Muscle Soreness: Applying What We Know

By Kayla Schatz and Graduate Student Mentor, Eric Leslie

Introduction

Feeling sore after a long, grueling workout can result in a feeling of satisfaction. Alternatively, the absence of soreness after a workout may feel as though no progress was made. The phrase "no pain, no gain" is often heard in the world of fitness and it may have had more of an impact than we think. People who consistently perform resistance training use delayed-onset muscle soreness (DOMs) as an indicator of the effectiveness of their training program (1). But this may not be the best way to gauge an effective workout. This review will explore how the attitudes of muscle soreness have changed, the physiology behind muscle soreness, the relationship between muscle soreness and progress (or lack thereof) and the possible treatments and remedies to combat muscle soreness.

The Lactate Myth

For decades, lactate was believed to be the primary cause of delayed onset muscle soreness (DOMS). DOMS is a type of muscle pain that occurs around 24 to 48 hours after exercise. This is different from acute muscle soreness, which occurs immediately after exercise. Lactic acid, or lactate, is a byproduct of a process called anaerobic respiration. Anaerobic respiration is the steps that cells must take to produce energy without oxygen. Lactate was believed to be a metabolic waste product that would lead to an impairment in athletic performance. The man who made this connection, Otto Meyerhof, won a Noble Prize for Physiology or Medicine in 1926 for proposing that lactate was the cause of muscle soreness. In his experiment, he placed a dead frog's legs in a jar and shocked them with electricity to cause the muscles to contract. Once the muscles stopped contracting, Meyerhof dissected the legs and found that they were coated in lactate. Meyerhof's thought process was as follows: since the muscles had no circulation, no oxygen source, and had converted glycogen into lactate, then the reason the muscles could not contract is because they were too acidic. This ultimately leads to fatigue in the muscle. Unfortunately for Meyerhof, this theory turned out to be false.

Dr. Meyerhof's research gave scientists a great starting point in determining the real cause of muscle soreness. Many studies have been conducted that have proved Dr. Meyerhof false. Most notably, a study was done back in 1983 that tested the production of lactic acid before, during, immediately after, and 72 hours after 45 minutes of treadmill running. Subjects

were instructed to run for 45 minutes on a treadmill, either at a decline, a 10% incline, or no decline or incline. Lactate levels never accumulated in the downhill runners, despite them reporting a significant perception of muscle soreness. Those who ran with no incline showed a high lactate concentration but did not report significant muscle soreness and for those who were running at an incline had very high lactate levels but no reported muscle soreness. This study concluded that there is not a connection between lactate levels and delayed-onset muscle soreness (DOMS). The lack of relationship between muscle soreness and lactate levels caused researchers to explore other possible explanations for the cause of muscle soreness after exercise.

So What is the Cause?

Currently, there are several hypothesized theories that have been proposed for the mechanisms behind delayed-onset muscle soreness (DOMS). Each theory involves slightly different physiology. It is very possible that the explanation for DOMS may be an integration of multiple theories. Cheung (2) explains six theories in depth. A few theories that will be highlighted are the inflammation theory, the muscle damage theory, and the connective-tissue theory.

The inflammation theory (2) is based on the inflammatory response that is present after repetitive movements. After a bout of eccentric muscle action, protein and lipid structures in cells deteriorate. This deterioration is initiated by proteolytic enzymes within the muscle fibers. The breakdown of damaged muscle fibers causes monocytes and neutrophils to migrate to the site of the injury. The accumulation of these monocytes and neutrophils create a sensitivity in both the type III and IV nerve endings. It takes around 24-48 hours for the accumulation to occur. This theory provides a possible explanation as to why muscle soreness is delayed.

This next theory explores the components of the sarcomere, the functional unit of skeletal muscle. The muscle damage theory (2) highlights the contractile component of muscle tissue, specifically z-lines, which mark the borders of the sarcomeres. During the lengthening phase of a muscle contraction, myofilaments are stretched apart. These myofilaments return to their normal function during the relaxation phase of a muscle contraction. However, some myofilaments become overstretched during eccentric actions. Repeated eccentric contractions increase the number of disrupted myofilaments. This disrupts overall construction of the sarcomere. This damage is also a result of the smaller amount of motor units that are active during an eccentric action. This disruption allows the diffusion of certain enzymes, such as creatine kinase (CK) into

the interstitial fluid. CK can show the level of muscle membrane permeability. At resting levels, CK is approximately 100 IU/L. After eccentric exercise, CK levels can reach 40,000 UI/L (3). There is a correlation between peak muscle soreness and peak serum CK levels.

The connective-tissue damage theory (2) also looks at the effects of muscle strain at the cellular level. This theory explores the role of connective tissues that surround bundles of muscle fibers. The type of muscle fibers, such as type I and type II, determines the composition of connective tissue. Type I muscle fibers are tougher than type II muscle fibers, they contain a higher number of capillaries and mitochondria, making them more robust. Type II muscles are relatively faster and stronger. This puts them at a greater risk of injury because they are the dominant fiber type in intense exercise. This strain of the connective tissue could explain the feeling of soreness in the muscle.

Does Soreness = Progress?

Those who participate in resistance training may have been told to regularly switch up their routines in order to see continued strength gains. Adding and adjusting certain exercises often result in muscle soreness in the days following. This is because the muscles are not adapted to new exercises. This can cause strain to the connective tissues or a microscopic injury within the sarcomere. It is easy to associate soreness with progress for this reason. Shoenfeld and Contreras (4) provide a theoretical basis for using muscle soreness as a gauge for muscular adaptions. They begin by establishing whether this theory has any biological plausibility. It's clear that there is correlation between exercise-induced muscle damage (EIMD) and DOMS. EIMD is associated with structural changes that work to prevent further injury by strengthening tissues.

Shoenfeld and Contreras (4) hypothesize that acute inflammatory response to muscle damage from exercise can cause muscle hypertrophy. Macrophages, specialized cells that can initiate inflammation, promote rebuilding post-damaging exercise. Macrophages do this through the secretion of cytokines, which signal molecules that can signal for anabolic mechanisms that relate to muscle growth. It's believed that these phagocytic cells are essential for muscle growth because they recruit other cells which are necessary for regenerating muscle (5). Neutrophils may also play a similar role in muscle regeneration, but through reactive oxygen species (ROS). It's suggested that ROS can produce hypertrophic effects on skeletal muscle (6). Hypertrophy may be achieved through muscle damage by activating satellite cells, which are stem cells in the

muscle. When muscle is exposed to any mechanical stress, they migrate to other areas and fuse to existing myofibers or produce new fibers. This provides the ingredients needed to remodel the muscle tissue. The activity in these satellite cells is increased in response to EIMD. However, these satellite cells are also responsive to exercise that is non-damaging (7).

Although it's enticing to create a link between DOMS and hypertrophy because research has shown a correlation between EIMD and DOMs, there is still evidence that suggests otherwise. In a study from Yu et. al. (8), subjects performed different eccentric exercises and developed DOMS. It was found that even if a subject had developed DOMS, there was no evidence of inflammatory markers. In addition, studies have found that there are no hypertrophic adaptations associated with soreness after a long-duration event, such as a marathon (8).

The presence of DOMS can be affected by the training status of an individual. If an athlete repeats the same bouts of exercise, soreness tends to dissipate with time. This is because the muscles adapt to exercises in order to better prepare themselves for the next bout of exercise. This now non-damaging exercise still activates satellite cells and promotes tissue remodeling. Frequently training a muscle group with the same exercises results in less soreness, but could still produce hypertrophy. A study from Sikorski et al. (9) found that some muscles do not experience the same degree of soreness as others. There may be several explanations as to why this occurs such as connective tissues being strengthened, or the muscle has adapted to an increased efficiency in recruiting motor fibers.

Just because there is evidence that DOMS can promote muscle growth and hypertrophy, does not mean that it should be actively pursued. DOMS can prevent athletes from meeting goals during a training season due to pain. It can also disrupt the entire training cycle for this same reason. Luckily, there are ways to ease DOMS internally with nutrition and externally with a variety of treatments.

How Can You Treat Muscle Soreness?

Hot and cold treatments have typically been prescribed for muscle soreness by altering blood flow and increasing flexibility. Petrofsky et al. (10) conducted a study about the efficacy of sustained heat treatment for easing DOMs in large muscle groups. In this study, the participants were asked to perform back squats in three 5-minute bouts, performed at a set pace of 3 seconds per squat for 5 minutes in order to cause the onset of DOMS, The participants were divided into two groups. The first group was given a ThermaCare heat wrap to wrap on each leg

immediately after exercise. The second group was not given the heat wrap until a full 24 hours had passed after exercise. The results of this study showed a reduction in soreness in both groups after utilizing the heat wraps. However, the most significant benefits were found in the group who had applied the heat wraps immediately after exercise. By increasing flexibility and blood flow of the tissue, heat decreases muscle damage and the effects of DOMs.

In addition to treating muscle soreness externally, it may also be possible to reduce DOMS internally with nutritional interventions. In a review article by Meamarbashi (11), multiple herbs and natural supplements were highlighted for their prevention and treatment of delayed-onset muscle soreness. Meambarbashi discusses caffeine's role in the reduction of DOMs. Caffeine can extend time to fatigue and increase alertness by blocking the adenosine receptors. But how does it prevent soreness? Caffeine has a pharmacological effect by blocking specific adenosine receptors. Adenosine is responsible for activating pain receptors within the cells. By blocking these receptors, there is a decreased perception of pain, making DOMs less debilitating.

A study from Mashhadi et al. (12) explored the influence of both ginger and cinnamon intake on muscle soreness after exercise. Both ginger and cinnamon have been found to contain anti-inflammatory components. Sixty female martial athletes were enrolled in this study. These participants were randomly divided into three groups; cinnamon, ginger, or placebo. These women consumed 3 grams of their assigned powders daily for six weeks while completing their existing training programs for their competitive season. The participants were asked to report their level of muscle soreness according to the Likert Scale of Muscle Soreness in 24-hour intervals post-exercise after specific resistance exercises within their training programs. The cinnamon group reported a significant reduction in muscle soreness compared to the placebo group. The ginger group also reported a significant reduction in muscle soreness compared to the placebo group. Incorporating ginger and/or cinnamon into your diet during periods of intense exercise can be helpful in preventing the development of muscle soreness.

Conclusion

Everyone has experienced the uncomfortable and sometimes painful effects of muscle soreness. Athletes are always dealing with the returning effects of muscle soreness following new training protocol. Although muscle soreness isn't pleasant, it can be one of many indicators of progress. We know that lactate acid is not responsible for muscle soreness, although it was a

previously accepted explanation. We have new and exciting theories that can help us understand why we feel sore after a grueling workout. There may be a connection between muscle soreness and hypertrophy and strength improvements. But this is situational. And lastly, there are ways to both combat and prevent DOMs.

Apply it:

- Lactate is not a harmful byproduct of exercise. You don't need to "flush it out" by stretching or taking a hot bath. Instead, applying a hot compress to the sore muscles will increase blood flow and decrease DOMs.
- The mechanisms behind DOMs may be an integration of several theories. Creating an exercise program based on one theory may limit productivity. Focus instead on achieving hypertrophy and meeting personal goals.
- Muscle tissue will experience remodeling regardless of whether or not DOMs is present post-exercise. The presence of muscle soreness should not be used as the tell-all indicator of progress.
- Treating DOMs from the inside out may be the best way to combat pain. Adding a nutritional component such as ginger or cinnamon to your diet may help you avoid muscle soreness following an intense workout.

Bridging the gap:

- Lactate is not the cause of delayed-onset muscle soreness.
- The inflammation theory gives us insight as to what inflammation does to the muscle. The muscle damage theory incorporates creatine kinase into the discussion. And the connective-tissue theory looks at the strain of the muscle in depth. It's likely that a combination of these theories provide the explanation that we are searching for, with lactate not playing a significant role in any of them.
- Adding anti-inflammatory foods, such as cinnamon and ginger, can reduce the effects of muscle soreness.

Summary Statement: New theories such as the inflammation theory, the muscle damage theory, and the connective-tissue theory have replaced lactate as the proposed mechanisms behind muscle soreness. These theories can be applied to better understand the relationship between muscle soreness and progress and, furthermore, determining the best treatments for reducing muscle soreness post-exercise.

Pulled text:

- “Hypertrophy may be achieved through muscle damage by activating satellite cells, which are stem cells in the muscle. When muscle is exposed to any mechanical stress, they migrate to other areas and fuse to existing myofibers or produce new fibers. This provides the ingredients needed to remodel the muscle tissue.”

Bio:

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