DELAYED ONSET MUSCLE SORENESS (DOMS) – A THREAT OR A HARMLESS CONDITION

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INTRODUCTION

Muscle soreness is a well-known phenomenome for all categories of people and in different age (Dimitrova, 2016; Dimitrova, 2019; Ignatova, 2018; Ignatova [Игнатова], 2018; Chipeva, 2018). This can happen in our everyday life due of the professional fatigue (Dimitrova, 2019a; Dimitrova, 2020a; Polimenov, 2019). Sometimes it's come after some fitness exercises (Nesheva, 2019; Dimitrova, 2017). It is possible to feel muscle pain during your walking promenade (Dimitrova, 2019b; Dimitrova, 2019d). In all this situations is useful to apply Spa therapies (Dimitrova, 2019c; Dimitrova, 2020).

This original paper presents a specific analysis of the muscle soreness process in both points of view from athletes, as well from doctors, working in the field of sports medicine. Still, there are many uncertain facts, which concern this interesting and important area.

Immediate and delayed-onset muscle soreness differs mainly in chronology of presentation. Both conditions share the same quality of pain, eliciting and relieving activities and a varying degree of functional deficits.

As I previously said, Delayed onset muscle soreness (DOMS) is a familiar experience for the elite or novice athlete. Symptoms can range from muscle tenderness to severe debilitating pain. One of the specific features of DOMS is the development of clinical symptoms with delay-peak soreness at 48 - 72 h post-exercise, as a result of complex sequences of local and systemic physiological responses.

The mechanisms, treatment strategies, and impact on athletic performance remain uncertain, despite the high incidence of DOMS. DOMS is most prevalent at the beginning of the sporting season when athletes are returning to training following a period of reduced activity. DOMS is also common when athletes are first introduced to certain types of activities regardless of the time of year. Eccentric activities induce micro-injury at a greater frequency and severity than other types of muscle actions. They are the strongest trigger of DOMS (e.g. lengthening contraction, such as quadriceps while descending). DOMS is much weaker after the next workout, but the first bout can be so fierce that people avoid starting valuable exercise programs, especially strength training. It's worse for some people due to genetic factors and other biological stresses. The intensity and duration of exercise are also important factors in DOMS onset. Its typical sign is mechanical hyperalgesia (tenderness and movement related pain). The involved muscles, after such exercise, adapt rapidly to prevent muscle damage, and thereby occure soreness, if the exercise is repeated.

METHODS

Although the exact pathophysiological pathway of DOMS remains unknown, the primary mechanism is currently considered to be the ultrastructural damage of muscle cells due to unfamiliar sporting activities or eccentric exercise, which leads to further protein degradation, apoptosis and local inflammatory response. It is considered that there are up to six hypothesized theories, which have been proposed for the mechanism of DOMS. Namely they are: lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation and the enzyme efflux theories. However, an integration of two or more theories is likely to explain muscle soreness.

The developing pathway - begins with micro trauma to muscles and then surrounding connective tissues. Microtrauma is then followed by an inflammatory process and subsequent shifts of fluid and electrolytes. Throughout the progression of these events, muscle spasms may be present, exacerbating the overall condition .

In the pathogenesis of DOMS, it is revealed that the increased staining of actin and desmin reflects an increased synthesis of these proteins as part of an adaptation process following the unaccustomed eccentric exercise.

In the process of developing DOMS, there are two pathways involved in inducing mechanical hyperalgesia after lengthening contraction : activation of the B2 bradykinin receptor-nerve growth factor (NGF) pathway and activation of the COX-2-glial cell line-derived neurotrophic factor (GDNF) pathway. These neurotrophic factors are produced by muscle fibers and/or satellite cells. This means that muscle fiber damage is not essential, although it is sufficient, for induction of DOMS instead, NGF and GDNF produced by muscle fibers/satellite cells play crucial roles in DOMS.

It is also investigated that neutrophil mobilization and migration after exercise may be involved in the muscle damage and inflammatory processes of this type of muscle soreness.

Besides, the following model explaining the etiology and cellular mechanisms of the phenomenon DOMS, may be proposed: 1) high tensions (particularly those associated with eccentric exercise) in the contractile/elastic system of the muscle result in structural damage; 2) cell membrane damage leads to disruption of Ca++ homeostasis in the injured fibers, resulting in necrosis that peaks about 2 days post-exercise; and 3) products of macrophage activity and intracellular contents accumulate in the interstitium, which in turn stimulate free nerve endings of group-IV sensory neurons in the muscles leading to the sensation of DOMS.

Recently, it is considered that Delayed Onset Muscle Soreness is, in fact, neural microdamage rather than muscle damage. According to this hypothesis, DOMS is an acute compression axonopathy of the nerve endings in the muscle spindle. It is caused by the superposition of compression when repetitive eccentric contractions are executed under cognitive demand. The acute compression axonopathy could coincide with microinjury of the surrounding tissues and is enhanced by immune-mediated inflammation. DOMS is masked by sympathetic nervous system activity at initiation, but once it subsides, a safety mode comes into play to prevent further injury. DOMS becomes manifest when the microinjured non-nociceptive sensory fibers of the muscle spindle stop inhibiting the effects of the microinjured, hyperexcited nociceptive sensory fibers, therefore providing the 'open gate' in the dorsal horn to hyperalgesia. Reactive oxygen species and nitric oxide play a cross-talking role in the parallel, interlinked degeneration-regeneration mechanisms of these injured tissues. Probably, the mitochondrial electron transport chain generated free radical involvement in the acute compression axonopathy. 'Closed gate exercises' could be of

nonpharmacological therapeutic importance, because they reduce neuropathic pain in addition to having an anti-inflammatory effect.

RESULTS

For the last 3 decades, the DOMS phenomenon has gained a considerable amount of interest amongst researchers and specialists in exercise physiology, sports, and rehabilitation fields. There has been a variety of published studies investigating this painful occurrence in regards to its underlying mechanisms, treatment interventions, and preventive strategies. However, it is evident from the literature that DOMS is not an easy pathology to quantify, as there is a wide amount of variability between the measurement tools and methods used to quantify this condition. It is obvious that no agreement has been made on one best evaluation measure for DOMS, which makes it difficult to verify whether a specific intervention really helps in decreasing the symptoms associated with this type of soreness or not. Thus, DOMS can be seen as somewhat ambiguous, because many studies depend on measuring soreness using a visual analog scale (VAS), which is a subjective rather than an objective measure. Even though needle biopsies of the muscle, and blood levels of myofibre proteins might be considered a gold standard to some, large variations in some of these blood proteins have been documented, in addition to the high risks sometimes associated with invasive techniques.

Therefore, in order to diagnose DOMS, a thermal infra-red (IR) imaging technique of the skin above the exercised muscle to detect the associated muscle soreness, is used. The main purpose is to examine changes in DOMS using this safe and non-invasive technique.

It is of great importance to investigate the mechanisms of DOMS, in order to draw conclusions about its consequences. DOMS can affect athletic performance by causing a reduction in joint range of motion, shock attenuation and peak torque. Alterations in muscle sequencing and recruitment patterns may also occur, causing unaccustomed stress to be placed on muscle ligaments and tendons. These compensatory mechanisms may increase the risk of further injury if a premature return to sport is attempted.A number of treatment strategies have been introduced to help alleviate the severity of DOMS and to restore the maximal function of the muscles as rapidly as possible.

To summarize, DOMS is probably not caused by micro-trauma — a popular old idea — although it might be a mild form of “rhabdomyolysis,” which is caused by muscle proteins spilling into the blood. Some kind of “metabolic stress” may be a more likely culprit, and yet there is no clear link between DOMS and any specific biological marker. There are even clues that DOMS is neurological. Certainly it is not straightforwardly inflammatory: evidence suggests that inflammation is what reduces DOMS pain as you continue to exercise. Mysterious indeed!

Furthermore, to define muscle fever — such a wonderfully descriptive term — is that distinctive muscle pain that nearly everyone experiences after intense or unfamiliar exercise, often peaking as long as a day or two later. Because of the delay, it is best known as DOMS for delayed-onset muscle soreness. Sometimes DOMS is so severe that it is mistaken for a muscle strain, an actual injury.

Muscle fever is a great term because DOMS makes your muscles feel sickly and gross as well as sore. Weakness is another symptom, major and measurable — but only hardened competitors are likely to test their strength while feeling so sore (though they probably should not). The nastiness starts after a bit of

a delay, often after sleeping, and then continues for 24 to 72 hours. Some people don't even notice it until the second day. If you do the same workout again a few days later, it's nowhere near as bad.

DISCUSSION

There are treatment strategies, which have been introduced to help alleviate the severity of DOMS and to restore the maximal function of the muscles as rapidly as possible.

- Nonsteroidal anti-inflammatory drugs have demonstrated dosage-dependent effects that may also be influenced by the time of administration.
- Similarly, massage has shown varying results that may be attributed to the time of massage application and the type of massage technique used.
- Compression garments used during the post exercise period could be an effective way to reduce DOMS and accelerate the recovery of muscle function.
- Cryotherapy, stretching, homeopathy, ultrasound and electrical current modalities have demonstrated no effect on the alleviation of muscle soreness or other DOMS symptoms.
- Exercise is the most effective means of alleviating pain during DOMS, however the analgesic effect is also temporary.
- Exercises targeting less affected body parts, while the most affected muscle groups recover

Athletes who must train on a daily basis should be encouraged to reduce the intensity and duration of exercise for 1-2 days following intense DOMS-inducing exercise. Alternatively, exercises targeting less affected body parts should be executed, in order to allow the most affected muscle groups to recover. Eccentric exercises or novel activities should be introduced progressively over a period of 1 or 2 weeks at the beginning of, or during, the sporting season in order to reduce the level of physical impairment and/or training disruption.

The features of these eccentric contractions can be seen on chart 1 below.

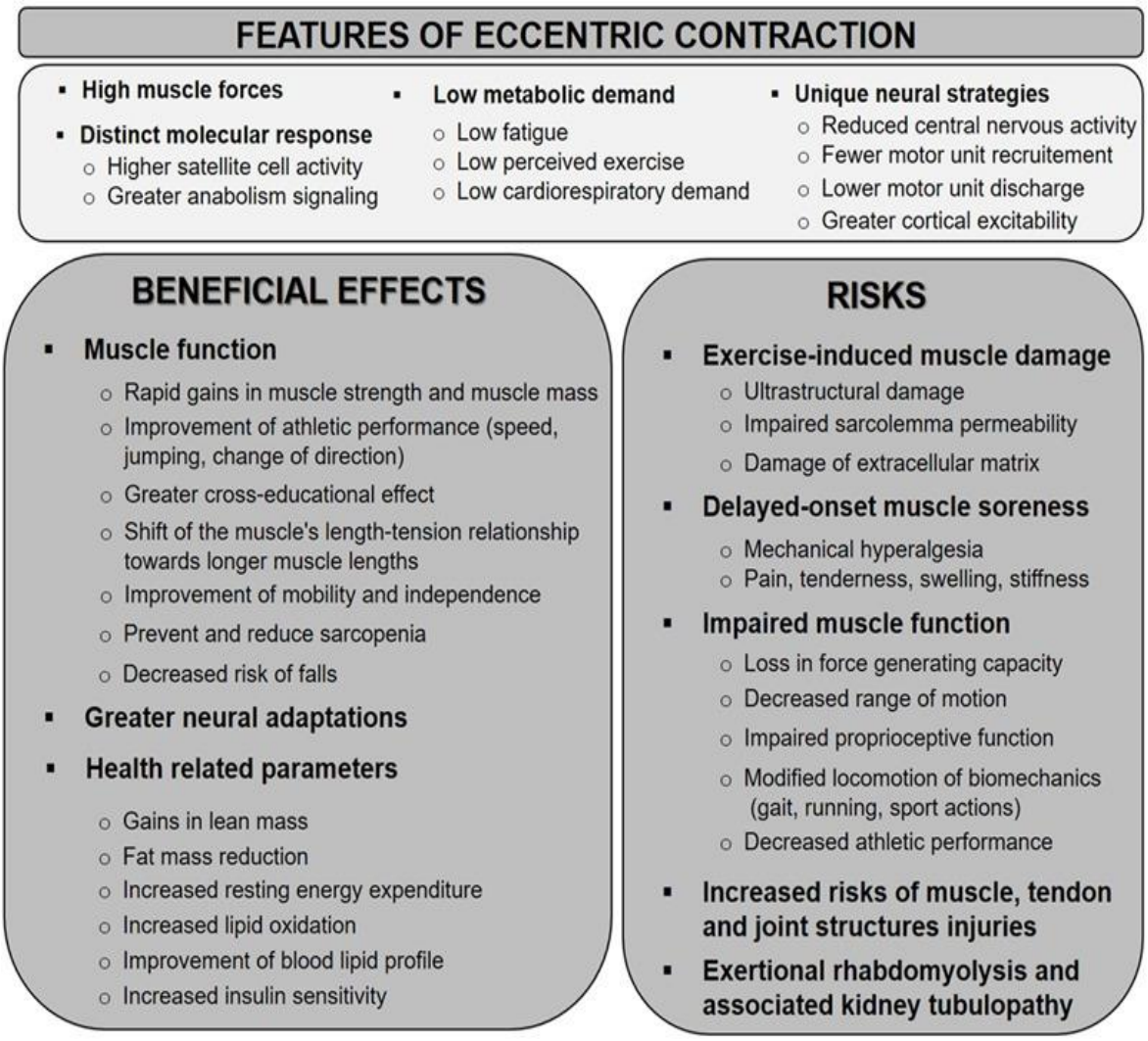

Chart 1. *Eccentric contractions- when to use and when to avoid them*

Although training is considered to prevent muscle soreness, even trained individuals will become sore following a novel or unaccustomed exercise bout. The most useful preventive strategy to avoid the adverse effects of DOMS, consists of repeating sessions involving submaximal eccentric contractions whose intensity is progressively increased over the training.

Despite an increased number of investigations focusing on the eccentric contraction, a significant gap still remains in the understanding of the cellular and molecular mechanisms underlying the initial damage response and subsequent adaptations to eccentric exercise. Yet, unraveling the molecular basis of exercise-related muscle damage and soreness might help uncover the mechanistic basis of pathological conditions as myalgia or neuromuscular diseases. In addition, a better insight into the mechanisms governing eccentric training adaptations should provide invaluable information for designing therapeutic interventions and identifying potential therapeutic targets.

CONCLUSION

Inspire everything known about DOMS, there are still many unanswered questions and many potential areas for future research.

Overall, DOMS is annoying and style-cramping, particularly if you're starting or restarting an exercise regimen, especially strength training — which is much more worthwhile than most people realize, and DOMS is one of the main things that discourages people.

That is why, it is strongly recommended to investigate more performant factors and mechanisms of this type of muscle soreness, as well as find better treatment strategies to cure clinical symptoms of athletes in order to help them improve their achievements.

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