DOMS is the main cause of decline in physical performance including muscle strength and range of motion, both in athletes and non-athletes, and often brings a psychological distress.

For many years the DOMS phenomenon has been erroneously attributed to the accumulation of lactate in muscles after an intense workout, bringing this baseless theory to be widely spread among athletes and amateurs. However, this hypothesis has been demonstrated inconsistent. The pain that comes from intense eccentric exercise and its perception, are not at all related to lactate accumulation [5].

Indeed, blood and muscle lactate levels rise significantly during intense eccentric exercise and concentric anaerobic exercise, but they return to baseline in a shorter time than DOMS, which increases in the first 24 hours following the exercise, and peak between 24 and 48 hours, when blood lactate levels are already largely at a normal level [6, 7].

### Theory of DOMS

Although several factors have been suggested as possible causes of DOMS, there is not one clear and satisfactory

explanation. The precise causes of DOMS are currently unknown, and DOMS is generally described as a consequence of mechanical and metabolic stress induced by physical activity [4, 8]. Muscle soreness is often associated with muscle microtraumas, but it seems a quite vague definition. Although it appears likely that intense exercise can cause microtraumas, evidence does not strongly support this idea, and some studies seem to contradict it [9, 10].

In the past, some researchers suggested that DOMS-related factors presumably resulted to a great extent from tearing of the muscle connective tissue and its tendon insertion. They noted that the urinary excretion of hydroxyproline amino acid, a specific product of the connective tissue catabolism, was higher in subjects who had felt muscle soreness than those who had not. Because a significant increase in urinary hydroxyproline levels indicates also an increased degradation of collagen synthesis, they concluded that an intense workout damages the connective tissue, and thus increases the degradation of collagen, creating an imbalance in its metabolism [11]. Other researchers have supported this theory, claiming that the occurring breakage in non-contractile elements of sarcomeres (such as the sarcoplasmic reticulum) and connective tissue that surrounds muscle proteins (sarcolemma), resulting from an irreversible deformation during an eccentric contraction, which destroys components of the sarcomere and can lead to an apoptotic response [12, 13].

In a comprehensive review, Armstrong in 1984 proposed his own theory on DOMS development, according to which the exercise-induced structural damage to the sarcolemma altered the permeability of cell membranes, allowing an influx of calcium into the interstitial site. High levels of calcium in the cell activate a calcium-dependent proteolytic enzyme that degrades Z lines, troponin, and tropomyosin. This gradual destruction of the sarcolemma after exercise allows intracellular components to diffuse into the interstitial space and plasma, attracting lymphocytes in the damaged area, causing accumulation of substances such as histamine, quinine and potassium, which could stimulate nociceptors [14].

In 1991, Smith suggested that the acute inflammation in response to muscle and connective tissue damage caused by eccentric exercise is the main mechanism involved in DOMS development, since many symptoms of acute inflammation such as pain, swelling, and loss of functionality, occur concurrently with DOMS [15]. However, recently Mizumura et al. have found that muscle fiber damage is not essential, although it is sufficient, for induction of DOMS. Instead, nerve growth

factor (NGF) and glial cell line-derived neurotrophic factor (GDNF) produced by muscle fibers/satellite cells play crucial roles in DOMS. They observed in rats that two pathways were involved in inducing mechanical hyperalgesia after eccentric contraction: activation of the B2 bradykinin receptor-NGF pathway, and activation of the COX-2-GDNF pathway [16].

Another less popular theory is the “metabolic stress”, which argues that muscle cells produce some substances during intense activity to which they may be unable to adapt. However, this concept is still hard to define. Researchers have identified several molecules produced by the cells during exercise, but are not certain of the molecules-metabolic stress link. A link between DOMS and any biological marker has not been found yet [9].

Studies have also focused on the production of free radicals. These highly reactive molecules are an inevitable by-product of cellular metabolism. There has been growing evidence that the involvement of reactive oxygen species (ROS) can be detected at the beginning of DOMS. However, there is no correlation between the peak of free radicals concentration and the peak of DOMS. On the contrary, the increase in free radicals occurs after the peak of muscle activity and DOMS [17]. In other words, they may be involved, but the relationship is indirect and unclear.

More recently, a new theory on DOMS has emerged, examining its possible association with the mechanism of “coupling excitation/contraction” of the myosin bridges attached to actin [18]. Lamb [19] explains that the release of calcium ions (from the sarcoplasmic reticulum), which initiates the “power stroke movement”, i.e. actin sliding on myosin, can be stretched more significantly with eccentric rather than concentric contractions. According to Lamb, this mechanism of “coupling excitation/contraction” followed by the substantial release of calcium ions causes a rupture of the sensory regulators in the sarcomeres (controlling the neural input into the muscle), which also contributes to the DOMS formation after eccentric exercise.

Moreover, recent evidence shows that DOMS can actually spread, likely through a neurological mechanism, to adjacent muscle groups, even if they are not trained, and this creates an additional question on the causes and mechanisms involved in this reaction [20].

### **Types of contraction**

It has been well documented that eccentric contractions produce greater muscle damage and deficits than concentric or isometric contractions such as running downhill, plyometric exercises, and traditional resistance

training [12]. The harm itself is the result of eccentric exercise, which causes muscle cell damage, and triggers an inflammatory response. This damage is noticeable with the breaking of the alignment bands or with the complete interruption of the Z-lines of the sarcomere. This damage causes the release of enzymes, including creatine kinase (CK), constantly on the increase within 1-3 days of eccentric exercise, contributing to the strength deficit typical of DOMS [3].

In the eccentric muscle contraction, swelling due to the production of prostaglandin E2 can be observed from 24/48 up to 72 hours after exercise. Prostaglandin E2 also sensitizes the afferent fibers of type IV muscle, responsible for dull pain transmission to the central nervous system [21].

### **Symptoms and training**

Symptoms typically associated with DOMS include loss of strength, pain, muscle weakness, stiffness and swelling. The loss of strength reaches the peak in the first 48 hours following exercise, and full recovery can extend up to five days. The pain peaks within 1-3 days after exercise, and generally regresses within a maximum of 7 days. Stiffness and swelling may rise after 3-4 days of exercise and usually resolve within 10 days. It is important to note that these symptoms are not dependent on each other, and do not always co-occur [3, 7]. Although DOMS was in the past associated with muscle swelling (temporary hypertrophy) [22], more recent research has belied this connection [23].

Since DOMS is an indication of tearing of muscle fibers as a result of eccentric muscle work, some coaches might advise against exercise until the pain is completely gone. This is because of the assumption that new eccentric training during DOMS would exacerbate muscle damage and have a negative impact on recovery and supercompensation. Actually, some studies have disproved these theories confirming instead that training with DOMS is possible without worsening muscle damage [24]. Even more so the intensity of DOMS perception is not proportional to muscle damage [25]. Indeed, one study found that nearly one third of subjects undertaking maximum eccentric contractions did not report any significant muscle soreness [26].

Because DOMS may be erroneously interpreted as a signal of necessary recovery, a strength decrease in the affected muscle can be assumed as long as it is perceived. It has been shown that DOMS affects expression of muscle strength for up to 24 hours after exercise, even by altering the activity of antagonist muscle through reducing the discharge rate of motor units.

This response could be attributed to a self-protection mechanism preventing further injuries, because DOMS has also been shown to alter the biomechanics of walking and running [27]. This may be correct only if it could stimulate the same muscles the next day – a strategy not practiced because of the recovery need of at least 48 hours. Respecting, however, a muscle rest on the day stressed, DOMS does not increase perceived exertion [28].

### **DOMS treatment**

DOMS prevention and treatment are of great interest to coaches, instructors and therapists because of associated pain and discomfort, which may affect athletes' physical training and performance. Despite a significant amount of research on the DOMS treatment, few authors have indicated a predominant way of treating or preventing it.

### **Stretching**

For several years in the past, static stretching was recommended as a way to warm up muscle groups at the beginning of resistance training, since it was believed that this kind of stretching could prevent delayed onset muscle soreness. However, much scientific evidence has univocally denied the effectiveness of this practice [29]. Contrary to other treatment methods, which can be relatively useful to contain DOMS development, it has been shown that stretching has no beneficial effects on stopping this physiological reaction, either before, during, or after physical training [30, 31].

### **Supplementation**

Supplements have emerged as one of DOMS treatment methods. Supplementing endogenous defense systems with additional oral doses of antioxidants has received much attention as a non-invasive strategy to prevent or reduce oxidative stress, reduce muscle damage and improve athletic performance. Coenzyme Q and l-carnitine have been examined as potential DOMS treatment supplements; however, none has been shown to effectively treat this reaction, but to possess the capabilities to cause adverse effects [3].

Some studies have shown negative effects of antioxidant supplementation on health and exercise performance. Indeed, although ROS are associated with harmful biological events, they are essential for cell development and function [32, 33]. There is also some evidence of DOMS attenuation following the use of some antioxidants such as epigallocatechin gallate (in green tea) and N-acetyl-cysteine [34].

Jackman et al. [35] reported a reduction in DOMS after an intake of branched-chain amino acids (BCAA). They found that BCAA intake in strategic phases throughout the day reduces DOMS caused by the eccentric high intensity. Also a decrease in muscle soreness by 64% was noted up to 72 hours following the intake of BCAAs compared to placebo.

Other authors examined the impact of a 5 g intake of BCAAs before a high volume squat session, and found that DOMS levels were significantly reduced and power had been maintained at 48 hours after exercise compared to the effects of an isocaloric placebo [36]. The same researchers confirmed the BCAA benefits on DOMS, testing an exercise consisting of squats (7 sets of 20 repetitions with 3-minute rest): a placebo or 100 mg/kg of BCAAs (about 9 g for a person of 90 kg) was administered to subjects. The group that had taken BCAAs experienced a DOMS reduction at 48 and 72 hours post-exercise [37].

More recent evidence has shown that also taurine, citrulline malate, N-acetyl-cysteine and glutamine intake can contribute to DOMS reduction [34, 38, 39, 40].

#### **Dietary intake**

Some studies have shown that consumption of milk or protein/carbohydrate-based supplements with milk immediately after exercise can limit DOMS perception from 24 to 48 hours later [41].

Another recent study showed that an intake of chocolate milk is capable of reducing DOMS more than an isocaloric drink based on carbohydrates [42], and watermelon juice, probably due to citrulline naturally contained in watermelons [43]. More recently, also curcumin displayed similar properties, reducing inflammatory cytokines and increasing muscle regeneration in mice [44].

Furthermore, it has been noted by many authors that caffeine can lead to a DOMS reduction. Taking caffeine within 24-48 hours after an eccentric activity (approximately two cups of coffee) can produce a significant reduction in pain up to 48% [45]. Hurley et al. [46] have shown that consuming 5 mg/kg of caffeine (about three cups of coffee) one hour before resistance training could possibly reduce DOMS.

#### **NSAIDs**

Nonsteroidal anti-inflammatory drugs (NSAIDs) have been said to have a strong counteracting effect on inflammation and swelling that occur with muscle damage. Despite this theory research on the effectiveness of NSAIDs has produced conflicting results. Because

of the inconsistencies of various studies regarding the types and doses of several NSAIDs, as well as the negative NSAIDs associated side effects including gastrointestinal pain and hypertension, NSAIDs do not seem to be an optimal choice for DOMS treatment [3]. It has been found that these drugs can rather compromise the hypertrophic response [47, 48], with the inhibition of the pathway by which nitric oxide (NO) and hepatocyte growth factor (HGF) activate satellite cells in the early stages of recovery, which seems to be partially regulated by the cyclooxygenase-2 (COX-2) enzyme, which releases various prostaglandins known to stimulate satellite cells [49, 50].

#### **Cryotherapy and cold water immersion**

For a few years the use of unconventional cryotherapy (extreme cold air exposure) [51] or methods commonly used to relieve inflammation due to injuries have been proposed as ways of DOMS treatment. However, researchers have not been able to clearly demonstrate any significant therapeutic difference after the application of such methodology.

The protocol of ice-water immersion used by Sellwood et al. [21] was ineffectual in minimising DOMS markers in untrained individuals, but results are still conflicting. One study compared DOMS treatments with cold water (20°) and hot water (38°) for 30 minutes, and it was found that an immersion in hot water could reduce DOMS most efficiently [52].

A review of 27 articles by Hohenauer et al. [53] revealed that cooling and especially cold water immersions significantly affected DOMS symptoms as compared with control conditions after a 24-hour recovery. Also Machado et al. [54] claim that cold water immersion for 11-15 minutes can be slightly better than passive recovery in the management of muscle soreness.

#### **Concurrent training**

Concurrent training involves resistance training and aerobic training performed in succession within a single training session. Aerobic exercise may be a recommended practice following eccentric exercise as a method to mitigate DOMS.

Tufano et al. [55] tested the effect of 20 minutes of low and moderate intensity aerobic activity or complete rest following an exercise with overloads consisting of 60 eccentric repetitions on the subsequent manifestation of DOMS. The exercise at a moderate intensity was shown to lead to a DOMS reduction compared to low intensity or complete rest.

### Repeated bout effect

The “repeated bout effect” (RBE) is the muscle’s ability to adapt, by reducing DOMS response, to mechanical stimuli caused by muscle contraction. In other words, the RBE indicates that during a workout with weights, repeated in the next training session, DOMS response and, therefore, perceived pain, are weaker than in the first session [56].

It seems that one of the ways to prevent or reduce DOMS (or to accelerate the recovery) is to stimulate muscles with lower volume eccentric exercises about a week or more before the high volume eccentric training session [57, 58].

The mechanisms underlying the RBE are not completely understood, although Deyhle et al. [59] have recently demonstrated that inflammation is not attenuated following a repeated bout of lengthened contraction.

### Conclusion

The current literature concerning DOMS management is still emerging. The results of this systematic review indicate that mechanisms underlying DOMS are still not well understood, and so are the pathways of their limitation and management. There are conflicting results for almost any therapy considered in this review, especially cryotherapy, NSAIDs, and antioxidants. Instead, encouraging results come from the study of other supplements or special foods, as well as from the different kinds of training before or after the onset of muscle soreness.

The existing literature provides some evidence for utility of methods in DOMS management, but their limitations should be considered prior to their application.

#### What this study adds?

This study is an up-to-date discussion about the possible ways of prevention or at least reduction of the DOMS syndrome effects, e.g. stretching, dietary supplements, nonsteroidal anti-inflammatory drugs, cryotherapy and cold water immersion, concurrent training and repeated bout effect.

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