EFFECT OF MYOFASCIAL DECOMPRESSION ON DELAYED ONSET MUSCLE SORENESS FOLLOWING EXERCISE-INDUCED MUSCLE DAMAGE

by

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ABSTRACT

The purpose of this study was to determine the effect of myofascial decompression (MFD) on delayed onset muscle soreness (DOMS) and associated responses following an exercise-induced muscle damage protocol (EIMD). Ten college students underwent a battery of baseline testing including relaxed and flexed arm angle, arm girth, isometric strength, muscle tenderness, and muscle soreness, followed by an EIMD protocol of 25 eccentric bicep curls. MFD was performed on the experimental arm, the control arm received no treatment, and the same battery of testing was performed post, 24, 48, and 72 hours after intervention. A two-factor ANOVA of condition by time with repeated measures and a $p < .05$ showed no differences between conditions. Main effects of time existed for relaxed and flexed arm angle ($p= .000$), isometric strength ($p=. 038$), muscle tenderness ($p= .006$), and muscle soreness ($p=. 000$). EIMD produced expected muscle damage effects in all variables except arm girth and isometric strength. This was the first study to implement MFD at an early stage of the muscle repair/DOMS process; however, no statistically significant results appeared between conditions. These findings suggest that for MFD to be successful in reducing DOMS and the effects of EIMD, it may be necessary for MFD to be used later in the repair process.

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CHAPTER I

INTRODUCTION

As athletes continue to grow faster and stronger, there is a heightened desire for athletic trainers and physical therapists to keep their athletes performing at optimal levels on a daily basis. Therefore, it is important to examine treatment modalities in order to optimize recovery and enhance performance. Myofascial decompression is a movement based set of manual therapy treatment techniques, which evolved out of addressing sports and orthopedic injuries that did not respond to traditional soft tissue interventions or therapeutic exercises.[1] Cupping and MFD both produce large, circular bruises commonly seen on Olympic athletes like Michael Phelps. While both are similar in that they are therapeutic modalities, MFD involves an active movement component.[1] A therapeutic modality is needed when there is damage to muscle tissue. Exercise-induced muscle damage (EIMD), which manifests itself as delayed onset muscle soreness (DOMS), is one form of damage that could potentially benefit from an effective intervention.[2] An eccentric exercise, such as the downward phase of a bicep curl, lengthens the muscle while it simultaneously contracts. This causes microtears in the myofibrils of the muscle.[3] As a result, the inflammatory process begins, whereby blood is shunted to the area of damage allowing white blood cells (WBCs) to phagocytize and dispose of damaged tissue.[3]

By 12-24 hours after the primary damage, DOMS begins, which is due to an exaggerated inflammatory response, including heat, redness, swelling, pain, and loss of function.[4] The inflammation-induced damage is known as secondary damage and worsens between 24-48 hours, and starts declining at 72 hours.[4] MFD lifts the tissues and allows more blood flow to the area of damage so WBCs can initiate repair.[5] The active movement allows the new tissue (collagen) to be re-aligned in the correct manner as the muscle moves through commonly used motions. For

example, active flexion and extension at the elbow joint with the cups on the biceps allows the biceps muscle to align in a manner that is conducive for these movements.

Although the inflammatory response is believed to be a necessary process for the purposes of promoting repair of damaged tissue, excessive inflammation may be harmful. The goal of a therapeutic modality is to control the inflammatory response in order to optimize recovery. The effectiveness of MFD in controlling inflammation following EIMD has not been determined; thus, controlled studies are needed to examine the effectiveness of this modality in reducing the associated responses of DOMS (loss of range of motion, functional strength, and increased pain).

PURPOSE

The purpose of this study was to determine the effect of MFD on DOMS and associated responses following an EIMD protocol in uninjured, non-athlete college students by measuring relaxed arm hang angle, flexed arm angle, isometric elbow flexor strength, arm girth, muscle soreness, and muscle pain.

RESEARCH QUESTION

- Can MFD reduce DOMS and its associated effects?

HYPOTHESIS

H1: In comparison to a non-treated control condition, MFD will decrease the markers of EIMD as indicated by smaller reductions in relaxed arm hang angle and isometric elbow flexor strength; and smaller increases in flexed arm angle, arm girth, muscle tenderness, and muscle soreness.

ASSUMPTIONS

- The primary investigator will be performing all the myofascial decompression treatments.
- All subjects will comply with pre-treatment instructions and will not have participated in any prior stretching or treatment the day of study participation.
- No subject will have participated in previous cupping therapy on the biceps.
- No subject will have injured his or her biceps within the past six months.

DELIMITATIONS

- All subjects will be non-athletes from Texas Christian University.
- Subjects will be between ages 18-35 years old.

LIMITATIONS

- No device was used to ensure perfect range of motion measures each time.
- Only one male was successfully recruited.
- Subjects will know which arm is the experimental arm because there is no way to make this a blind study.

CHAPTER II

REVIEW OF LITERATURE

The purpose of this study was to determine the effect of myofascial decompression (MFD) on delayed onset muscle soreness (DOMS) and associated responses following an exercise-induced muscle damage (EIMD) protocol in non-injured, non-athlete college students. This literature review will focus on what fascia is and how it is related to muscle function, muscle damage causes and responses, delayed onset muscle soreness and associated responses, and the effect that myofascial decompression has on the muscle repair process.

Anatomy Review

Fascia

As the name suggests, myofascial decompression involves decompressing the fascia. According to Stecco,[6] the newest medical definition of fascia is "a sheath, a sheet, or any number of other dissectible aggregations of connective tissue that forms beneath the skin to attach, enclose, and separate muscle and other organs."[6] On the other hand, a relatively older and broader definition according to Robert Schleip and Thomas Findley[6] is "fascia is the soft tissue component of the connective tissue system that permeates the human body, forming a whole-body continuous three-dimensional matrix of structural support."[6] Fascia can be organized by location; the two types of fascia that are involved in muscle damage and MFD are the superficial fascia and deep fascia. The superficial fascia is a fibrous layer of loose connective tissue directly underneath a slightly more superficial layer of adipose tissue, which is directly underneath the skin. It separates skin from muscles, which allows normal sliding action over each other. Deep fascia, which is known as the epimysium, is denser and covers the muscles forming discrete pockets around each muscle. It keeps everything separate yet interconnected and allows for

sliding on each other, like the superficial fascia. The roles of fascia include serving as a support structure, tension, and suspension for tissues.[6]

One of the most important features of fascia is tensegrity: tension and integrity. When a strain is put on a tensegrity structure, such as the body, deformation will be distributed all over the structure. Therefore, when there is a strain placed on a muscle and its surrounding fascia, such as from an eccentric exercise as is used during EIMD, the strain is distributed to only the muscle and fascia, but also the surrounding soft tissue. As a result of muscle and fascial damage, they both become restricted; however, the fascia itself can be expanded due to its tensegrity property.[7] This expansion is transmitted throughout all the soft tissue layers and area around the portion of fascia that is decompressed with the cups during MFD.

Fascia is essential for transmitting muscle contraction force to the bones. Without the fascia, the muscles would not be able to generate nearly the same amount of force necessary for proper movement and function. New research has shown active contractile cells within fascial tissue itself, indicating that it is able to contract and elongate with some degree of neurological control.[8]

Fascia may be injured in various ways. It is extremely elastic, but extreme tensile stress, such as eccentric exercise, can cause the fascia to tear or perforate. Many injuries involve not only damage to the fascia, but also the tissue it envelops, such as the muscle. After injury, fascia remains in a shortened position. If there is not a therapeutic modality to fix this position, it will remain in the shortened position for an extended time and adopt that shortened position. Furthermore, tension in the associated muscle fibers activates the contractile fibers within the fascia to maintain the shortened position. This extended time in the shortened position leads to fibrous cross-linking within the fascial tissue, resulting in limited motion.[8] Associated with

limited motion is a decrease in range of motion (ROM), decrease in functional strength, and increased pain, all which result from the initial muscle damage that affects the surrounding fascia as well.

Muscle Damage

EIMD

Exercise-induced muscle damage is characterized by muscle soreness, muscle swelling, temporary muscle damage, an increase in intramuscular protein in the blood, greater passive muscle tension, and a decrease in muscular strength and ROM.[9] The most common type of exercise to induce this type of muscle damage is eccentric exercise. Eccentric exercise also serves as the mechanical stressor causing the primary damage that triggers the inflammation and repair process.[11]

Eccentric Exercise- Primary Damage and Inflammatory Response

Eccentric exercise is characterized by muscle lengthening during contraction, which leads to muscle damage and is distinguished by loss of range of motion and development of DOMS.[11] Specifically, it has been shown that the lengthening of the muscle during contraction causes myofibrillar disturbances consisting of Z-band disruption and streaming. The cause of this disturbance is likely due to the high local muscle tensions that characterize eccentric exercises. The high-tension overstretches the sarcomeres, which causes sarcoplasmic reticulum damage via tearing of the t-tubules.[13] Calcium is released uncontrollably from its stores, causing contractures of the muscle fiber. Eventually the muscle fiber dies and the restoration of normal calcium levels is delayed. Furthermore, the ability to generate normal levels of force because of abnormal calcium levels is impaired and loss of strength results. Additionally, the death of fibers from the primary damage triggers an inflammatory response, resulting in swelling, soreness, tenderness,

and loss of ROM. As the inflammatory response breaks down the dead fiber, the breakdown products are sensed by nociceptors, also known as pain receptors, thus causing the sensation of pain. Furthermore, inflammatory responses result in further injury; the associated breakdown products that the nociceptors sense are normally non-stimulatory, so the muscle becomes tender to palpation, stretch, and contraction, thus further reducing ROM, functional strength, and increasing pain.[9]

Delayed Onset Muscle Soreness

Inflammatory response

Pain, inflammation, decreased muscle function, and decreased ROM are results of EIMD. This physiological response begins to develop 12-24 hours after exercise has been performed, and may produce the greatest pain up to 24-72 hours after exercise has been performed.[13] Delayed onset muscle soreness results from unaccustomed exercise and results from the primary damage and associated inflammatory response from eccentric exercise. The mechanically-induced primary damage then produces secondary damage, with the associated structural damage to the muscles and connective tissue. It is believed to be due to an accentuated inflammatory process triggered by the eccentric exercise. This secondary damage may result in altered muscle function and joint mechanics, thus reducing performance and restricting athletes from optimal performance.[14]

As EIMD results in DOMS, which includes the typical symptoms of strength loss, pain, muscle tenderness, stiffness, and swelling, it's logical that treatments for DOMS are often aimed at reducing the inflammatory response.[10] Currently, there is no universal treatment to prevent or adequately reduce DOMS; however, it is of great interest because of the significant impact it has in athletes training and performance.

Therapeutic Modalities

DOMS often interferes with rehabilitation, activities of daily living, or hinders an athlete from continuously training at optimal levels, so a therapeutic modality that eliminates the effects of DOMS is warranted. Many therapeutic modalities claim to help alleviate symptoms of DOMS; however, many of these claims are based off anecdotes or are not uniform in methodology. The main goal of a therapeutic modality in relation to DOMS is to reduce soreness and return muscular strength to initial levels.[15] Based on the prior studies on therapeutic modalities aiming to reduce symptoms of DOMS, it is clear that there is no conclusive evidence supporting one modality over another. Three of the most popular modalities are ice, foam rolling, and massage.

Ice

Cold water immersion, or use of ice to remove heat and lower tissue temperatures, is purported to reduce inflammation, edema, and pain sensation, which all occur as a result of EIMD.[16] Within the past 10 years, cold-water immersion, specifically, has gained popularity to improve recovery from EIMD and its associated responses, but most benefits are from anecdotes rather than by empirical evidence.[17]

A study with twenty male soccer players from two national league teams who just completed a match analyzed the effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. Biochemical, neuromuscular, and perceptual markers of muscle damage, blood samples, perceived muscle soreness, and functional data were obtained at baseline, within 30min of the end, and 24 and 48hr after a one-off friendly soccer match. Immediately after the match, subjects in the cold-water immersion group immersed their legs in the 10-degree C water for 10 minutes. The results revealed that the

cold-water immersion group reported less DOMS in hip abductors at 30 min and in calf and quadriceps at 24 hr compared with a thermoneutral water immersion group at the same time points. This study suggests that water immersion immediately after a one-off soccer match reduces muscle damage and discomfort.[18] It is important to note that this study did not control for the type or extent of muscle damage, which may affect the reliability of the results.

Unlike the previous study, a study by Isabel et al[19] did not find the application of ice to be effective at reducing the effects of DOMS. They hypothesized that using ice closer to the time of muscle damage would improve results, which was in response to Davis's[20] study that used application of ice too late post-exercise and not frequently enough to produce statistically significant results. Participants included twenty-two volunteers enrolled in basketball activity classes with no upper arm pain and pain-free range of motion of the elbow. Pre-exercise measurements for strength, ROM, perceived soreness, and CK levels were obtained prior to inducing muscle soreness. The muscle soreness was induced by having subjects perform 30 sets of 10 reps of dumbbell curls at 90% of their one-repetition max. Next, subjects underwent eight 15-minute treatments at 0, 2, 4, 6, 24, 48, 72, and 96 hours post-exercise. In the ice massage experimental group, the subjects used ice to massage the entire length of their biceps and the proximal portion of the brachialis. Post-exercise measures were taken in the same manner as the pre-exercise measures. Results showed that neither the repetition of the use of ice nor timing of the application of ice improved DOMS effects.[19,20] This study may have produced different results than the previous study because of the additional measures taken to control the type and extent of muscle damage.

Goodall and Howatson[17] studied the efficacy of repeated cold-water immersion in the recovery of exercise induced muscle damage, similar to the study by Isabel et al. [19] Eighteen

physically active males completed a muscle damaging protocol of 100-drop jumps. Then, the cold-water immersion group underwent a seated immersion in an ice bath of 15 degrees C for 12 minutes. The subjects repeated the ice bath immersion every 24 hours for the following three days. Variables were measured at post, 24, 48, 72, and 96 hours post-exercise. Results showed that the cold-water immersion did not attenuate any of the dependent variables, which included maximal voluntary contraction of the knee extensors, creatine kinase activity, muscle soreness, range of motion, and limb girth, at 24, 48, or 72 hours. Therefore, the authors concluded that cold-water immersion did not enhance recovery from a bout of eccentric contractions, [17] which coincides with the results from Isabel et al.[19]

From the above studies, the exact mechanisms of the cooling effect on the perception of soreness and pain are unclear; however, according to Ascensão, the most widely accepted mechanism is the analgesic effect of ice.[20] Generally, temperatures utilized in these studies fall within 10-15 degrees C for approximately 10-15min and are repeated 1-3 times.[17] Therefore, this analgesic effect is probably because muscle tissue temperatures of 10-15 degrees C reduce nerve conduction velocity, mechanoreceptor activity including muscle spindles with a resulting blunted stretch reflex response, and inhibition of the pain-spasm cycle. [20, 21] However, overall, there are discrepancies in temperature, frequency, time, and method of temperature reduction. As a result, ice or cold temperatures may be have more psychological effects because of the feeling of lessened fatigue and pain rather than physiological modalities due to the lack of supporting scientific evidence.[22]

Foam Rolling

The purpose of foam rolling in relation to alleviating DOMS is to use one's own body mass on a foam roller to exert pressure on one's soft tissue. The rolling motions place direct and

sweeping pressure on the soft tissue, which stretches it and generates friction between it and the foam roller. As a result, it is believed that foam rolling reduces muscle soreness.[14] Furthermore, anecdotal accounts claim that foam rolling corrects muscular imbalances, improves muscle soreness, and improves range of motion.[9]

In a study of eight healthy, physically active males, the effects of foam rolling as a recovery tool after an intense exercise protocol were measured by assessing pressure-pain threshold, sprint time, change-in-direction speed, power, and dynamic strength-endurance. The intense exercise protocol consisted of 10 sets of 10 repetitions of barbell back squats at 60% of one's one-repetition maximums. Next, participants in the foam-rolling group rolled out their muscles using their own body mass for 45 seconds followed by 15 seconds of rest. This was repeated for each muscle group in each lower extremity and repeated once. Results showed that foam rolling improved quadriceps muscle tenderness, but no other muscle group, by a moderate to large amount in the days after fatigue. Substantial effects were also found in sprint time, power, and dynamic strength-endurance, which are all dynamic performance measures. The authors concluded that foam rolling effectively reduced DOMS and associated decrements in most dynamic performance measures.[14] Although the results seem promising, the sample size was rather small and specific.

Similarly, MacDonald et al[9] looked at foam rolling as a recovery tool after an intense bout of physical activity. Twenty physically active, resistance-trained male subjects performed an EIMD protocol of 10 x 10 squats. Then the subjects in the foam-rolling group performed five different foam-rolling exercises in order to target the major muscle groups of the thigh and gluteal region. The subjects performed each of the five exercises on both legs for two 60sec bouts each. Results showed that foam rolling had a "moderate" effect post-24hr and a "large"

effect post-48hrs on muscle soreness. A “moderate” effect was found for passive range of motion on the quadriceps at post-48 and post-72 hours; a “moderate” effect post-72hrs for passive hamstrings range of motion; and a “moderate” effect post-24hr for dynamic hamstring range of motion. Substantial decreases in pain were found in the foam-rolling group compared to the control group. Most importantly, the improvement in ROM for the foam rolling group was attributed to the similar mechanisms between myofascial decompression and foam-rolling, such as potentially reducing adhesions between layers of fascia, reducing inflammation, and/or reducing muscle soreness.[9] While this study was extremely similar to the previous study in subject ability and intense physical activity protocol, this study increased the duration of foam rolling and looked at more variables.

A systematic review was conducted to determine if self-myofascial release with a foam roller improved ROM, reduced acute muscle soreness, and reduced DOMS. After the review of 14 peer-reviewed journal articles, Cheatham et al[22] concluded that self-myofascial release using a foam roller appears to have short-term effects on increasing ROM and may decrease DOMS after intense exercise. Due to the differences in methods among the studies, however, there is no consensus on the optimal duration and timing of a foam-rolling program.[22]

Massage

Massage affects DOMS during its early stages of inflammation by applying mechanical pressure, which may decrease neutrophil margination, therefore reducing inflammation and DOMS. Massage has been hypothesized to control inflammation, improve blood flow, and reduce stiffness, swelling, and muscular tone, resulting in a reduction in the sensation of pain.[23, 24]

Hilbert et al[25] looked at the effects of massage on DOMS in eighteen volunteers who were randomly assigned to control or massage groups. DOMS was induced by six sets of maximal eccentric contractions with the right hamstring with one-minute rests between sets followed by five more maximal eccentric contractions. Then the subjects received either a 20-minute massage or a 20-minute sham massage (control). In the massage group, the massage consisted of a classical Swedish technique including stroking, percussion, and kneading. After analyzing results, no significant differences for peak torque, ROM, neutrophils, unpleasantness of soreness, or mood were found between groups, so massage administered 2 hr after exercise-induced muscle injury did not improve hamstring function. [25] Therefore, this study did not align with the literature hypothesizing that massage improves symptoms of DOMS.

In a systematic review analyzing if post-exercise massage treatment reduces delayed onset muscle soreness, a uniform consensus was not drawn. While some of the seven studies reviewed produced less soreness and less pain in the massaged leg, there were serious methodological flaws, such as sample size. As a result, massage may only be a promising rather than a definitive treatment.[24] Overall, massage has not been shown to be an effective method in improving ROM or muscular strength after EIMD, but it has been shown to be beneficial in treating EIMD by restoring blood flow, decreasing soreness and inflammation.[9] Further investigation into this therapeutic modality is necessary in order to draw a reliable conclusion about the effectiveness of massage as a means of relieving DOMS and its associated effects.

After reviewing the literature of the most popular anecdotal methods of reducing DOMS, it's evident that there is not one therapeutic modality that is more beneficial than the others. More research is necessary to discover what therapeutic modality is the most successful in controlling the inflammatory response and reducing DOMS. Cupping used to be one of the most

popular and desired therapeutic modalities to relieve pain; however, a similar yet different modality of MFD is a promising option based off its underlying mechanism.

Cupping vs. myofascial decompression

Cupping

While cupping and MFD are both therapeutic modalities and leave large, circular bruises, there are notable and important differences. To start, cupping therapy has been used for centuries in China, and around the world, as a special healing skill.[26] The purpose is to cure disease and associated pain by using cups that target soft tissues by applying local pressure to acupoints.[27] Amongst 550 studies included in a systematic review, the top three diseases/conditions that were treated by cupping included pain, herpes zoster, and cough or asthma.[28] In Traditional Chinese medicine, it is believed that pain is mainly caused by disorder of *qi* (energy) and blood circulation, causing blood stasis or *qi* blockage in organs, energy channels, and other parts of the body. Therefore, the physiological purpose of the cups is to compress the tissue and create a vacuum, which results in local hyperemia or homeostasis. Thus, capillaries expand and the amount of fluid, and associated nutrients and oxygen, entering and leaving the tissues increases.[28] The Chinese believe that cupping treatment regulates and promotes movement of qi and blood, which, by doing so, alleviates pain caused by blood stasis and qi blockage. Cao H et al[27] showed that cupping therapy does effectively reduce acute and chronic pain due to illness in the short-term; however, this reduction in pain was only based off of participant self-reporting.[27] There was no statistical data, such as ROM or blood marker levels, to prove the reduction in muscle damage or inflammation, and thus a reduction in pain.[27] Additionally, the systematic review suggests that there is insufficient high-quality evidence to support the use of cupping therapy on relevant disease.[28]

Myofascial decompression

MFD is a novel therapeutic modality that is replacing the traditional soft tissue manipulation of cupping. MFD is similar to cupping because it is a manual therapy that uses negative pressure technology via the cups; however, there are several differences. While cupping treats ailments and associated pain, especially those issues associated with *qi*, MFD focuses on movement-based impairments from muscle damage. The main purpose of MFD is to treat muscle injury, such as hamstring injuries, improve muscular healing from prior damage, and improve ROM.[29] Recently, MFD has been used to reduce DOMS and associated responses resulting from exercise-induced muscle damage. While the cups are placed on acupoints in Traditional Chinese cupping, in MFD the cups are placed along myofascial tracks where palpable taut bands of skeletal muscle lie with their associated trigger points. They are placed on trigger points because of the low levels of oxygen, nutrients, and blood perfusion, which contribute to decreased healing after muscle damage.[26] The cups decompress the tissue which lifts the superficial and deep fascia.[1] Blood flow increases, bringing with it leukocytes that can destroy and dispose of damaged tissue along with new, random, crisscrossed tissue that has been laid down as a result of the initial inflammatory response. Within these damaged and abnormal tissues are adhesions, which act as unwanted glue between layers of muscle, fascia, and skin, and decrease the strength and normal range of motion of the muscle. However, the leukocytes can break down these adhesions, so the tissue can be mobilized. This mobilization is created via active movement, which is the most important part of MFD.[1] Western medicine-based movement paradigms and algorithms determine how to move the body through active motions.[26] The active movement allows the new tissue that had been laid down to be re-mobilized in the correct alignment and pattern.[1] Thus, the normal function of the muscle is restored. Along with the leukocytes, blood

brings its associated nutrients. As a result of the increased blood flow, endogenous opioids or endorphins that act as natural painkillers are produced, which thus reduce the sensation of pain.[29]

A previous study by LaCross[5] on Division 1 athletes with hamstring injuries showed no difference in increasing ROM between MFD and other modalities (heat and foam rolling), but did show an increased perception of overall health. Seventeen Division I college athletes were randomly assigned to the control group of a moist heat pack and foam rolling treatment or the experimental group of MFD. All subjects were selected on the criteria suggesting symptoms of a hamstring strain, as reported by the athletic trainer. The symptoms ranged from acute to chronic, but none of the subjects presented anything greater than a Grade I hamstring injury and all were participating in some form of activity. The associated symptoms of a hamstring strain that the subjects complained of included tightness, pain, decreased strength, and decreased flexibility, all of which are typical symptoms of DOMS. Prior to MFD, pre-testing measures of ROM and the Perceived Functional Ability Questionnaire (PFAQ) survey were completed. For the experimental group, the intervention began with light scraping of the area using an Instrument Assisted Soft Tissue Mobilization (IASTM) soft tissue instrument to increase blood flow and screen for soft tissue adhesions. After that, the cups were placed on the skin statically for three minutes, followed by series of active movement patterns, ten hamstring curls, and ten prone straight leg raises all with the cups in place. Then the clinician passively moved the subject's leg through passive ROM with the cups still on the skin. After that, the primary investigator slid the cups along the treatment area in a distal to proximal pattern. Immediately after removal of the cups, ROM, PFAQ, and Global Rating of Change Scale (GROC) were completed. After analysis, it was concluded that both therapeutic modalities produced similar gains in ROM measures. The main difference between modalities was that the MFD group felt significantly better after this

intervention as compared to the control group, as indicated by the GROC scale. This scale asked subjects to select a number that described their overall attitude toward how their hamstring felt immediately after the treatment.[5] Therefore, the main results of this study are perceived rather than physiologically-based. Additionally, this study used subjects that had previous injuries, didn't induce controlled muscle damage, and intervened with MFD at an unknown point in the repair process.

Smith[29] conducted a study on the effect of myofascial decompression on shoulder range of motion and strength of healthy overhead athletes. The purpose of the study was to understand the immediate effects of MFD on range of motion strength of internal and external rotation of the shoulder, and to explore MFD as an effective treatment for overhead athletes. Thirty subjects with no shoulder pain or injury within the past six months performed 10 repetitions of maximal effort and speed in internal and external rotation. Average torque of both internal and external rotation of the ten repetitions was calculated. Then half of the subjects that were assigned to the experimental treatment group received a single MFD treatment. Treatment started with a light scanning of the area using an IASTM tool to increase blood flow and screen for soft tissue adhesions. Then the cups were placed in an anatomically inspired pattern and left on the skin statically for five minutes. The pattern consisted of two standard (2in. in diameter) cups placed medial of the scapula with one at the inferior angle of the scapula and the other at the superior angle; one large (3in. in diameter) cup placed inferior of the spine of the scapula over the infraspinatus; one standard size (1.5in diameter) curved cup placed over the medial supraspinatus with the small curved cup over the lateral aspect of the muscle. Each cup was pumped three times or until pressure was complete with the suction tool. After the static five minutes, the subjects performed a series of active movement patterns for ten repetitions of shoulder flexion,

should abduction, internal and external rotation through total arc of motion with the cups in place. The cups were then removed and a flush, using the IASTM tool, was completed on the areas that the cups were in contact with the skin. The results showed that there was no difference in decreasing loss of functional strength with MFD or control, but results did show an increase in external ROM after MFD compared to the control. The author concluded that the increase in external ROM after MFD was most likely because of increasing blood flow and lengthening of the external rotator muscles.[29] This study was similar to that of LaCross[5] because both subject populations were athletes, but these athletes were uninjured. Although this study aimed to induce muscle damage, 10 repetitions may not have produced adequate muscle damage.

Lastly, a study by Xie[30] examined the effects of static versus dynamic MFD on hamstring flexibility in a college-aged population. Twenty-nine healthy college-aged students who were assessed as having limited hamstring muscle flexibility were randomly assigned to a static or dynamic group. Baseline measures of hamstring flexibility and hamstring strength were performed prior to the interventions. The interventions consisted of MFD applied for 5 minutes to the area with the greatest muscle flexibility restriction under the constraints of either static or dynamic MFD. Subjects receiving both interventions were asked to rate their pain using the Visual Analog Scale after the first 10 sec of cup application, at 2.5 minutes, and immediately following removal of the cups at 5 minutes. Immediately after the intervention, 24 hours and 48 hours post-intervention, subjects underwent hamstring flexibility, strength testing, and filled out the GROC scale. In the static MFD intervention, three cups were placed along the hamstrings at positions commonly used in clinical practice: one cup was applied near the ischial tuberosity below the gluteal fold, one at the insertion of the biceps femoris, and one between the two in the middle of the muscle belly. The cups remained static for 5 minutes. In the dynamic MFD

intervention, two cups were placed along the hamstrings. A thin layer of soft tissue massage cream was applied to the thigh to help gliding of the cups. The cups also remained on the hamstrings for 5 minutes. The results of this study showed that both static and dynamic MFD improved measured and perceived hamstring flexibility.[30] This study may have produced statistically significant results, but it did not induce muscle damage, so it is difficult to determine what role MFD played compared to the other studies.

In previous literature, MFD was utilized after the primary damage, inflammatory response, and secondary damage from DOMS had begun or had never been induced; thus, if the damage had been initiated and carried out, the new tissue would had been laid down in an incorrect manner, adhesions would had formed, and DOMS responses would have been in full effect. In the current study, I proposed that if MFD is used earlier in the repair process, then the initial inflammatory response may be controlled and the secondary damage and DOMS may be reduced.

CHAPTER III

METHODOLOGY

Myofascial decompression was compared to a control condition with active flexion and extension of the elbow to determine the effects on exercise-induced muscle damage and its associated responses of swelling, girth, strength, tenderness, and soreness. The focus of this study was to determine the effectiveness of myofascial decompression intervention immediately post exercise-induced muscle damage.

Subjects

Ten college students ages 19-22, 9 females and 1 male, from Texas Christian University were recruited to participate in this study. Subjects' arms were randomly assigned to control and experimental. Each subject served as his or her own control. Four dominant arms (right) and six non-dominant arms (left) received the intervention of MFD, while the opposite arm served as a control for each subject. A department review board approved this study. Subjects were asked to sign a consent form and fill out a health history form. None of the subjects in the study had received any type of cupping therapy on the biceps prior to this study. None of the subjects had any biceps injury within six months of participating in this study. Prior to data collection the participants were informed to tell the investigator if the treatment became too painful or uncomfortable, at which time the treatment would stop. All of the subjects in this study were able to complete the full intervention for both experimental and control arms.

Materials

The cupping set used was a Kangzhu Biomagnetic Chinese Cupping Therapy Set of 24 plastic valve cups that use a hand pump to control suction levels. A massage cream was used between the cups and the skin to allow for a more comfortable sensation while subjects actively

flexed and extended their bicep. The range of motion measurements were taken with a goniometer. Isometric elbow flexor strength and muscle tenderness were measured with a load cell. Arm girth was measured with a body tape measure. Muscle soreness was measured with a visual analog scale (VAS) soreness scale.

Image 1: Kangzhu Biomagnetic Chinese Cupping Therapy Set of 24

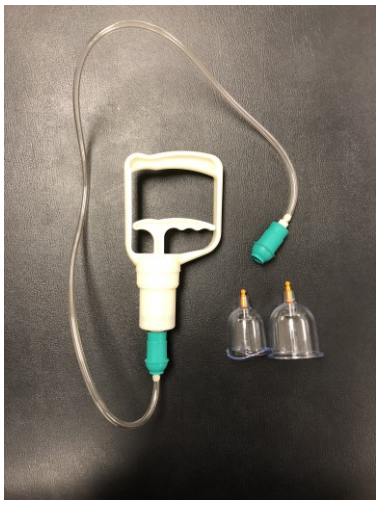

Image 2: Two cups most frequently used in the study with hand suction pump

Procedure

Before any measures were recorded each subject signed a consent form. Experimental testing began with one arm randomly assigned to experimental testing, with the other arm

designated as the control. Next, a series of baseline measurements were completed. These measures include: 1) the subject's relaxed and flexed arm angle measured at the elbow joint, 2) isometric elbow flexor strength, 3) muscle soreness, 4) muscle tenderness, and 5) arm girth. Relaxed arm angle was obtained by having the subject stand in the anatomical position with the arm hanging at their side. Flexed arm measurements were obtained by instructing the subjects to flex at the elbow and try to touch the shoulder while keeping the elbow tucked towards his/her side. Muscle strength, measured as isometric peak torque of the elbow flexors, was measured using a modified arm curl bench. The arm curl bench was equipped with a lever arm and a handgrip that were attached to a load cell. The load cell was interfaced with a digital indicator and a microcomputer. Subjects had their elbow joint positioned at 90 degrees, and the subject performed two maximal efforts by trying to curl the lever arm with the handgrip, which is locked in position. Each maximal effort lasted 4 seconds with a rest period of 30 seconds between trials.

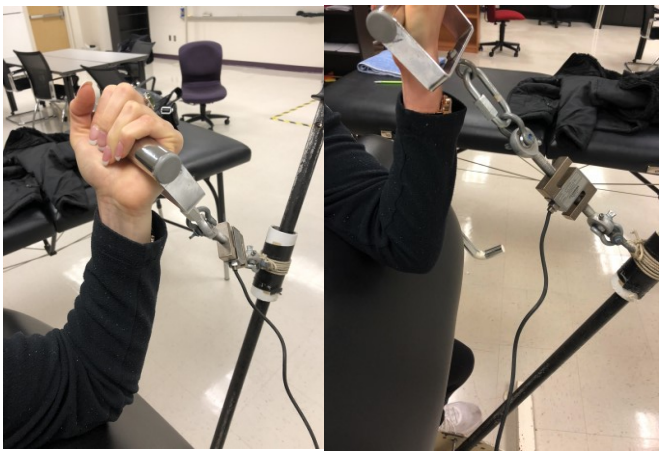

Image 3: Isometric elbow flexor strength measure using arm curl benched equipped with lever arm and handgrip attached to a cell.

Muscle soreness was recorded by having the subject mark on a 10-cm VAS with the zero end anchored with the descriptor, "no soreness" and the 10-cm end anchored with the descriptor, "extreme soreness". Muscle tenderness was determined using a dolorimeter, a device that

measures the force applied to the muscle necessary to elicit a mild pain response from the subject. Finally, muscle girth was measured at the maximum diameter of the muscle belly using a body tape measure.

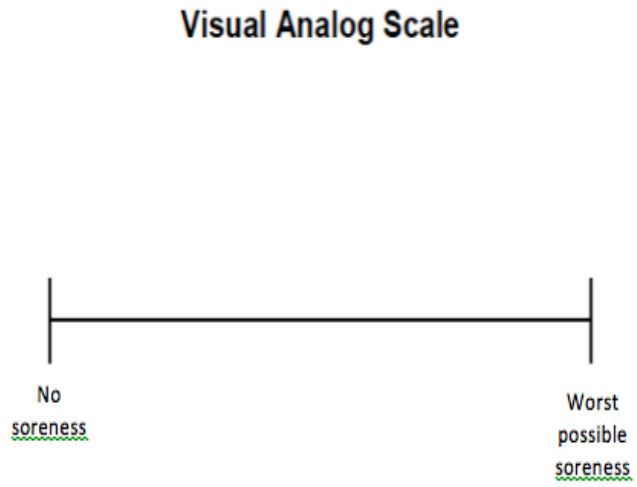

Image 4: Visual Analog Scale (VAS) used to measure muscle pain

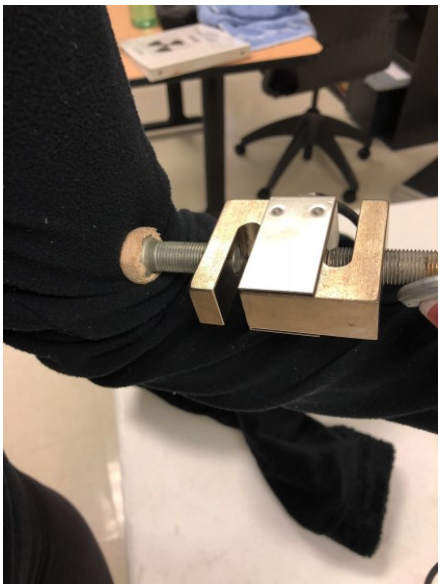

Image 5: Dolorimeter pressing into muscle belly to measure muscle tenderness

Following baseline testing, the eccentric exercise bout was performed. Subjects performed one set of 25 maximal eccentric contractions at a speed of 30 degrees per second from 60 to 180 degrees of flexion. The subjects began with their forearm flexed at 60 degrees, and the

forearm was forcibly extended to 180 degrees in four seconds. The primary investigator moved the arm through the concentric phase of the contraction to the starting position of 60 degrees at a speed of 10 degrees s-1 so no concentric contraction was performed by the subject.

Image 6: Starting position of arm for eccentric exercise: 60 degrees flexion

Image 7: Ending position of arm for eccentric exercise: 180 degrees extension

Immediately post-exercise, the MFD technique was performed on the experimental arm. Two cups of various sizes, depending on the size of the subject's arm, were placed along the biceps brachii myofascial track, specifically on the muscle belly and on the distal portion of the

myofascial track. A negative pressure was applied via the pump with three pumps to ensure the initial seal, and up to five total pumps to generate the necessary tissue lift. The cups remained on the skin with the patient performing active flexion and extension in supination for two minutes. For the control condition, the same baseline measures and eccentric exercise protocol were followed; however, no MFD protocol was implemented. The control subjects did, however, perform the same active movements with the control arm to replicate the 2 minutes of movement used in the MFD protocol.

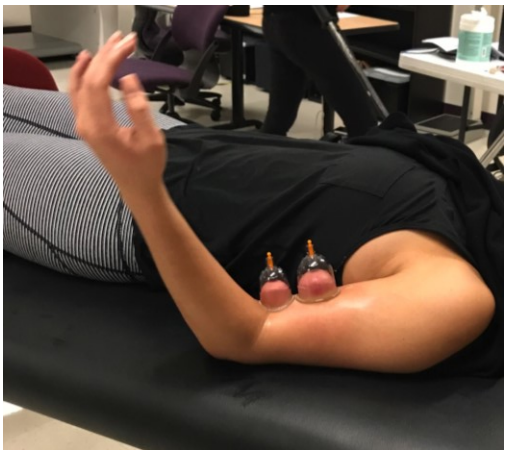

Image 8: Two cups, one on muscle belly of biceps m. and one on antecubital fossa near insertion of biceps m. into radial tuberosity

Immediately after the MFD protocol, the same battery of tests used in the baseline testing was administered. These tests were also conducted at 24, 48, 72 hours of recovery from the eccentric exercise protocol. In total, four visits were required for approximately a total of a two-hour time commitment.

Statistical Analysis

A two-factor analysis of variance (ANOVA) with repeated measures was used to detect differences between conditions (MFD and CON) and across time. The time factor had five leves with data sampled at before the EIMD procedure, immediately after exercise, and at 24, 48, and

72 hours after exercise-induced muscle damage (EIMD). A p-value was set at $p < .05$. A summary of the results can be found in Table 2.

CHAPTER IV

RESULTS

Data were collected from a total of 10 students [9 females (21.1+/- years, 166.8+/-cm, 59.8+/-kg) and 1 male (22 years, 182.9 cm, 93.0 kg)]. Ages of participants raged from 19-22 years old. Subjects were non-athletes without a history of a biceps injury within the past six months. Four dominant arms (right) and six non-dominant arms (left) received myofascial decompression (MFD) intervention. The means and standard deviations for the experimental and control groups can be found in Table 1.

Statistically significant differences were found between time measures for relaxed arm hang angle ($p = .000$) (Figure 1), flexed arm angle ($p = .000$) (Figure 2), muscle tenderness ($p = .006$) (Figure 5), and muscle soreness ($p = .000$) (Figure 6). No statistical significance was found between conditions. There was a tendency towards a main effect for isometric elbow flexor strength ($p = .051$) (Figure 3).

	Experimental			Control	
Variable	Measure	Mean	SD	Mean	SD
Relaxed arm angle (Degrees)	Pre	20.9	4.09	21.7	4.92
	Post	26.3	4.37	29.4	7.14
	24	26.1	7.62	28.6	6.55
	48	28.1	7.52	30.2	7.76
	72	28.1	5.20	23.8	9.20
Flexed arm angle (Degrees)	Pre	144.2	5.43	142.8	5.71
	Post	134.5	7.71	133.3	7.82
	24	136.8	11.93	139.2	8.48
	48	143.6	11.21	142.2	8.47
	72	141.6	7.18	143.7	10.40
Isometric strength (lbs.)	Pre	26.77	13.17	28.10	9.68
	Post	23.88	15.27	28.89	12.50
	24	28.44	15.86	30.18	13.53
	48	30.42	19.76	32.58	17.92
	72	32.94	16.74	34.84	17.69
Arm girth (cm)	Pre	28.82	3.43	28.83	3.15
	Post	28.81	3.49	29.17	3.57
	24	29.35	4.24	30.73	5.05
	48	28.68	4.13	29.9	4.79
	72	27.56	3.01	28.32	3.49
Muscle tenderness (lbs.)	Pre	6.5	3.52	7.9	2.29
	Post	8.61	2.87	7.38	3.59
	24	5.61	2.87	5.19	2.90
	48	4.38	2.46	5.04	2.36
	72	4.83	2.39	5.9	1.75
Muscle soreness (cm on 10cm VAS)	Pre	1.18	1.91	.65	1.07
	Post	3.69	2.69	3.43	1.73
	24	5.19	2.41	4.7	2.46
	48	4.94	2.62	3.93	2.42
	72	2.78	2.21	1.91	1.25

Table 1: Means and standard deviations for variables across time points (N=10).

Variable	Main effect condition	Main effect time	Interaction
Relaxed arm angle	.634	.000 *	.282
Flexed arm angle	.956	.000 *	.538
Isometric strength	.051	.038 *	.369
Arm girth	.153	.166	.226
Muscle tenderness	.820	.006 *	.114
Muscle soreness	.137	.000 *	.748

Table 2: Two-factor ANOVA Results Comparing Efficacy of Myofascial Decompression and Control Condition across Time (N= 10). * Indicates significance at p= < .05 level.

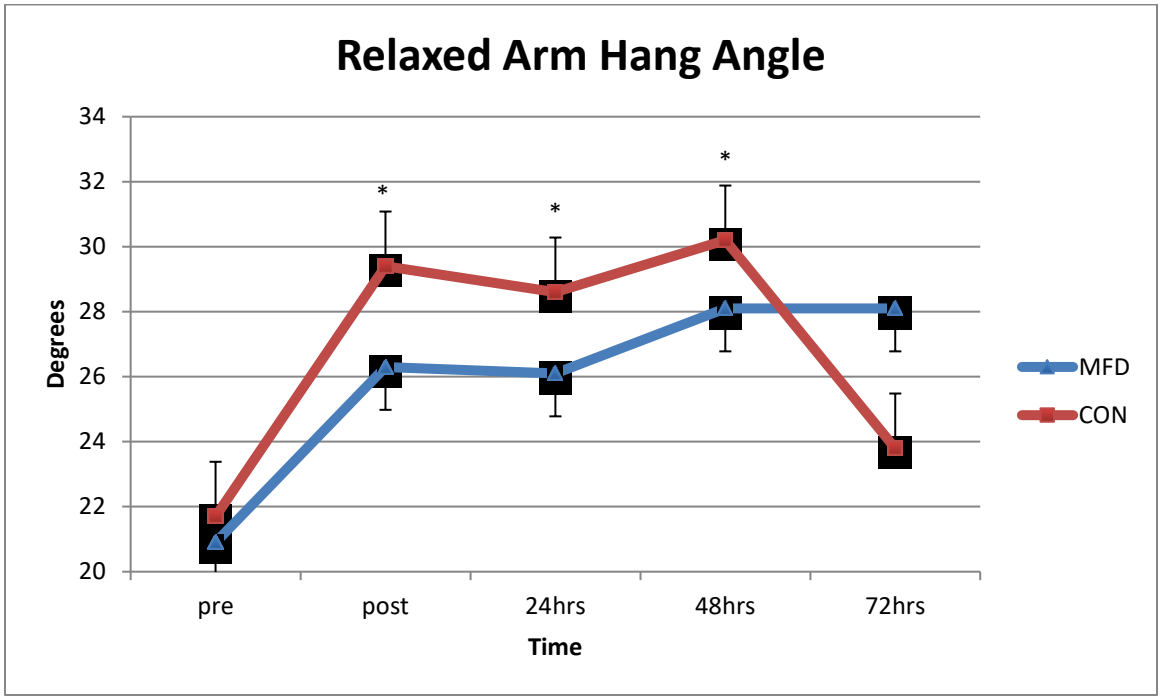

Figure 1: Relaxed Arm Hang Angle. The * indicates a main effect for time, P < 0.001 with marked time points different from pre and 72 hours.

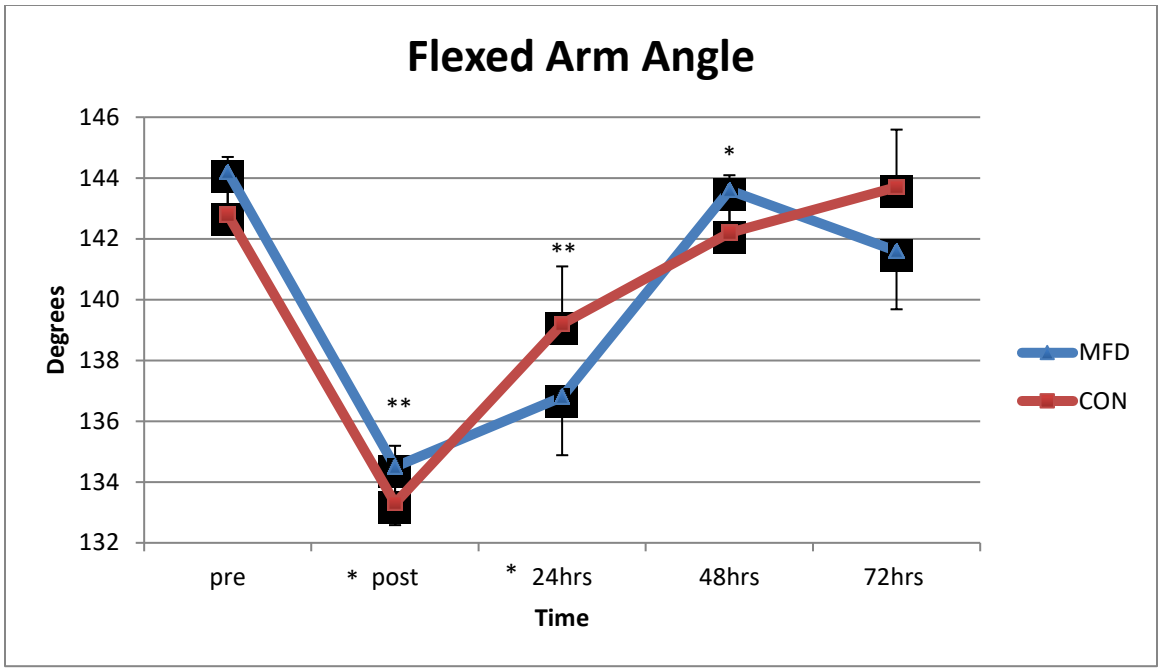

Figure 2: Flexed Arm Angle. The * indicates a main effect for time, $P < 0.001$ with marked time points different from pre, 48, and 72 hours.

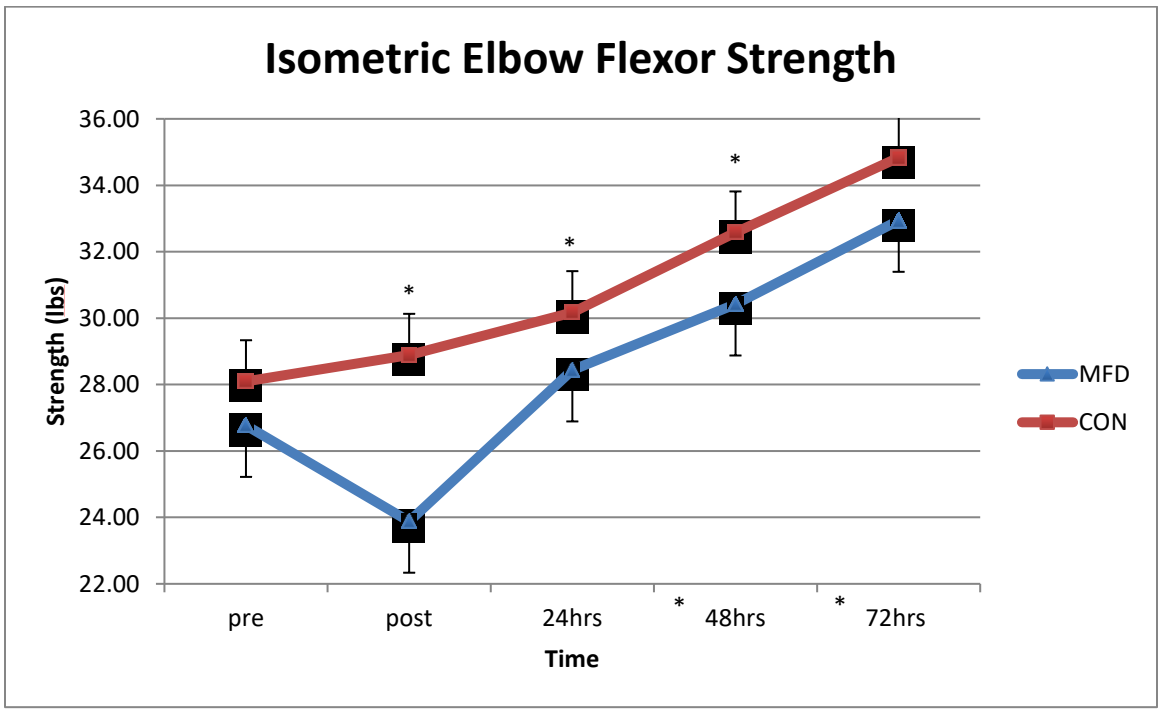

Figure 3: Isometric Elbow Flexor Strength. The * indicates a main effect for time, $P < 0.001$ with marked time points different from pre, post, and 24 hours.

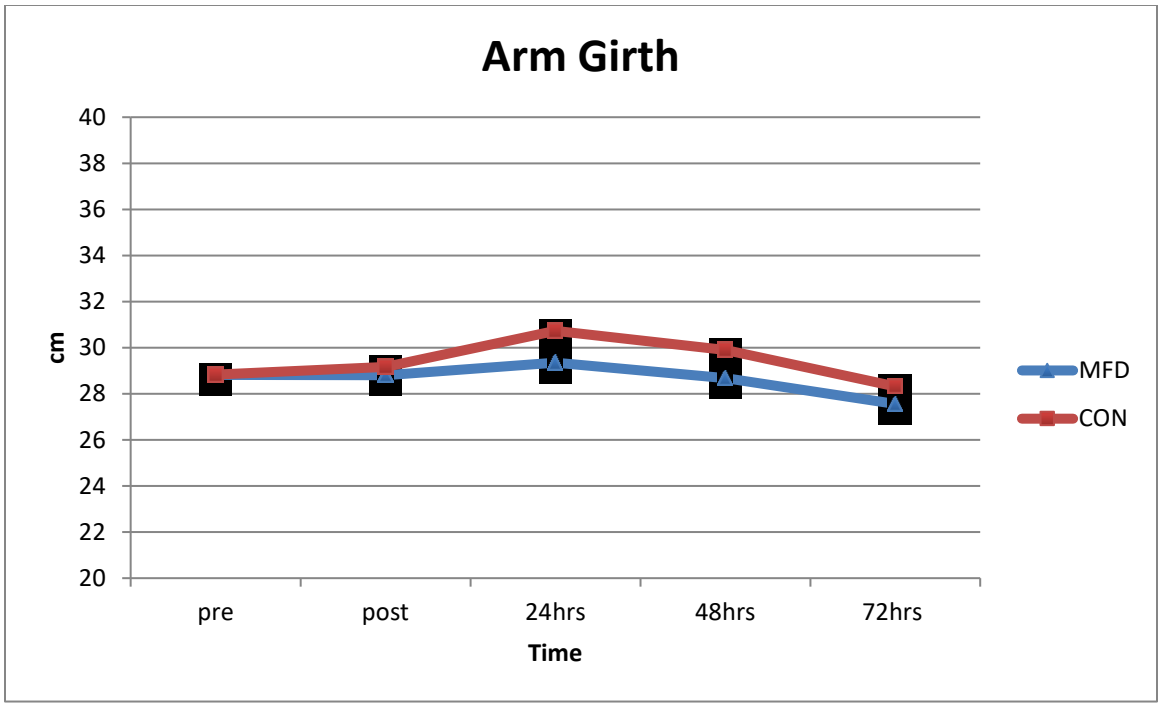

Figure 4: Arm Girth. No main effects.

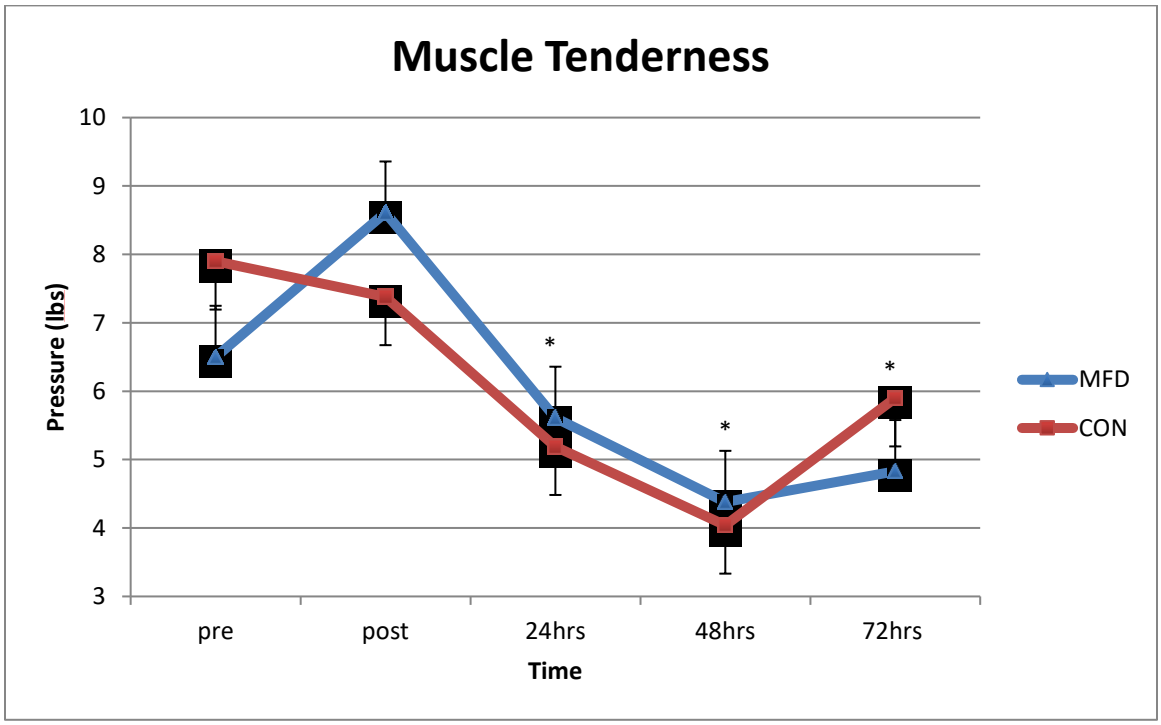

Figure 5: Muscle Tenderness. The * indicates a main effect for time, $P < 0.00$ with marked time points different from pre and post.

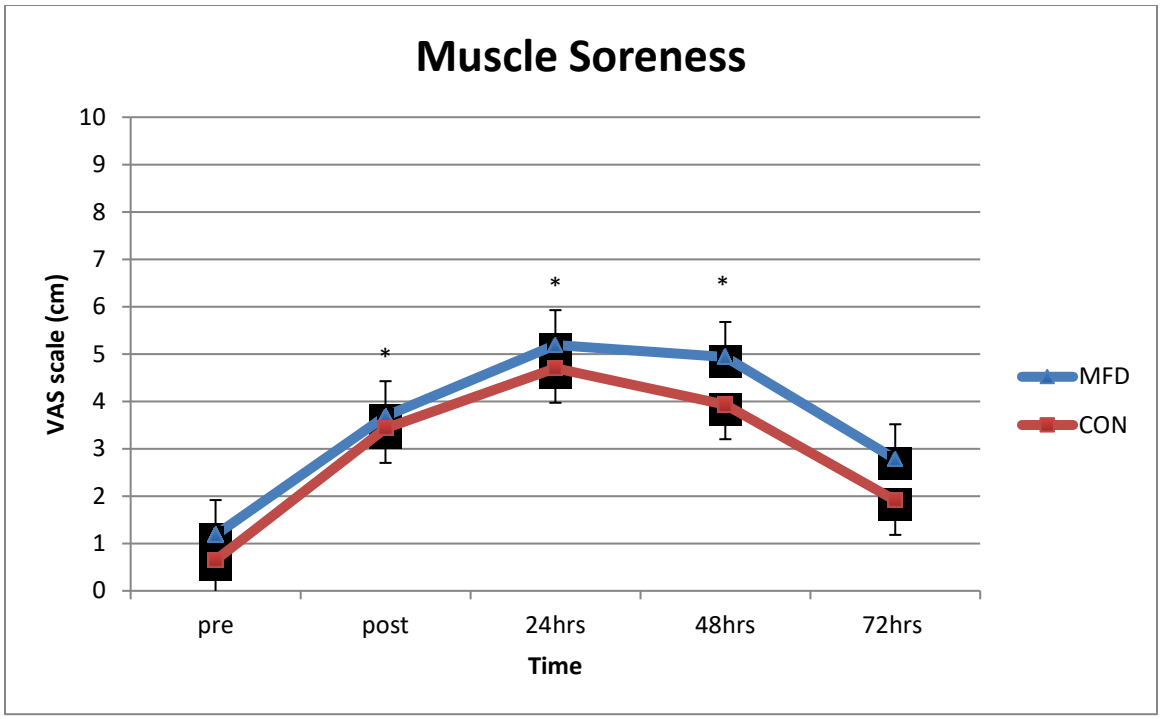

Figure 6: Muscle Soreness. The * indicates a main effect for time, $P < 0.00$ with marked time points different from pre and 72 hours.

CHAPTER V

DISCUSSION

The results of this study did not confirm the hypothesis and indicate that there is no difference in range of motion, pain, or functional strength after intervening with MFD compared to a control group immediately following EIMD. More specifically, there were no differences in relaxed arm hang angle, flexed arm angle, isometric flexor strength, arm girth, muscle tenderness, or muscle soreness following immediate use of MFD after EIMD compared to a control intervention of active flexion and extension of the biceps without cups suctioned on the biceps.

In relaxed arm hang angle (Figure 1), a measurement closer to zero degrees indicates fuller extension. In the relaxed arm hang angle, although the differences by condition were not significant, the MFD intervention yielded smaller measurements at post, 24hr, and 48hr time points. If this difference had been statistically significant or if the approximate difference of two degrees between MFD and control was clinically significant, it would indicate that MFD controlled inflammation and the associated swelling in the experimental arm than the control condition until the 72hr time point. In flexed arm angle in Figure 2, a measurement closer to 180 degrees yields fuller flexion. In the flexed arm angle, the MFD intervention yielded greater measurements post and 48hrs; however, there was greater variability in these responses, so speculation as to the effectiveness of the MFD is even more doubtful. Therefore, the flexed arm angle does not aid in confirming the reduction in swelling due to MFD intervention, as the relaxed arm hang angle does. In arm girth seen on Figure 4, a smaller measurement of diameter in centimeters yields a reduction in swelling. At all time points, MFD measurement of arm girth were less than the control measurements, designating a reduction in swelling as seen in relaxed

arm hang angle. Relaxed arm hang angle, flexed arm angle, and arm girth all impact ROM, so overall, ROM was not reduced with MFD compared to control; thus, it would suggest that the MFD did not effectively alter the inflammatory response.

For isometric elbow flexor strength in Figure 3, a greater measurement of strength yields less loss of functional strength due to EIMD responses. However, MFD produced lower measurements across all time points, meaning that the intervention did not prevent loss of functional strength as expected from EIMD. This lack of functional strength loss is likely due to the practice effect. As the subjects continued to perform this test, they discovered that they could see the amount of pounds they were exerted on the transducer, so they tried to beat their previous score no matter the soreness of their muscle.

Next, a greater measurement on the muscle tenderness scale in Figure 5 yields a greater tolerance to pressure exerted on the muscle by a dolorimeter. A greater pressure measurement equals a less pain measurement. According to Figure 6, MFD reduced pain post, 24hr, and 48hrs times. Lastly, a smaller measurement on the VAS scale yields less soreness. According to the VAS scale, zero equals no soreness and 10 equals greatest soreness. According to Figure 6, MFD produced greater measures on the VAS scale, so the intervention did not decrease muscle soreness. In all, it's concluded from the Figure that there was not a significant increase in ROM, functional strength, or decrease in pain. This mirrors the information that is present in Tables 1 and2 stating that there are no main effects of condition.

As a result of analyzing these results, it is clear that MFD did not reduce in inflammatory responses associated with EIMD, including DOMS and its associated responses. In order to explain this lack of reduction in DOMS, it is logical to analyze the control of the initial inflammatory response as a response to the primary damage introduced by exercise-induced

muscle damage. This inflammatory response to muscle damage is observed mainly in post-24hr time points. As there are no interactions across any variables, there were no significant differences between conditions at these time points. Furthermore, it's evident that the inflammatory response due to primary muscle damage was not controlled.

While there were no differences between conditions, there were differences in time points, which confirms that the EIMD protocol produced significant muscle damage. Therefore, there was primary muscle damage for MFD to act on, but that it was a matter of lack of secondary damage because the entire inflammatory response was not complete due to the timing of the intervention, the size and magnitude of the biceps muscle damage was too small, or the duration of the intervention was too short. I believed that intervening early could provide possible benefits by controlling the inflammatory response and thus reduce secondary damage. I proposed that MFD could alter the inflammatory response with the use of active movement. The inflammatory response inevitably breaks down damaged tissue; consequently, it lays down new tissue as well, but in a randomized, crisscrossed pattern. Active movement during early MFD intervention would ensure the correct alignment of the new tissue being laid down. While there is no way for MFD to reduce the secondary damage of cell membrane disruption, I believed that the correct alignment of the tissues during the initial inflammatory response would reduce the symptoms of DOMS because of the correct functional pattern of the muscle. It is evident from this current study that MFD did not reduce secondary damage nor significantly reduce the symptoms of DOMS. If this were true, there would be a reduction in loss of functional strength and ROM, and a smaller increase in pain. Perhaps if MFD were administered early in the inflammatory response to correctly align the damaged tissues, as in this current study, and later, as in previous studies, in order to continue to break down damaged tissue from the secondary

damage, there would be significant reductions in loss of strength and ROM, and even smaller increases in pain.

In regards to the research questions,, MFD does seem to be able to reduce DOMS, although not significantly in comparison to a control intervention consisting of active movement.

Implications

Based off the findings and associated analysis, MFD used on the biceps for EIMD immediately post exercise is not as beneficial as MFD used on larger muscle areas, for longer durations, and later in the repair process. However, based off previous studies, MFD is a reasonable intervention for soft tissue injuries.[5] On the other hand, the EIMD protocol did produce sufficient muscle damage, so this protocol may be used in future studies examining the effects of MFD on DOMS following exercise-induced muscle damage.

Limitations

A limitation and likely explanation for the lack of difference between conditions is the timing of the MFD intervention. In all previous studies, MFD was used later in the repair process, such as to already injured tissue, or to non-damaged tissue. As a result, the timing immediately after exercise-induced muscle damage may be too premature to yield statistically significant results. The timing may be critical because of the amount of damage that has formed. When MFD is used so early, there may not be enough muscle damage for it to break down and repair in order to produce statistically significant results. Additionally, the inflammatory response as a result of DOMS may produce greater and more noticeable changes in ROM, strength, and pain. In this study, MFD was used to early to have any direct effect on the damage resulting from DOMS.

Another limitation and possible explanation for the lack of difference between conditions is the site of MFD. One issue with the biceps area is the size of the muscle. In previous literature, MFD was used on the hamstrings and three rotator cuff muscles, which both have a greater mass than the biceps. The biceps may not have enough mass for MFD to produce statistically significant results for the muscle damage. Similarly, the hamstrings and three rotator cuff muscles have a greater surface area than the biceps, which allows for more cups to be used during the intervention. Using only two cups on the biceps during MFD may not be adequate decompression to produce statistically significant results in muscle damage improvement.

Lastly, a limitation and possible explanation for the lack of difference between conditions is the duration of the intervention. In previous literature, the duration of treatment was approximately five minutes with the cups static followed by approximately two minutes of active movement. In this study, the cups were never static; subjects performed active movement immediately after the initial seal for two minutes. This lack of static cup time may have limited the amount of blood flow into the site of damage. Furthermore, there may have been a lack of leukocytes to break down the damaged tissue. If this were the case, the active movement would not have had enough tissue to mobilize and be laid down in the proper alignment to improve muscle function and reduce pain.

A shorter duration was used in this study because of subject comfort levels. During pilot testing, two minutes was determined to be a bearable duration. Longer durations were not as bearable in this study because of the close proximity of the cups. The resulting sensation during active flexion and extension was uncomfortable to the point of wanting to quit. Therefore, in order to maintain subjects for the full duration of the study, it was decided to use a bearable, yet possibly effective duration. Additionally, since such a short duration was used, no static cup time

was included because of the desired emphasis on the difference between cupping and MFD. In order to differentiate between traditional cupping and MFD, specifically the active movement component, it was decided that this would be the most logical protocol.

Future Research

In order to determine what is the main factor affecting the success of MFD, there are multiple future directions that can be taken. First, using for cups on the biceps myofascial track will determine if the surface area is the main factor. Secondly, increasing the decompression duration will determine if the length of MFD is the main factor. Furthermore, manipulations may be made between the ratios of length of static cupping vs. active movement. Next, MFD could be used on the biceps as a therapeutic modality for chronic pain in order to determine if the amount or extent of muscle damage is the main factor. Similarly, MFD used in the same protocol as this study may be used on the hamstring to determine whether the size or surface area of the muscle is a factor in treatment efficacy. Lastly, using the same protocol as this study, but with uninjured, non-athletes who typically use the same workout protocol may reveal what factors, such as ROM, functional strength, or pain MFD affects the most since the participants will be more standardized.

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APENDICES

APPENDIX A

McKenzie Smith
Dr. Joel MItchell
Dr. Stepanie Jevas
Dr. Andreas Kreutzer

It gives me great pleasure to inform you that your protocol "Effect of myofascial decompression on delayed onset muscle soreness following high intensity strength training " (DRB-1802-62) has been approved with minimal risk by the Texas Christian University Institutional Review Board for the period from February 22, 2018 to February 21, 2019.

I wish you the best on your research endeavors.

Best,

Dr. Morrison G. Wong
Professor of Sociology
TCU Institutional Review Board

Texas Christian University
Fort Worth, Texas

CONSENT TO PARTICIPATE IN RESEARCH

Title of Research: Effect of Myofascial Decompression on Delayed Onset Muscle Soreness Following High Intensity Strength Training

Funding Agency/Sponsor: None

Study Investigators:
Mackenzie Smith

Joel Mitchell, Ph.D.

Stephanie Jevas, Ph.D., ATC, LAT

Andreas Kreutzer

What is the purpose of the research?
The purpose of this research is to determine the effect of myofascial decompression on delayed onset muscle soreness following high intensity strength training.

How many people will participate in this study?
Approximately 10 participants.

What is my involvement for participating in this study?
On the first day, prior to experimental testing, the purpose and procedures will be explained to you, followed by an explanation and subsequent completion of the consent document, the medical history questionnaire, and a HIPAA form.

On a subsequent day, experimental testing will begin with one arm randomly assigned to experimental testing, with the other arm designated as the control. Next, a series of baseline measurements will be completed. These measures will include: 1) your relaxed and flexed arm angle measured at the elbow joint, 2) isometric elbow flexor strength, which is how hard you can try to flex your bicep against resistance, 3) muscle soreness, 4) muscle tenderness, and 5) arm girth. Relaxed arm angle will be obtained by having you stand with your arms straight hanging at your side and palms facing forward. Flexed arm measurements will be obtained by instructing you to flex at the elbow and try to touch your shoulder while keeping your elbow tucked towards your side. Muscle strength will be measured using a modified arm curl bench. The arm curl

bench is equipped with a lever arm and a handgrip that are attached to a load cell. You will have their elbow joint positioned at 90 degrees, and will perform two maximal efforts by trying to curl the lever arm with the handgrip, which is locked in position. Each maximal effort will last 4 seconds with a rest period of 30 seconds between trials. Muscle soreness will be recorded by having you mark on a 10-cm visual analogue scale with the zero end anchored with the descriptor, "no soreness" and the 10-cm end anchored with the descriptor, "extreme soreness". This measure will be made once with the muscle at rest and again as you move your arm through an active range of motion. Muscle tenderness will be determined using a dolorimeter, a device that measures the force applied to the muscle necessary to elicit a mild pain response you. Finally, muscle girth will be measured at the maximum diameter of the muscle belly using a tape measure.

Following baseline testing, the eccentric exercise bout will be performed. You will perform five sets of ten maximal eccentric contractions at a speed of 30 degrees per second from 60 to 180 degrees of flexion. You will begin with your forearm flexed at 60 degrees, and your forearm will be forcibly extended to 180 degrees in four seconds. A member of the research team will move your arm through the concentric phase of the contraction to the starting position of 60 degrees at a speed of 10 degrees per second so no concentric contraction is performed. A one-minute rest will separate each of the five sets.

Immediately post-exercise, the MFD technique will be performed on your experimental arm. Two cups of various sizes, depending on the size of your arm, will be placed on your bicep, specifically on the muscle belly and near the crease of your elbow. A negative pressure will be applied via the pump with three pumps to ensure the initial seal, and up to five total pumps to generate the necessary tissue lift. The negative pressure developed within the cup will be measured using a gauge tied into the cup system, allowing us to standardize the negative pressure across all subjects. The cups will remain static on your skin while you're relaxed for five minutes. Then, you will actively move your experimental arm through elbow flexion and extension with your palm facing up with the cups still place for two minutes at a programmed speed. For the control condition, the same baseline measures and eccentric exercise protocol will be followed; however, no MFD protocol will be implemented. You will, however, perform the same active movements with the control arm to replicate the 2 minutes of movement used in the MFD protocol.

Immediately after the MFD protocol, the same battery of tests used in the baseline testing will be administered. These tests will also be conducted at 24, 48, 72 hours of recovery from the exercise protocol. After the first condition, there will be a washout period of one-week, followed by the exact same protocol, but with the opposite arm and the alternate condition. In total, eight visits are required for approximately a total of a two-hour time commitment. At the completion of the study, you will receive your individual results.

How long am I expected to be in this study for and how much of my time is required?
There will be a total of eight visits to participate in the study. The first visit will be about 45min, the second, third and fourth will be about 10min, the fifth will be about 30 min, and the sixth, seventh, and eighth will be about 10min as well. In total, participation in this study should be about two total hours of your time.

What are the risks of participating in this study and how will they be minimized?
Potential risks from participation in the high intensity strength training portion of this study will be muscle strains, severe muscle fatigue, and delayed onset muscle soreness. In addition, potential risks from myofascial decompression will be mild to serious bruising and/or welts on the skin in areas of placement of the cups, pain or pinching from the cups, and discomfort from the skin sucked into the cup. in areas of placement of the cups. Risks will be minimized by a professor trained in using the eccentric bicep machine monitoring you while you perform the strength exercise and a certified athletic trainer who has completed the required myofascial decompression classes will place the cups and administer the myofascial decompression therapy for you.

What are the benefits for participating in this study?
You will be able to decide if myofascial decompression is a therapy that you would like to utilize in the future so you can relieve muscle soreness faster and return to optimal performance level as soon as possible after a strenuous training session or performance.

Will I be compensated for participating in this study?
No.

What is an alternate procedure(s) that I can choose instead of participating in this study?
There is no alternate procedure other than simply not participating.

How will my confidentiality be protected?
You will be assigned a research participant number and all your data will be entered in our computers using that number. In addition, all research reports will use averages of the entire group without specific reference to a participant's individual data.

Is my participation voluntary?
Yes.

Can I stop taking part in this research?
Yes, you may withdraw at any time.

What are the procedures for withdrawal?
Notify the primary investigators or a member of the research team by e-mail, phone, or in person.

Will I be given a copy of the consent document to keep?
Yes.

Who should I contact if I have questions regarding the study?
Mackenzie Smith. Phone: (913) 484-3239. E-mail: Mackenzie.smith@tcu.edu
Joel B. Mitchell, Ph.D. Phone: (817) 257-7665. E-mail: j.mitchell@tcu.edu

Who should I contact if I have concerns regarding my rights as a study participant?
Dr. Cathy R. Cox, Chair, TCU Institutional Review Board, (817) 257-6418, c.cox@tcu.edu.

Dr. Bonnie Melhart, TCU Research Integrity Office, (817) 257-7104, b.melhart@tcu.edu.
Dr. Tim Barth, Associate Dean for Research & Graduate Studies and Co-Chair, TCU Institutional Review Board, (817) 257-6427, t.barth@tcu.edu

Your signature below indicates that you have read or been read the information provided above, you have received answers to all of your questions and have been told who to call if you have any more questions, you have freely decided to participate in this research, and you understand that you are not giving up any of your legal rights.

Participant Name (please print): _____

Participant Signature: _____
Date:_____

Investigator Name (please print):_____Date:_____

Investigator Signature: _____
Date:_____

PROTECTED HEALTH INFORMATION AUTHORIZATION FORM

Researchers from the study "Effect of myofacial decompression on delayed onset muscle soreness following high intensity strength training" would like **your permission** to **use your health information** which will be gathered as a part of this study.
Ma
The following **health information** will be **gathered** from you:

Medical history

The **names of the TCU researchers** who will gather this information from you are (insert the names of all TCU researchers starting with the lead researcher):

Mackenzie Smith
Joel Mitchell, Ph.D.
Stephanie Jevas, Ph.D., ATC, LAT
Andreas Kreutzer

Your **health information may be shared** with others who are working with the TCU researchers on this study, institutes that are paying for this study or involved in any other way, or as required by law. The names of these other researchers (include name, affiliation, and role in the study) or institutions (name and role in the study) are listed below.

The TCU researchers and other researchers who work with TCU will **protect** your **health information** in the following ways:

- Your health information will be kept **private**
- Your **name or any other identifying information will not** be made known
- Your health information may be shown in research papers or meetings **without any information about you** that will link it to you.
- Your health information will be given a **special code** for security
- Your health information will be **grouped together with other people's** health information to form an average
- Your health information will be **locked in a cabinet** and kept safe

You can agree or not agree to sign this form. If you agree to sign this form but change your mind, you can **choose to stop** being in the study at any time. If you decide to stop being in the study, you will need to contact the researcher (insert the name, telephone, and e-mail of the PI): Mackenzie Smith, 913-484-3239, mackenzie.smith@tcu.edu

You will be **given a copy** of this form to keep.

If you have any **questions or concerns** about **your rights** as a study participant, you can contact:
Dr. Cathy R. Cox, Chair, TCU Institutional Review Board, (817) 257-6418, c.cox@tcu.edu.
Dr. Bonnie Melhart, TCU Research Integrity Office, (817) 257-7104, b.melhart@tcu.edu.
Dr. Tim Barth, Associate Dean for Research & Graduate Studies and Co-Chair, TCU Institutional Review Board, (817) 257-6427, t.barth@tcu.edu

By signing your name below, **you are saying** that you **understand what is being said in this form**, you have **received answers** to all your questions, you have **freely agreed to sign** this form, you have been told **who to contact** if you have questions regarding **your rights** as a participant, and you have **allowed TCU to gather, use, and share your health information** as described in the form.

Participant's Name (please print): _____

Participant's Signature: _____ **Date:** _____

Investigator's Signature: _____ **Date:** _____

Legal Representative of Research Participant (if applicable):

Legal Representative's Name (please print): _____

Relationship to research participant: _____

I certify that I have the legal authority as a _____ (e.g., parent, legal guardian, person with legal power of attorney, etc.) to make this authorization on behalf of the research participant named above.

Signature of the Legal Representative: _____ Date: _____

Investigator's Signature: _____ Date: _____

Certificate of Completion

The National Institutes of Health (NIH) Office of Extramural Research certifies that **Mackenzie Smith** successfully completed the NIH Web-based training course "Protecting Human Research Participants".

Date of completion: 11/26/2016.

Certification Number: 2244784.

Certificate of Completion

The National Institutes of Health (NIH) Office of Extramural Research certifies that **Joel Mitchell** successfully completed the NIH Web-based training course "Protecting Human Research Participants".

Date of completion: 11/20/2014

Certification Number: 1622628

Certificate of Completion

The National Institutes of Health (NIH) Office of Extramural Research certifies that **Stephanie Jevas** successfully completed the NIH Web-based training course "Protecting Human Research Participants".

Date of completion: 09/12/2013

Certification Number: 1240734

COLLABORATIVE INSTITUTIONAL TRAINING INITIATIVE (CITI)

HUMAN SUBJECT PROTECTION COURSE CURRICULUM COMPLETION REPORT

Printed on 08/13/2014

LEARNER	Andreas Kreutzer (ID: 4283114)
DEPARTMENT	IMC/IEEM
PHONE	2143454619
EMAIL	akreutzer82@gmail.com
INSTITUTION	University of Texas Southwestern Medical Center
EXPIRATION DATE	

HUMAN SUBJECT PROTECTION COURSE

COURSE/STAGE:	Basic Course/1
PASSED ON:	08/08/2014
REFERENCE ID:	13661968

REQUIRED MODULES	DATE COMPLETED
Introduction	08/07/14
Why Study HSP?	08/07/14
History	08/08/14
Research Standards	08/08/14
Statutory Framework	08/08/14
Regulatory Oversight	08/08/14
Definitions	08/08/14
What is an IRB?	08/08/14
IRB Membership	08/08/14
Criteria for Approval	08/08/14
Assessment of Risks and Benefits	08/08/14
Types of IRB Review	08/08/14
Informed Consent	08/08/14
Recruitment of Subjects	08/08/14
Documentation of Consent	08/08/14
Elements of Consent	08/08/14
Special Issues Regarding Informed Consent	08/08/14
Capacity to Consent	08/08/14
Research with Children	08/08/14
Determining Voluntariness	08/08/14
Investigator Responsibilities	08/08/14
Subject Selection	08/08/14
Research with Prisoners	08/08/14
Other Special Issues	08/08/14
Continuing Review	08/08/14
Modifications to Approved Research	08/08/14
Adverse Event Reporting	08/08/14
Data and Safety Monitoring	08/08/14
Human Subject Protection	08/08/14

For this Completion Report to be valid, the learner listed above must be affiliated with a CITI Program participating institution or be a paid Independent Learner. Falsified information and unauthorized use of the CITI Program course site is unethical, and may be considered research misconduct by your institution.

Paul Braunschweiger Ph.D.
Professor, University of Miami
Director Office of Research Education
CITI Program Course Coordinator

BASIC LIFE SUPPORT

BLS Provider

American Heart Association®

MacKenzie Smith

has successfully completed the cognitive and skills evaluations in accordance with the curriculum of the American Heart Association Basic Life Support (CPR and AED) Program.

Issue Date
12/7/2017

Training Center Name
Save A Life

Training Center ID
TX05110

Training Center Address
3044 Old Denton Rd #111 162
Carrollton TX 75007 USA

Training Center Phone Number
(972) 672-0349

Recommended Renewal Date
12/2019

Instructor Name
Ben Timson

Instructor ID
08150353426

eCard Code
185501732240

QR Code

To view or verify authenticity, students and employers should scan this QR code with their mobile device or go to www.heart.org/cpr/mycards.