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The Physiological and Biomechanical Causes of Delayed Onset Muscle Soreness and Subsequent Methods of Intervention

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THE PHYSIOLOGICAL AND BIOMECHANICAL CAUSES OF DELAYED
ONSET MUSCLE SORENESS AND SUBSEQUENT METHODS OF
INTERVENTION

by

Jonathan Stevens
Bachelor of Science in Physical Therapy
University of North Dakota, 1997

An Independent Study
Submitted to the Graduate Faculty of the
Department of Physical Therapy
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in partial fulfillment of the requirements
for the degree of
Master of Physical Therapy

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This Independent Study, submitted by Jonathan H. Stevens in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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ABSTRACT

Delayed Onset Muscle Soreness (DOMS) is a common problem affecting many people with active lifestyles - from teenagers all the way to the elderly. DOMS is soreness arising from strenuous activity, and peaking in intensity 24-48 hours after the cessation of the activity. DOMS is commonly seen in the clinic and can often hinder a patient’s progress in therapy.

It is well documented in the literature that eccentric exercises promote an increase in the frequency and intensity of DOMS, however, research regarding the metabolic and structural changes of the affected muscles and subsequent effective preventative and treatment strategies are widely scattered. Thus most therapists do not have the time or the resources to track down all the relevant literature.

My objective with this study is to provide a concise and organized reference for therapists in order to assist them and their patients in avoiding the occurrence of DOMS. Also, this paper will provide therapists with information as to the causes and treatment of DOMS so that they may minimize the soreness and functional deficits in their respective patients in cases when DOMS does occur.
CHAPTER 1

INTRODUCTION: REVIEW OF MUSCLE PHYSIOLOGY

Delayed onset muscle soreness (DOMS) is a common problem among people with active lifestyles. Most of us have experienced DOMS in varying degrees, and can readily identify with the debilitating effects it can have upon a person’s athletic endeavors, as well as activities of daily living. Therefore, when a patient exhibits increased muscle soreness it is not surprising that they are less likely to apply themselves as well during therapy for fear of bringing about more pain.

There are two types of exercise induced soreness: acute muscle soreness, which occurs during and immediately following exercise due to ischemia of the active muscles, and DOMS, which is defined as the muscle soreness occurring 24 – 48 hours after the cessation of exercise. DOMS can affect anyone from an geriatric patient to a world class athlete. Through the course of this paper, I will examine the various theories given as to the causes of DOMS, physiological and structural changes of the muscle due to DOMS, exercises that promote increased frequency and intensity of soreness, as well as discussing various techniques to prevent and treat DOMS. The purpose of this paper is to provide physical therapists with a concise reference as to the mechanism and effects of DOMS, methods to treat DOMS, in addition to techniques to aid in the prevention of DOMS.
Each of our 430 skeletal muscles is an organ, composed of many important, interconnecting parts and containing muscle tissue, connective tissue, nerves, and blood vessels. Epimysium is a fibrous connective tissue that surrounds the entire muscle and is continuous with the tendons at the ends of the muscle. Underneath the epimysium, muscle fibers are grouped into bundles called fasciculi which are surrounded by connective tissue called perimysium. The muscle fibers that compose the fasciculi are in turn ensheathed by the endomysium. All three of the connective tissue layers are continuous with the tendon, so tension developed in one muscle cell will develop tension in the tendon.

The inside of the muscle fiber is the sarcoplasm, which contains the contractile components of the muscle, other proteins, stored glycogen and fat particles, enzymes, and specialized structures such as mitochondria and sarcoplasmic reticulum. The sarcoplasmic reticulum serves as a storage area for calcium ions. When an action potential reaches the plasma membrane (sarcolemma), it is carried into the t-tubule system which connects the sarcolemma to the interior of the cell. As the action potential arrives at the cell interior it causes the release of calcium ions from the sarcoplasmic reticulum into the sarcoplasm. Hundreds to thousands of myofibrils dominate the sarcoplasm, each containing the contractile components myosin and actin. In the presence of calcium ions, the myosin heads bind to the active binding sites on the actin molecule.

The most widely accepted theory as to the mechanism of skeletal muscle contraction is the sliding filament theory. According to this theory, after myosin binds to actin the myosin head moves pulling the actin towards the center. The
myosin head then detaches and moves in a ratchet fashion to a new binding site on the actin and once again pulls the actin, thus further shortening the sarcomere. This process is repeated so long as calcium ions are present.

Skeletal muscles are divided into two main types: Type I and Type II. Type I fibers are also known as slow twitch, oxidative, or red muscle fibers. Type II fibers are fast twitch, anaerobic, and white muscle fibers. The type I fibers are active during aerobic training and endurance events, while type II fibers are better equipped for short, high intensity anaerobic exercise such as sprinting. The basic energy source needed for all muscle contractions is adenosine triphosphate (ATP). Since the functions of each fiber type differ, it stands to reason that the energy source (ATP) must be provided by different methods to accommodate the needs of the particular muscle fiber.

There are three main energy systems through which ATP is used by the contracting muscle. The first system is the ATP-PC system in which phosphocreatine and ATP are stored in the muscle cell and available for immediate use, no oxygen is required, and the power of the system is significant (3.7 moles of ATP per minute). Due to the absence of oxygen and small amounts of ATP available, this system provides energy for quick bursts of activity (anaerobic – type II fibers) and thus is the major fuel source during the first 30 seconds of activity. The second system is the anaerobic glycolytic system. This system uses glycogen as the food fuel source, and like the ATP-PC system, no oxygen is required. Thus it is used for moderate intensity and short duration events and is the major energy source from the 30th to 90th seconds of exercise. Lactic acid is produced in this system. The
aerobic system (aerobic glycolysis) uses glycogen, fats, and proteins as the food fuel sources and requires oxygen. ATP is resynthesized in the mitochondria of the muscle cell. The ability to metabolize oxygen is related to the concentration of mitochondria. This is the energy system of choice after the second minute of continuous exercise.

How does the body recover after an exercise bout? In the case of the first two systems, the body needs to replenish its oxygen debt – the amount of oxygen needed during recovery above that which would have been consumed at rest. This oxygen debt is used to replenish ATP, PC, restore blood hemoglobin and muscle myoglobin levels, replenish O₂ levels, and supply oxygen to the heart and lungs which are working hard during recovery. During submaximal exercise (aerobic energy system), the presence of oxygen prevents the conversion of pyruvic acid to lactic acid, thus, there is little lactic acid buildup and recovery time is shortened. However, ATP, PC, and blood and tissue levels of O₂ still need to be replenished. Hence, passive recovery is best after an aerobic workout.

In cases of intense exercise of short duration (ATP-PC system and anaerobic glycolysis), lactic acid accumulates and must be removed along with rebuilding the ATP, PC, and O₂ levels. The removal of lactic acid takes much longer since oxygen, which is already depleted, needs to be present.

Before we continue, the mechanism of acute muscle soreness must first be discussed briefly so as to not confuse acute soreness with DOMS. As mentioned earlier, acute soreness results from ischemia of the working muscles. In the case of short duration, high intensity exercise, this ischemia is caused by lactic acid
accumulation after the anaerobic threshold has been reached. The anaerobic threshold is the point where lactic acid starts to build up – normally around 50% of a person’s maximal volume of oxygen consumption (MVO₂). Therefore, the better trained the athlete (higher MVO₂), the more anaerobic exercise they can perform before they go into oxygen debt (lactate accumulates). In cases of submaximal (aerobic) exercise, the cause of muscle fatigue and subsequent acute soreness appears to be glycogen depletion.
CHAPTER 2
PHYSIOLOGICAL CAUSES AND STRUCTURAL CHANGES OBSERVED IN DOMS

DOMS is soreness that occurs after strenuous exercise, peaking in intensity 24-48 hours after the exercise bout, with soreness resolved 5 - 7 days post exercise.¹ The soreness has been reported to be the most evident at the musculotendonous junction initially.⁴ Generally, more severe cases of DOMS result in the spreading of the pain to the center (belly) of the muscle during days two and three, as has been found in this study⁴ involving the gastrocnemius and quadriceps. However, MacIntyre⁴ concluded that these are currently the only muscles studied with documentation of pain behavior so we can’t definitively apply this rule to all muscles.

It has been well documented that eccentric exercise causes the most frequent and intense cases of DOMS.⁵,⁶ There are two main schools of thought as to why this occurs. First, eccentric exercise requires lower energy costs than concentric exercise since fewer motor units are needed for a given load, thus resulting in higher tensions per cross-sectional area of muscle fibers. Secondly, researchers⁷ have proposed that the strain during active lengthening of the muscle also contributes to Z-band damage by exceeding the limits of the cytoskeletal framework. Furthermore, the force developed during eccentric activity is approximately twice that of isometric
contractions, however the total number of crossbridges is only about 10% greater during eccentric work.\textsuperscript{8}

Interestingly, the mechanism of injury to the muscle depends on the type of activity (aerobic/anaerobic) that was overloaded. For example, Appell et al\textsuperscript{5} found that damage incurred by the vastus lateralis of endurance training subjects was observed primarily in those fibers that were also severely glycogen depleted, suggesting a metabolic etiology for the breakdown. On the other hand, subjects receiving eccentric power training demonstrated myofibrillar damage immediately after exercise as well as excessive structural damage in areas that were not glycogen depleted, suggesting the existence of a mechanical etiology. In other words, the damage seen in the endurance group stems from an ischemic rather than a mechanical origin due to the unaccustomed aerobic demands placed on the muscle. This ischemic state results in an autophagic, inflammatory response in which neutrophils, as well as macrophages and other inflammatory cells are needed to clear away damaged debris and begin the rebuilding and regenerating process.\textsuperscript{9}

Overloading the muscle with either aerobic or anaerobic activity can cause mild to severe structural changes in the respective muscle(s).\textsuperscript{9} The two types of exercise promote muscle damage in different manners, yet they have similar outcomes on the structural changes and repair of the muscle. According to the study by Appell,\textsuperscript{5} structural changes seen after strenuous endurance exercise included early development of edema followed by disarray of myofilaments, occurrence of necrotic fibers and invasion of inflammatory cells. A case study described by Sjostrom\textsuperscript{10} involved a man who ran the equivalent of 1.7 marathons per day for seven weeks.
Biopsies were taken from the vastus lateralis muscle one month before and immediately after the training period. The muscle was invaded with inflammatory cells and an increased amount of connective tissue was found. In addition, necrotic fibers, phagocytosed fibers, and regenerated fibers were all found. This example shows the ability of skeletal muscle to regenerate through the action of neutrophils and other phagocytic cells.

Having discussed the physiologically damaging effects seen in muscle following strenuous exercise, what types of exercise cause these deleterious effects? In studies involving soreness induced by eccentric activity, histological examinations revealed focal disruption of the banding pattern (Z bands primarily) of the muscle fibers, with subsequent fiber necrosis and regeneration. More specifically, Fielding studied nine male volunteers who ran downhill on a 16% decline for three sets of 15 minutes with five minutes rest between sets. This protocol had both aerobic and anaerobic components. Upon histological examination, electron microscopy revealed an increase in the ratio of damaged to undamaged Z-bands (p < .01). This type of quantitative data on Z-band disruption is the first and only of its kind presently and represents a big step forward from past articles that just stated that there was structural damage.

Furthermore, DOMS resulting from powerful eccentric exercise has been found to affect type II fibers the most, while these are the least glycogen depleted. The preferential tearing of the type II fibers may be explained by the fact that their cytoskeleton is less developed and they have the narrowest Z-band of all fiber types. This preferential tearing of type II fibers can explain why researchers have
always found a loss of muscle strength on days two and three of DOMS, but the fatigability of the muscle has not changed, and in some cases has actually improved. While all of the preceding studies have consisted of isotonic, isometric, or running activities, at least one study was performed analyzing soreness levels in response to isokinetic quadriceps exercise. In this experiment, the authors postulated that, if exercised at the same power level, there would be no difference in pain levels between the eccentric or concentric groups. As suspected, the groups that worked at the same power levels and intensity showed no difference in DOMS values between the concentrically and eccentrically exercised groups; however, the other two groups (one performed eccentric contractions, the other concentric) that trained simply with maximal effort showed the usual results of increased perception of pain in the eccentrically exercised group.

Physiological Causes

The causes of DOMS, specifically the perception of the pain component, have been much debated. The most widely accepted theories are the micro muscle and connective tissue tears and the inflammatory (biochemical) response. First of all, however, I'd like to discuss some past theories that no longer are looked upon as being valid.

As far back as 1956, Asmussen theorized that the pain associated with DOMS was brought about by metabolic waste products, namely lactic acid. Appell noted that eccentric contractions require less energy expenditure than concentric (due to fewer motor units recruited), thus for a given resistance eccentric work requires lower oxygen consumption and hence produces less lactate than concentric work, thus
eccentric exercises should produce less pain. It has also been found that lactic acid is removed (as stated in the introduction) from the muscle long before the peak levels of pain seen two to three days post exercise.

Another theory as to the cause of soreness seen in DOMS is serum creatine kinase (CK) levels. CK has been shown to increase in response to unaccustomed work, particularly eccentric, as well as showing a reduction in the increase after a period of training. Biopsies are used to identify damaged muscle tissue, as well as chemical indicators such as CK. CK, as well as myoglobin, has long been used as an indicator of people who have recently had a myocardial infarct. As a result, researchers have looked into the possibility that the elevated levels of CK may not be only an indicator of skeletal muscle damage, but may also be at least partially responsible for DOMS. In a study of subjects performing a downhill walk, Balnave and Thompson found that the CK levels peaked 24 hours post exercise. In addition, after repeated bouts of exercise over the next couple weeks, the increase in CK levels diminished (as did the DOMS values), exhibiting adaptation to the increased load. However, in two studies involving eccentric forearm flexor contractions, the authors found much higher elevations in CK and with a later peak value (5 days post exercise). The CK argument has been refuted on the basis that subjects with minimal CK increases have reported as great, if not greater, levels of DOMS (using the visual analogue scale) than those with huge increases in CK. Furthermore, elevated CK has not been shown to correlate well even with objective measures of muscle damage, such as biopsies analyzing the extent of Z-band streaming. Thus CK levels are an
above average indicator of skeletal muscle damage, but can be useful in cases where a biopsy is not an option.9

Researchers17 have also dealt with intramuscular pressure (IMP) as the causative factor of DOMS, or at least an indicator of the high muscle tensions needed to inflict DOMS. IMP is the fluid pressure created by a muscle during contraction and has been shown to correlate linearly with the force of contraction during isometric and isokinetic exercise. Since IMP has been shown to increase with increased muscle tension, it was thought that IMP could be a possible indicator or cause of DOMS. In this study by Crenshaw et al17 eight male subjects performed eccentric quadriceps contractions on one leg and concentric on the other leg. Basically the authors found no significant difference in peak IMP between concentric and eccentric legs; however, IMP levels did tend to be slightly higher in the concentric leg. Thus, they also found the concentric leg to have the higher IMP to peak torque coefficient (secondary to concentric leg being unable to generate as much torque coupled with slightly higher IMP). The authors concluded that the higher knee extension torques did not result in elevated IMP values, hence IMP is neither a reliable indicator nor a cause of DOMS.

One of the original theories dealing with muscle fiber necrosis and regeneration proposed that micro muscle tears (specifically the Z-band and other components of the cytoskeleton) are responsible for DOMS, as well as the adaptation that muscles undergo in response to consistent training.18 The authors proposed that a pool of stress susceptible fibers exists that are more fragile than other fibers and so develop a lethal injury when they are put under unusual stress eccentrically.
Therefore, the number of weaker fibers (those towards the end of their lifespan) is diminished and the stronger fibers survive. Furthermore, up until the last couple of decades, it was in doubt whether or not muscle fibers were even capable of regeneration.\textsuperscript{4} While overload is obviously important to a certain point, it has been shown that less intense eccentric exercise protocols, which do not lead to an elevated CK, are still sufficient to bring about a reduction in the CK response over time, demonstrating adaptation of the muscle.\textsuperscript{6}

In cases of strenuous endurance exercise, an autophagic response is initiated, during which neutrophils migrate to the site of injury and assist macrophages in the removal and repair of necrotic tissue.\textsuperscript{19} Also, Smith\textsuperscript{19} found that neutrophilia occurred to a greater extent after eccentric than concentric exercises when performed by the same group of subjects at the same level of oxygen consumption. Fielding’s\textsuperscript{9} results showed extensive Z-band damage that correlated with high neutrophil counts based directly on muscle biopsy specimens, with significant accumulation of neutrophils 45 minutes after the exercise and lasting until five days post exercise. Based on this and other similar studies, several authors\textsuperscript{4,9,13} have proposed that the disturbed muscle fragments and released intracellular material may then form a chemotactic gradient that attracts neutrophils. This in turn may contribute to a delayed metabolic response in the muscle after exercise before the clearance of the damaged material occurs, resulting in pain as seen with any tissue inflammatory process.

The current school of thought as to the production of the symptoms seen with DOMS has to do with a combination of mechanical and biochemical factors. In terms
of mechanical factors, Stauber et al\textsuperscript{20} have suggested that tissue swelling and
disruption of the extracellular matrix as a result of the autophagic response may be
more important in the production of pain than the torn muscle fibers. Furthermore,
tissue swelling and connective tissue damage have been implicated as the probable
causes for muscle "stiffness" -- the resistance to stretch commonly seen in DOMS.\textsuperscript{4}

From the biochemical point of view, the arguments are much more complex.
Basically, mast cell degranulation could lead to the production of histamine, and
kinins may also be released, both of which would result in pain.\textsuperscript{4} Smith\textsuperscript{21} proposed
that the most likely pain source, however, seems to be prostaglandin E2 (PGE2),
which can be synthesized by macrophages resulting in increased sensitivity of pain
receptors. Furthermore, during tissue repair, fibrinogen is converted to fibrin which
also activates prostaglandins. Taking into account the aforementioned mechanical
and biochemical factors, coupled with the function of PGE2, the afferent nerves may
become more sensitive to mechanical stimuli (such as swelling and stretch) causing
delayed muscle soreness.\textsuperscript{4}

According to Armstrong,\textsuperscript{22} a cascade of events results in DOMS. High
eccentric forces cause disruption of muscle fibers and connective tissue, which leads
to an influx of calcium into the fiber (mitochondria) where it inhibits cellular
respiration. The progressive deterioration of the sarcolemma would attract
neutrophils to the area. Further accumulation of histamine and kinins in the
interstitium resulting from cellular necrosis as well as increased pressure from edema
could then activate the nociceptors and result in the sensation of DOMS.
CHAPTER 3

RELIABLE INDICATORS AND EFFECTS OF DOMS

Due to the fact that DOMS is a subjective measurement, researchers have had difficulty finding one measure (such as CK or myoglobin) that shows a strong correlation to DOMS values. CK and myoglobin (Mb) levels, both physiologic effects of DOMS, have been used as indicators of MI’s for years, thus they should also be good indicators of skeletal muscle damage. Balnave and Thompson tried to analyze other effects of eccentric exercise (DOMS). Specifically, they attempted to show a correlation between functional, as well as biochemical (CK and Mb) outcomes, and DOMS. The functional outcomes measured were maximal force (MVC) and flexion/extension angle. However, they ran into the same problems that the previous studies did using CK and Mb as indicators, namely the subjective nature of DOMS values leading to poor correlation statistics.

Subsequently, Rodenburg then conducted a study using the same functional measures outlined above as well as the traditional biochemical indicators (CK and Mb). However, his study was different in that he examined the correlation between each of these indicators as well as each of the indicators’ correlation to the DOMS values. He had 27 male subjects perform eccentric only contractions of the elbow flexors for 30 minutes (4 contractions per minute), with measurements taken at 1, 24, 48, 72, and 96 hours post exercise. He subsequently found significant positive
correlation between Mb and CK from 24-72 hours post exercise, a significant negative correlation between MVC and flexion angle at 1-24 hours post, as well as a significant negative correlation between flexion and extension angles throughout all time periods. Thus, Rodenburg\textsuperscript{24} concludes, of all the effects of DOMS, no one factor can be labeled as the sole indicator since they all play important outcome roles.

Rodenburg’s\textsuperscript{24} experiment mentioned above failed to show a significant correlation between extension angle of the elbow and DOMS values due to their different time courses and paths. However, the peak extension angle deficit occurred at the same point in time (48 hours) as the peak DOMS value. Furthermore, the extension angle then began its return to normal as the DOMS scores also diminished.

Regarding MVC, MacIntyre\textsuperscript{4} found that the loss of force has continued up to one hour post exercise. Newham\textsuperscript{25} reported an attainment of pre exercise strength levels within 24 hours for his subjects after they had performed a step test to induce soreness. Whereas Rodenburg\textsuperscript{24} has reported that it takes as long as one week to attain pre soreness levels of eccentric strength. However, Balnave\textsuperscript{6} conducted an aerobic activity to induce soreness, and DOMS levels associated with aerobic activity have shown the tendency to be eradicated after only two aerobic exercise bouts.

A biphasic decline in muscle strength has been shown to be the path that the MVC follows after anaerobic overload.\textsuperscript{4} Biphasic decline means that there is a decline in strength immediately after exercise till 1-2 hours post exercise, accompanied by a second decline 24 hours later. The initial decrease in strength is due to neuromuscular fatigue, the early changes shown in morphological studies, and possibly proprioceptive impairment.\textsuperscript{24} The second decline is due to DOMS,
morphological damage to the structural proteins of the muscle, and proprioceptive impairment. This biphasic decline in strength also supports the notion that there are many factors at work causing, and resulting from, DOMS -- each with a specific time course.
CHAPTER 4
BENEFITS OF ECCENTRIC EXERCISE

In relation to training just the concentric portion of the contraction, research has found that not only is it impractical but that higher increases in isotonic strength have been found to occur when both movements are used as part of an exercise, as opposed to using either movement on its own.\textsuperscript{1} Furthermore, as joint angular velocity increases, maximal eccentric torque also increases until approximately 90 degrees per second, after which it declines gradually. This is in contrast to the classic force tension curve which showed a decrease in force production as the velocity of concentric contraction increased.\textsuperscript{2} This physiologic advantage of the eccentric portion is valuable in the clinic when therapy is geared towards sport specific power training where increased speed of contraction is needed, as seen in plyometrics.\textsuperscript{26}

Also, since an eccentric contraction requires the recruitment of fewer muscle fibers for a given load, it is more energy efficient than concentric actions, as shown through EMG studies.\textsuperscript{1} Therefore, eccentric exercises are very useful as the initial exercise in cases of extreme weakness. Functionally, eccentric strength and control has been implicated as a major determinant in various tendinitis cases, as tendinitis is thought to develop when eccentric control of the particular muscle(s) is lacking.\textsuperscript{26}

Lastly, eccentric muscle actions have also been thought to promote greater increases in muscular strength and hypertrophy than concentric actions.\textsuperscript{27} In
Hortobagyi’s study involving 15 male subjects who participated in two, twelve week training sessions, seven subjects trained the quadriceps eccentrically and eight trained them concentrically. He found that the eccentric training increased eccentric strength 3.5 times more than concentric training increased concentric strength. Also, type II muscle fiber area increased approximately 10 times more in the eccentric group than in the concentric group.

One characteristic of aging is a decrease in a person’s fat free mass, which declines by about 15% from the third to eighth decades of life, contributing to the lower basal metabolic rate seen in older people. Also, reduced muscle strength in the elderly is a major cause for their increased prevalence of disability. Data also suggests that with increasing age and decreased activity levels, walking ability is impaired. A study by Whipple on institutionalized elderly found a lower incidence of falls in those who were undergoing a regular strengthening program. An increase in activity among the elderly, especially strength training, could also help chronic problems such as poor bone density and osteoporosis, as well as insulin dependancy. For the healthy elderly, Evans found a 107% increase in quadriceps strength and a 227% increase in hamstring strength after he employed a high intensity (80% of 1 RM) training protocol for 12 weeks, as opposed to other studies that showed improvement but at lower intensity levels (60% of 1 RM).
CHAPTER 5
TREATMENT OF DOMS

To test the ability of massage to increase blood flow, Tiidus\textsuperscript{30} performed a study in which subjects completed eccentric quadriceps exercises bilaterally on four consecutive days, each workout being followed by a 20 minute massage session to one of the legs. Using pulsed Doppler ultrasound velocimetry, Tiidus\textsuperscript{30} found that massage did not significantly elevate arterial or venous blood flow above resting levels, whereas light quadriceps contractions did. Furthermore, he found that the massage group had no significantly quicker recovery of peak muscle torque over the course of the four days as compared to the control group, but they did have less DOMS.

A study by Weber\textsuperscript{31} involved eccentric activity of the elbow flexors, in which one group received effleurage for 2 minutes, petrissage for 5 minutes, and effleurage for 1 minute. He found that the massage group had no significant decrease in DOMS, nor did they experience a quicker return to pre exercise muscle torque values. However, in another experiment, Smith\textsuperscript{32} performed a 30 minute sports massage (a combination of effleurage, petrissage, arm shaking, cross fiber, and wringing), also to the elbow flexors following an eccentric bout. He found that the massage was successful at decreasing DOMS, decreasing CK, and increasing neutrophil presence in the affected area.
Smith\textsuperscript{32} was the first to document a reduction of DOMS through the use of massage, and it's interesting to note that the massage wasn't applied to the subjects in this study until two hours after the cessation of the activity. Thus Smith\textsuperscript{32} postulated that the lower DOMS values were observed secondary to reduced emigration of neutrophils. More specifically, vigorous massage was thought to hinder the displacement of neutrophils into the tissue, thus rather than letting them marginate along the vessel wall as blood flow slows down and the arterioles dilate during the initial stages of inflammation. Thus, more neutrophils are kept in circulation. Tiidus\textsuperscript{30} concluded that the most important consideration regarding the effectiveness of massage is the aggressiveness and duration of the treatment, as he applied his massage immediately after the exercise bout and still observed a reduction in soreness values.

Ciccone\textsuperscript{33} and Hasson\textsuperscript{34} looked at the possibility of using ultrasound as the treatment of choice in reducing the effects of DOMS. Specifically, Ciccone\textsuperscript{33} tried using ultrasound and trolamine salicylate phonophoresis to alter the amount of soreness perceived by subjects. Phonophoresis consists of using ultrasound to drive a drug through to the underlying tissue, thus offering the potential to deliver a drug in a safe, effective manner to tissues deep within the body. In his study, Ciccone\textsuperscript{33} delivered trolamine salicylate, which is considered to have analgesic and anti-inflammatory effects as do the salicylates in general. The salicylates are believed to attenuate pain and inflammation through their inhibitory effect on prostaglandin production. Initially, he induced eccentric soreness through repeated contractions of the elbow flexors of 40 college-aged women. The women were then divided into one
of four treatment groups: sham ultrasound with placebo cream, real ultrasound (1.5 W/cm², 1 MHz sound head, continuous) with placebo cream, sham ultrasound with trolamine salicylate cream, and real ultrasound with salicylate cream. All treatments were given for 5 minutes.

Results of this study found that the group which received only trolamine salicylate cream, though not statistically significant, experienced the smallest increase in soreness values, while the group treated only with continuous ultrasound experienced the greatest soreness values. Ciccone theorized that in this case the continuous ultrasound would serve to only increase the inflammatory response by increasing blood flow and local heating. Furthermore, the cream was only applied once a day for three days, while in another study a statistically significant decrease in DOMS was reported when the same cream was applied four times per day. Also, the group which received real phonophoresis demonstrated less soreness than the control group, further showing the effectiveness of the cream by counter-acting the effects of the ultrasound. Conversely, in a study analyzing the effects of pulsed ultrasound on DOMS, Hasson induced eccentric soreness of the quadriceps on eighteen subjects aged 20-37 years. He found that the level of soreness perception was significantly less (p<.05) in the pulsed ultrasound group than either the sham ultrasound or control group at 48 hrs. post-exercise. The 1.0 MHz pulsed ultrasound sound head was used at an intensity of .8 w/cm², with a 1:4 pulsed ratio for twenty minutes.

The use of TENS is widely accepted in the treatment of a variety of conditions for the relief of acute and chronic pain. In a study by Craig, 24 male and 24 female
subjects were placed into a control, placebo, high TENS, or low TENS treatment groups. The high TENS group received 200 microsecond, 110 Hz stimulation, while the low TENS group was at a current level of 200 microseconds and 4 Hz. DOMS was induced in the elbow flexors, with treatment given to the appropriate groups at 24, 48, and 72 hours post exercise. DOMS values were obtained using the visual analogue scale on each of the three days in addition to the McGill Pain Questionnaire on day three only, with the results showing no significant effect of either high or low TENS on the severity of pain perception of the subjects.
CHAPTER 6
CONCLUSION

All of the preceding studies involving the use of various modalities and creams to treat the sometimes debilitating effects of DOMS are interesting and for the most part well executed; however, every one of them pointed to prevention as the key to eliminating DOMS. Perhaps it is more appropriate to use the term adaptation rather than prevention, since it is impossible to not get sore after initiating a fairly strenuous exercise program. It is widely accepted that DOMS will only occur after the first few bouts of exercise and therefore consistent training acts as a treatment in and of itself. Friden and colleagues\(^\text{37}\) have found less fiber damage after training, implying that there is an adaptation and protective mechanism associated with regular physical exercise.

In Balnave’s\(^\text{6}\) study, the 16 subjects in the treatment group performed a 40 minute downhill walk once per week for eight weeks, with elimination of soreness after the second walk (week two) through the end of the training program. This repeat bout adaptation was also observed by Byrnes\(^\text{38}\) and Nosaka\(^\text{39}\), who each put their subjects through aerobically strenuous exercise. Interestingly, more bouts of anaerobic exercise have been shown to be needed in order to demonstrate similar adaptations to training and corresponding reduction of DOMS values. In studies involving eccentric training of the elbow flexors, it was found that three to four
training bouts were needed to significantly abolish DOMS.\textsuperscript{15,16}

As the research in this chapter shows, while severe cases of DOMS may be beneficially treated with an anti-inflammatory cream, massage, or other modality, the best approach lies in the development of a gradually progressive exercise program.\textsuperscript{1,4,6} Likewise, it is important for therapists to understand the normal timelines and adaptation processes of aerobic vs. anaerobic exercise in order to know when the pain perceived by the patient is something other than DOMS.
References


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