

The Relationship Between Delayed Onset Muscle Soreness (DOMS) and Inflammation and Oxidative Stress

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Introduction

Muscle is the main part of the movement system, accounting for more than 40 per cent of the body's total weight. Muscles undergo wear and tear with the activities that take place during the day and as a result, muscle soreness occurs. The general features of muscle soreness are mainly widespread pain, located in the distal somatic area and usually accompanied by changes in muscle stiffness (Mizumura & Taguchi, 2024). Delayed onset muscle soreness (DOMS) is described as muscle tension characterised by persistent pain sensations and additional stiffness with increasing inflammation (Gulick & Kimura, 1996; MacIntyre, Sorichter, Mair, Berg, & McKenzie, 2001). Delayed onset muscle soreness (DOMS) is the process in which muscle soreness manifests itself very intensely in the 24-48 hour period, which manifests itself as muscle soreness 8 hours later exercise, and gradually alleviates the pain with physiological changes in muscle tissue within an average of 6 days (Wolska et al., 2023). DOMS is a common process in elite or beginner athletes. Symptoms can range from muscle tenderness to severe pain. Delayed onset pain is a gradual process that can lead to pain in the skeletal muscle and a decrease in muscle capacity. In addition, exercise-related complications such as fatigue, haematuria, etc. can be seen in a large proportion of patients. Delayed onset muscle soreness (DOMS) is often thought of as a condition that athletes experience when they return to training after a period of holiday, but in general it is characterised by its occurrence when athletes are new to certain types of exercise, regardless of the time period (Cheung, Hume, & Maxwell, 2003). Strenuous and unusual physical exercises of sufficient intensity and time involve extreme muscle contractures, which often result in temporary skeletal muscle microdamage (SMITH, 1991; Torres, Ribeiro, Alberto Duarte, & Cabri, 2012). Exercise-caused muscle injury is a difficulty faced by all athletes in different sports. Such muscle damage can disrupt the athlete's training continuity and consequently impair the athlete's overall performance. Mechanical damage to muscle fibres caused by exercise can lead to deformation of the fibres in the muscle (Owens, Twist, Copley, Howatson, & Close, 2019). This damage to muscle fibres may cause apoptosis of muscle fibres by disrupting the control of Ca^{+2} secretion (Gissel & Clausen, 2001). The apoptosis that occurs initiates an inflammatory chain that damages proteins and produces reactive oxygen species (ROS) that interfere with stimulation-contraction coupling, causing a negative process in muscle performance (Kozakowska, Pietraszek-Gremplewicz, Jozkowicz, & Dulak, 2015). One of the most important factors that protect our health and contribute to the prevention of diseases is regular exercise routine. On the other hand, performing physical activity at irregular intervals and with high intensity may lead to oxidative stress and inflammation, triggering numerous adverse processes in metabolism instead of promoting a positive impact on health. (Gomez-Cabrera,

Domenech, & Viña, 2008).

Aim of the Study

The aim of this study is to summarize and explain the relationship between oxidative stress and inflammation by interpreting the doms state seen due to muscle damage within the framework of relevant studies in the literature.

Delayed Onset Muscle Soreness (DOMS) and Oxidative Stress

“Delayed onset muscle soreness” (DOMS) is clinically recognised primarily by high expression of oxidative stress and pro-inflammatory markers (Kanda et al., 2013). During and after physical activity, mitochondrial activity increases, and factors such as reperfusion, NADPH oxidase complex, and leukocyte activation contribute to the generation of free radicals within the metabolism (Thirupathi & Pinho, 2018). Mitochondria, the energy centre of the cell, have a primary role in achieving homeostasis at the cellular level (Mezhnina, Ebeigbe, Poe, & Kondratov, 2022). It is also a source of free radicals in many pathways, especially during energy synthesis (Zhao, Li, Li, & Chen, 2022). The detection of very high amounts of biomarkers of free radical damage in muscle tissue in delayed onset muscle soreness (DOMS) suggests that damaged mitochondria produce more ROS than other mitochondria (Li, Peng, Sun, & Si, 2023). Exercise-induced free radicals production is thought to be a mechanism that causes muscle damage (LEE et al., 2002). In the process in which force is obtained by muscle contraction, low ROS level positively affects the process by inducing cell signalling, while high ROS level causes a decrease in force production and leads to muscle fatigue, although it varies depending on the process and dose (Radak, Zhao, Koltai, Ohno, & Atalay, 2013). Oxidative stress is a condition that occurs when there is an imbalance in metabolism, marked by an increase in free radicals and oxidant compounds and a decrease in antioxidant levels. This imbalanced process is due to many causes, but most often occurs either due to decreased antioxidant production within the body or inadequate antioxidant intake from external sources (Fernández-Lázaro et al., 2020). Increased oxidative stress leads to oxidative damage to essential cell-based compounds such as lipid compounds, protein molecules and hereditary material. (Mittal, Siddiqui, Tran, Reddy, & Malik, 2014). In this process, inflammatory enzymes such as neutral proteases, elastase, collagenase and acid hydrolases accelerate the formation of oxidative stress by releasing reactive species such as superoxide (O_2^-), hydrogen peroxide (H_2O_2), hydroxyl radical (OH^-), hypochlorous acid ($HOCl$), nitric oxide (NO) and extracellular messengers. The combination of these compounds exacerbates inflammation and muscle damage (Biswas, 2016). During muscle contraction, free radicals are produced by activation of endothelial xanthine oxidase, high catecholamine release, electron loss in the electron transport cycle, mitochondrial reaction and inflammatory response pathways. During exercise, free radical generation surpasses the antioxidant capacity of the muscle, usually during intense and demanding training that requires high power. Since the antioxidant system of the body is limited in the acute process, oxidant compounds produced in high amounts can cause damage to the cell membrane and oxidation of cellular components. (Powers & Jackson, 2008; Powers, Nelson, & Hudson, 2011). For instance, following exercise there is an increase in plasma levels of inflammatory determinants such as the acute phase reactants “C-reactive protein (CRP)” and “creatine kinase (CK)”, which often leads to muscle soreness (de Oliveira Teixeira et al., 2015; Righi et al., 2020). If this imbalance in the antioxidant system becomes permanent, it can lead to decreased muscle

power production, acute tiredness, poor sports performance, increased muscle injury, lack of regular exercise for beginners, and many other health problems (REID, 2001). At the same time, with continued exercise over the medium and long term, the capacity of the endogenous antioxidant system increases, resulting in adaptation and muscle remodelling (Righi et al., 2020).

Delayed Onset Muscle Soreness (DOMS) and Inflammation

Exercise is seen as a source of outside stressors similar to surgical intervention, shock or infection in that it causes significant physiological changes in the body by causing hormonal and immunological changes (Docherty et al., 2022).

Cytokines, which manage the immune system in case of wellness and illness, are included in the family of intracellular signalling molecules. Cytokines are divided into inflammation-promoting and inflammation-reducing groups, and the relationship among these two groups plays a crucial part in sustaining tissue balance. Disruption of both cytokine groups creates the potential for a negative process on other systems, especially on the immune system (Zhang & An, 2007). Pro-inflammatory cytokines include tumour necrosis factor- α (TNF- α) and interleukin 1 (IL-1). In contrast, anti-inflammatory cytokines consist of interleukin 1 receptor antagonist (IL-1ra), IL-4, IL-10, IL-11 and IL-13 (Dinarello, 2000; Umare et al., 2014).

Based on the research by Scheller and colleagues, IL-6 is identified as a cytokine that exhibits both inflammation-promoting and inflammation-reducing characteristics (Scheller, Chalaris, Schmidt-Arras, & Rose-John, 2011). It is suggested that one of the main mechanisms leading to DOMS formation is inflammation. At the beginning of the mechanisms causing inflammation is the process related to the increase and aggregation of histamines, neutrophils, bradykinins and prostaglandins in the tissue, which leads to the intensification of muscle pain. The triggering of IL-6 and TNF- α via Toll-like receptor 4 (TLR4) has been reported to be of critical importance in the formation mechanisms of DOMS (Dos Santos et al., 2020).

As a result of the exercise process, a process occurs in the cytokine profile that is thought to be effective on skeletal-muscle health (Steensberg et al., 2000). Exercise activates the main source of cytokines in the body, which is skeletal muscle. It has begun to attract attention as a secretory organ in muscles due to the cytokine secretion that occurs as a result of skeletal muscle contraction (Lightfoot & Cooper, 2016). Among the cytokines called myokines, which are produced by myocytes and exceed 3000 on average, there are cytokines such as IL-6, IL-7 and IL-15 (Piccirillo, 2019). Increased Tumor necrosis factor (TNF- α), raised to increased concentrations of CK and lactate dehydrogenase (LDH) in the serum in addition to the release of IL-1 β , IL-6 and IL-1, IL-1ra (SMITH, 1991). This regional inflammation results in DOMS, which is defined by increased muscle soreness, swelling and reduced muscle activity (Tan et al., 2022).

Tumor necrosis factor (TNF- α), a pro-inflammatory cytokine that is effective in inflammatory effects, has a critical structure in this environment. It has been shown that TNF- α not only initiates inflammatory gene expression, but also triggers inflammatory immune responses and disease process by indirectly inducing cell apoptosis, thus causing inflammatory responses (van Loo & Bertrand, 2023).

The main biomarkers involved in the DOMS mechanism were seen as TNF- α and IL-6. In particular, there is a linear relationship between high levels of TNF- α and inflammatory

activity of skeletal muscle (Dos Santos et al., 2020). Delayed onset myalgia (DOMS) triggers an inflammatory process, leading to raised concentrations of TNF- α in the blood, which indicates that muscle damage has occurred (Calder et al., 2009). After prolonged, irregular or intense exercise that can cause muscle damage, there is an rise in circulation immunocytes, resulting in an acute inflammatory Response (Smith, 1991). As a result of this inflammatory response, macrophages promote the mechanism of tissue breakdown through the release of inflammation-related cytokines, leading to an raised in C-reactive protein (CRP) concentration (MacIntyre, Reid, & McKenzie, 1995).

With damage to muscle tissue, there is an raised in the level of CK in the circulation. Elevated CK levels are considered a member of the major biochemical signs of myofibrillar damage and this increase is often directly proportional to the severity of the injury. Therefore, the level of damage to muscle tissue can be determined by measuring CK levels (Brancaccio, Maffulli, & Limongelli, 2007). The cytokine IL-6 plays a role in B and T cell differentiation, attracting neutrophils to the site of injury and regulating the inflammatory process in the innate and adaptive immune system (Scheller et al., 2011). The reaction of IL-6 to the exercise process has led it to be recognised as the most important cytokine in exercise physiology. Immediately after the onset of exercise, IL-6 levels increase approximately 100-fold and decline rapidly after the cessation of exercise (Steensberg et al., 2000). The rate of increase in interleukin-6 varies depending on many factors such as the strength of exercise, its duration and the person's exercise capacity (Febbraio & Pedersen, 2002). Pain in the muscles is often seen during the recovery period after vigorous exercise. Some studies have reported that this muscle soreness is due to IL-6 and TNF- α , which are inflammation-promoting cytokines released during and after exercise (Tejua, Purwantob, & Ayubic, 2022).

Cytokines are small soluble proteins that can control inflammatory and defense responses and alter the behaviour of other cells (Taherkhani, Suzuki, & Castell, 2020). Exercise can modulate the defense system and significantly inhibit the spread of pro-inflammatory cytokines and their permanent degradation. When damage occurs, the body responds by releasing catecholamines, which are stress hormones. These catecholamines bind to adrenergic receptors, stimulating a variety of cellular responses. This binding triggers the release of pro-inflammatory cytokines, including TNF- α and IL-1 β , which play a crucial role in the inflammatory process. These cytokines show a significant increase in the early stage of the inflammatory response. Pro-inflammatory cytokines like TNF- α and IL-1 have been demonstrated to regulate the inflammatory components of the body, facilitating the signaling of other anti-inflammatory cytokines such as IL-6 and IL-8. These cytokines are crucial for the defense system as well as for the health and healing processes, thus forming a complex network of communication that modulates the inflammatory response and tissue repair of the organism (Hartung, Ciszek, & Nackley, 2014).

The recruitment of additional leukocytes at the site of damage is assisted by raised in IL-6 and IL-8 concentrations. As the amount of leukocytes in the damaged area increases, interleukin-10 (IL-10) concentrations increase and IL-6 inhibits the expression of various inflammation-promoting cytokines (Ding et al., 2003). IL-10 inhibits the activity of immune cells and prevents tissue damage by reducing the production of other pro-inflammatory cytokines, thus supporting healing processes (Nagalakshmi, Murphy, McClanahan, & de Waal Malefyt, 2004). High levels of IL-10 help balance inflammatory

responses, reducing the extended range and therefore relieving pain. In this context, the anti-inflammatory properties of IL-10 play an important role in developments, contributing to the improvement of both maintenance and healing pain (FQ, 1999; Sluka, Frey-Law, & Bement, 2018). The inflammatory process that occurs following exercise is a complex physiological mechanism that plays a crucial role in tissue healing, muscle regeneration, and the adaptations required for effective functional recovery (Ota et al., 2013). Pain is a difficult and diverse activity that makes daily activities difficult to perform, one of the main goals is to break the pain. Therefore, the use of non-steroidal anti-inflammatory drugs to alleviate pain and support the healing process is a typical practice (Nahon, Lopes, Magalhães, Machado, & Cameron, 2021). It is a non-steroidal anti-inflammatory and acts by inhibiting the cyclooxygenase (COX) family enzymes. This inhibition leads to a decrease in the synthesis of events such as prostaglandins, prostacyclins and thromboxanes. This mechanism is essential for pain management and control (Schoenfeld, 2012).

Conclusion

In conclusion, the compounds such as lipid compounds, protein molecules and hereditary material. (Mittal, Siddiqui, Tran, Reddy, & Malik, 2014). In this process, inflammatory enzymes such as neutral proteases, elastase, collagenase and acid hydrolases accelerate the formation of oxidative stress by releasing reactive species such as superoxide (O_2^-), hydrogen peroxide (H_2O_2), hydroxyl radical ($OH^\cdot$), hypochlorous acid (HOCl), nitric oxide (NO) and extracellular messengers. This mechanism is essential for pain management and control after exercise (Schoenfeld, 2012).

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