FOAM ROLLING AS A RECOVERY TOOL FOLLOWING AN INTENSE BOUT OF PHYSICAL ACTIVITY

GRAHAM ZENO MACDONALD

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By

© Graham Zeno MacDonald

A thesis submitted to
The School of Graduate Studies
in partial fulfillment of the requirements for the degree of

Masters of Science in Kinesiology
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ABSTRACT

Purpose:

Understand the effectiveness of foam rolling as a recovery tool following exercise induced muscle damage (EIMD), analyzing: muscle soreness, dynamic and passive range of motion (ROM), along with evoked and voluntary neuromuscular properties.

Methods:

20 male subjects with 3+ years of strength training experience were randomly divided into either the control (CON) (n=10) or foam rolling (FR) (n=10) group. All subjects followed the same testing protocol. The only between group difference was that the FR group performed a 20-minute foam rolling exercise protocol at the end of the testing session at post-test 0, 24, and 48 hours (POST-0, POST-24, POST-48). Subjects participated in 5 testing sessions: [1] orientation and 1 repetition maximum (1RM) back squat, [2] pre-test measurements (PRE), 10 x 10 squat protocol (weight: 60% 1RM, tempo: 4,1,1,1) with 2 minutes rest between sets, and post-test measurements (POST-0), along with measurements at: [3] POST-24, [4] POST-48, and [5] POST-72. Test measurements included: thigh girth, muscle soreness, range of motion (ROM), evoked and voluntary contractile properties, vertical jump, along with perceived pain (FR-pain) and reaction forces (FR-force) while foam rolling.

Results:

Thigh girth showed no substantial between group differences at all time points. FR substantially reduced muscle soreness at all time points while substantially improving

ROM. FR negatively affected evoked contractile properties (twitch force, rate of force development, and potentiated twitch force) with the exception of half-relaxation time (½RT) and electromechanical delay (EMD). ½RT showed no substantial between group differences at all time points, while FR substantially improved EMD. Voluntary contractile properties showed no substantial between group differences for all measurements besides voluntary muscle activation, with FR substantially improving muscle activation at all time points. FR improved functional movement, with substantial between group differences in vertical jump height. When performing the five FR exercises at the three time points (POST-0, POST-24, POST-48), subjects FR-force ranged between 26-46kg (32-55% of subjects' body weight) with FR-pain measurements (based on NRS) ranging between 2.5-7.5 pts.

Conclusion:

The most important findings of the present study were that FR was beneficial in attenuating muscle soreness while improving vertical jump height, muscle activation, and passive and dynamic ROM in comparison to CON. FR negatively impacted a number of evoked contractile properties of the muscle, except for ½ RT and EMD, indicating that FR benefits are primarily accrued through neural responses and connective tissue.

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CO-AUTHORSHIP STATEMENT

This thesis was developed under the supervision of Dr. David Behm and Dr. Duane Button from the School of Human Kinetics and Recreation at Memorial University of Newfoundland, St. John's, Canada, along with the help of Dr. Eric Drinkwater from the School of Human Movement Studies at Charles Sturt University, Bathurst, New South Wales, Australia. Dr. Behm and Dr. Button played a fundamental role in my topic development, research design, proof reading and editing of my thesis, along with providing technical support in the lab. Dr. Drinkwater played a vital role in helping with the statistical analysis of my thesis. I was personally responsible for subject recruitment, research design, data collection, data analysis, statistical analysis, and final write-up of the enclosed research article "Foam Rolling as a Recovery Tool Following an Intense Bout of Physical Activity".

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CHAPTER 1: INTRODUCTION

1.1 BACKGROUND OF STUDY:

Over the past decade, foam rolling (FR) has become commonly used as a recovery tool following an intense bout of physical activity, believed to correct muscular imbalances, alleviate muscle soreness, relieve joint stress, improve neuromuscular efficiency, and joint range of motion (1, 3). FR has been implemented into a number of different rehabilitation and training programs to help treat myofascial adhesions, enhance joint ROM, and promote soft-tissue extensibility and optimal skeletal muscle functioning (1, 3, 5). FR works under the same principles as myofascial release, using both direct pressure and slow-sweeping pressure to promote soft-tissue extensibility while breaking up muscle adhesions and spasms (6). The difference between FR and myofascial release is that instead of a therapist applying pressure, a patient/client uses their own body weight on a foam roller to exert pressure on the opposing soft-tissue. With the patient/client being able to implement a FR exercise protocol on their own, it makes FR an easy, time efficient and cost effective way treat the soft-tissue of the body.

From the recreationally active to the elite athlete, many individuals commonly experience exercise induced muscle damage (EIMD) resulting in delayed on set muscle soreness (DOMS) following an intense bout of physical activity. EIMD is characterized by muscle soreness and a decrease in muscular strength and ROM do to temporary soft-tissue damage. (2, 8). In response to injury, fascial tissue is believed to lose its elasticity and becomes dehydrated, causing fibrous adhesion. Fibrous adhesions prevent normal muscle mechanics, decreases soft-tissue extensibility and causing pain either at the site of the malfunction, or remotely at other sites. (3, 7).

Although the theory behind FR is well understood among therapists, there has been limited scientific research conduct on FR to support the believed physiological and musculoskeletal benefits obtain by performing FR. Thus, it is prudent to evaluate the effectiveness of FR as a recovery tool following an intense bout of physical activity and analyze the mechanisms of how FR effects muscle soreness, joint range of motion, vertical jump, and voluntary and evoked contractile properties following EIMD.

1.2 SIGNIFICANCE OF STUDY:

Foam Rolling (FR) has become a common technique used to treat myofascial adhesions, enhance joint ROM, and promote soft-tissue extensibility and optimal skeletal muscle functioning (3). Although several researchers have discussed the applicability of FR, there is limited to no clinical data demonstrating the efficacy and mechanisms of the FR technique (3, 4, 6).

The objective of the present study is to determine the effects of implementing a FR exercise protocol on the recovery process following EIMD, through the analysis of: muscle soreness, thigh girth, joint range of motion, vertical jump height, and voluntary and evoked contractile properties.

1.3 HYPOTHESES:

Our hypotheses are that foam rolling will:

1) Aid in the recovery process from EIMD by reducing muscle soreness.
2) Aid in the recovery process, demonstrating less decrements in vertical jump height in the 72-hour period following EIMD.
3) Reduce decrements in joint range of motion at the knee (QP-ROM) and hip (HP-ROM & HD-ROM) in the 72-hour period following EIMD.
4) Have no effect on neuromuscular performance measures in the recovery process in the 72-hour period following EIMD.

1.4 REFERENCES:

1. Barnes MF. The basic science of myofascial release: morphologic change in connective tissue. *Journal of Bodywork & Movement Therapies*. 1997;1(4):231-8.

2. Cheung K, Hume P, and Maxwell L. Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine*. 2003;33(2):145-64.

3. Curran PF, Fiore RD, and Crisco JJ. A comparison of the pressure exerted on soft tissue by 2 myofascial rollers. *Journal of Sport Rehabilitation*. 2008;17(4):432-42.

4. Fredericson M, and Wolf C. Iliotibial band syndrome in runners: innovations in treatment. *Sports medicine*. 2005;35(5):451-9.

5. MacDonald G, Penney M, Mullaley M, Cuconato A, Drake C, Behm DG, and Button DC. An acute bout of self myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *Journal of Strength and Conditioning Research*. 2013;27(3):812-21.

6. Paolini J. Review of Myofascial Release as an Effective Massage Therapy Technique. *Athletic Therapy Today*. 2009;14(5):30-4.

7. Swann E, and Graner SJ. Uses of manual-therapy techniques in pain management. *Athletic Therapy Today*. 2002;7(4):14-7.

8. Torres R, Ribeiro F, Duarte JA, and Cabri JMH. *Evidence of the physiotherapeutic interventions used currently after exercise-induced muscle damage: systematic review and meta-analysis* 2012. 101-14 p.

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CHAPTER 2: REVIEW OF LITERATURE

2.1 INTRODUCTION:

Foam rolling (FR) is commonly implemented by therapists and fitness professionals as a recovery and maintenance tool to aid in the healing process of soft-tissue. Advocates (9, 25) claim that FR: corrects muscular imbalances, alleviates muscle soreness, improves neuromuscular efficiency, relieves joint stress, and enhance joint range of motion (ROM). FR has been implemented into a number of different rehabilitation and training programs to help promote soft-tissue extensibility, enhance joint ROM, relieve pain and muscle soreness, and promote optimal skeletal muscle functioning (9, 25, 60).

Although FR is strongly advocated and has been commonly implemented, there have only been three peer-reviewed research articles published to date. Pearcey et al. (76) examined the effects of FR after an intense exercise protocol on pressure pain threshold and dynamic performance measures, concluding that FR is an effective method in reducing delayed onset muscle soreness (DOMS) and associated performance decrements in sprint time, power, and dynamic strength-endurance. MacDonald et al. (60) investigated the effects of acute FR prior to physical activity and demonstrated that FR had no effects on neuromuscular performance, while significantly increasing ROM at 2 and 10 minutes post-FR by 10 and 8%, respectively. Curran et al. (25) determined that a higher density foam roller significantly increased soft tissue pressure and isolated the soft tissue contact area, potentially increasing the effects foam rolling has on improving soft tissue health.

FR has been referred to as a form of self-induced myofascial release (SMR) or self-massage (60, 75). SMR is believed to work under the same principles as myofascial release (MFR). MFR therapy is a manual therapy technique developed by John F. Barnes (10) and is believed to help reduce restrictive barriers or fibrous adhesions seen between layers of fascial tissue. The difference between the two techniques is that instead of a therapist providing manual therapy to the soft tissue, an individual uses their own body weight on a foam roller to exert pressure on the soft-tissue. The SMR technique involves small undulations back and forth over a dense foam roller, starting at the proximal portion of the muscle, working down to the distal portion of the muscle or vice versa (74). The small undulations place direct and sweeping pressure on the soft-tissue, believed to stretch the tissue, generating friction between the soft-tissue of the body and the foam roller. The friction generated from the undulations is believed to cause warming of the fascia, promoting the fascia to take on a more fluid like form (known as the thixotropic property of the fascia), allowing fibrous adhesions between the layers of fascia to be broken up, restoring soft-tissue extensibility (81).

In the past decade, therapists and fitness professionals have implemented foam rolling as a recovery and maintenance tool to aid in the process of soft-tissue healing in response to injury. Soft-tissue injuries often occur as a result of excess stress placed on the tissue from physical activity or from the tissue being under-stressed due to inactivity in an individuals daily routine (8). It is believed that connective tissue (fascia) surrounding the muscle is the common sight of injury. Fascia is believed to stiffen due to injury or inactivity, becoming less pliable, resulting in movement pattern restrictions and alterations in muscular force (8). Foam rolling is frequently implemented into different rehabilitation and training programs to address these fascial restrictions and promote soft-tissue extensibility, potentially enhancing joint ROM and promoting optimal skeletal muscle function.

With no current interventions having the ability to prevent exercise induced muscle damage (EIMD) and the subsequent onset of DOMS, and with eccentric activity being an integral part of everyday living, we must look at therapies and mechanisms to help treat EIMD. EIMD and the subsequent onset of DOMS can negatively alter an individual's ability to perform daily activities and/or their willingness to continue participation in physical activity, therapeutic exercise, and/or sport. Finding treatment methods to effectively alleviate EIMD and DOMS may increase participation in daily activities and may allow an individual to maintain their adherence to participate daily in physical activity.

With foam rolling commonly being implemented as a recovery tool following an intense bout of physical activity by therapist and fitness professionals, the purpose of the present literature review is to first gain an in-depth understanding of what EIMD is by looking at: the effect EIMD has on the body, the theories of how EIMD is induced, and the underlying mechanisms of the bodies recovery process following EIMD. Upon understanding EIMD, the next step is to gain insight into the effects massage has on the recovery process following EIMD along with other potential mechanisms that have been demonstrated to aid in the recovery process following EIMD. An in-depth analysis of massage is needed as foam rolling is commonly referred to as a form of self-massage. With foam rolling research still very much in an exploratory phase, the vast knowledge base provided with massage research will act as starting point to base the hypotheses in this thesis regarding the effects foam rolling may have in the recovery process following EIMD.

2.2 EXERCISE INDUCED MUSCLE DAMAGE: (EIMD)

2.2.1 INTRODUCTION:

From the recreationally active to the elite athlete, many individuals commonly experience EIMD resulting in DOMS following an intense bout of physical activity. EIMD is characterized by muscle soreness, muscle swelling, temporary muscle damage, an increase in intramuscular protein and passive muscle tension, and a decrease in muscular strength and ROM (18, 90). In addition to these responses, EIMD can affect neuromuscular performance by reducing shock attenuation and altering muscle sequencing and recruitment patterns, potentially placing unaccustomed stress on muscle tendons and ligaments (18). There are a number of proposed theories regarding the mechanisms of DOMS. The bulk of the literature reports that high mechanical stress placed on the myofibrils (most commonly seen during eccentric exercise) damages both muscle and connective tissue. This tissue damage subsequently triggers an acute inflammatory response consisting of edema and inflammatory cell infiltration that leads to a loss of cellular homeostasis, particularly due to high intracellular calcium concentrations (90). Sarcomere damage, calcium accumulation, protein degradation, and osmotic pressure all combine to sensitize nociceptors and other pain receptors, causing the sensation of DOMS (18). Methods that have shown potential benefits in treating symptoms of EIMD include: cryotherapy (23, 43, 90), light exercise (23, 90), nutritional supplementation (51), and compression (18, 23). Although these therapies have shown to be beneficial in treating EIMD symptoms, the contrary has also been demonstrated in the literature (18, 23, 43, 90). Although these methods have shown to be beneficial in treating EIMD symptoms, no one therapy has proven to be beneficial in treating the full array of symptoms often present with EIMD. Varying results demonstrated in the literature can be attributed to the varying EIMD protocols, along with the heterogeneity amongst studies assessing a given therapy in relation to the: dose, frequency, and intensity of the intervention.

2.2.2 EFFECTS OF EIMD:

2.2.2.1 MUSCLE SORENESS: (DOMS)

Muscle soreness is common following a bout of unaccustomed physical activity, especially from an unaccustomed activity involving eccentric contractions (43). The degree of discomfort (muscle tenderness – severe debilitating pain), or the intensity of the muscle soreness experienced depends on the duration the muscle fibers spend under-tension for a given bout of physical activity, which factors in the intensity, duration, and the type of activity. Eccentric activities are known to elicit greater muscle soreness due to the nature of the muscular contraction. An eccentric muscular contraction causes the muscle to elongate while under tension as a result of the opposing force placed on the muscle being greater than that of the force being produced by the muscle (51). Compared to concentric muscle contractions, eccentric contractions are known to cause greater muscle soreness since less muscle fibers are recruited to resist a given weight causing the recruited muscle fibers to be placed under higher tensile forces, increasing muscle fiber damage (71).

Muscle soreness can be divided into two categories, acute and DOMS. Acute muscle soreness is categorized as the soreness felt during the final stages of a fatiguing bout of physical activity, generally the result of muscular ischemia (29) along with the accumulation of metabolic waste products (35). DOMS refers to the muscle soreness occurring post-exercise. DOMS is the result of unaccustomed mechanical stress placed on the muscle resulting in muscle and connective tissue damage. Muscle and connective tissue damage leads to muscle edema and inflammation leading to further sarcomere damage, protein degradation, and the subsequent onset of DOMS (47) which generally reaches its peak intensity between 24-48 hours (18, 23), and generally subsides within 5-7 days post exercise (2, 3, 18).

2.2.2.2 NEUROMUSCULAR IMPAIRMENTS:

Neuromuscular impairments as a result of EIMD have been shown to cause a loss in proprioceptive functioning in the days following an intense bout of physical activity (78). These losses included attenuated: joint range of motion (ROM), muscular strength (67), joint-angle perception (78), and force perception

(78). Other studies have shown EIMD to cause losses in maximal voluntary force production (21, 50, 62, 67, 71), decreased muscle activation (39, 62, 77), and increased EMG:force ratios (58). Upon analysis of voluntary force production, Gibala et al. (39) and Martin et al. (62) results demonstrated that following a bout of eccentric exercise, maximal voluntary contractile force was significantly decreased in comparison to pre-test values at 24, 48, 72, and 96 hours post-exercise. Howatson et al. (50) also found significant decreases in maximal voluntary force production at post-48 hours. Newham et al. (67) and Prasartwuth et al. (77) both found decreases in maximal voluntary force immediately post-exercise of ~50% and 62 ± 3% respectively, with Newham et al. (67) and Prasartwuth et al. (77) both demonstrating maximal voluntary force production remaining below pre-exercise value for the entire time frame of the study.

Gibala et al. (39), Martin et al. (62), and Prasartwuth et al. (77) all showed a significant decrease in muscle activation immediately post-exercise, returning to baseline measures within 24 hours. All studies (39, 62, 77) showed no significant differences in muscle activation at 24, 48, 72, or 96 hours post exercise. Komi et al. (58) showed EMG recovery following eccentric exercise to remain below pre-test values up until 48 hours post-exercise, stating that substantially more neural activation is needed for a given force production in the days following EIMD in comparison to pre-test measures.

An evoked twitch is utilized for the analysis of electromechanical delay (EMD) (50, 95), rate of force development (RFD) (39, 62), twitch force (TF)

(39, 62, 77), and half relaxation time (½RT) (39). Zhou et al. (95) found that repeated isometric contractions significantly increased EMD, requiring approximately 10 minutes to recover after exercise. Howatson et al. (50) conducted a study looking at the effects of EIMD on EMD over a 96 hour period post exercise, taking measurements at 48 hours and 96 hours. While EMD was not significantly longer at 48 hours, EMD was significantly longer in duration at 96 hours. Howatson et al. (50) stated that EIMD induces alterations in EMD beyond the apparent recovery of maximal voluntary contractions, likely being attributed to post synaptic events, promoting the potential use of EMD as a tool for exercise prescription and recovery following EIMD. Gibala et al. (39) and Martin et al. (62) results demonstrated that following eccentric exercise RFD remained significantly lower than pre-exercise values at 24, 48, 72, and 96 hours post-test. Gibala et al. (39) and Prasartwuth et al. (77) results demonstrated that following eccentric exercise TF remained significantly lower than pre-exercise values at 24, 48, 72, and 96 hours post-test and remained significantly lower for up to 8 days post-exercise, while Martin et al. (62) only saw a significant deficit in TF immediately post-test, recovering within 24 hours post-exercise. Gibala et al. (39) results demonstrated that following eccentric exercise ½RT was significantly lower than pre-exercise values immediately post-exercise, as well as at 24 and 48 hours post-exercise.

2.2.2.3 ALTERED JOINT KINEMATICS:

EIMD following eccentric exercise has been reported (44, 52, 72, 79) to cause significant reductions in joint range of motion (ROM), altering joint kinematics. Changes in joint ROM have been attributed to damage to the connective tissue (14, 39, 65, 66, 69, 86) surrounding the muscle rather than an increase in muscle activity which was once believed to be the cause of the decrease in joint ROM (52). EIMD to the connective tissue surrounding the muscle, especially to the perimuscular connective tissue and regions of the myotendinous junction (57) causes swelling and inflammation in the affected area and a shortening of the connective tissue. Decreased joint ROM alters joint kinematics (18) potentially leading to functional impairments to everyday motor tasks.

2.2.2.4 FUNCTIONAL IMPAIRMENTS:

With EIMD causing damage to muscle and connective tissue (14, 18, 65), the afferent receptors (muscle spindles and Golgi tendon organs) located within the tissue may become damaged as well. This may potentially disrupt proprioceptive pathways resulting in neuromuscular impairments and altered communication patterns between the central nervous system (CNS) and the afferent receptors located in the damaged tissue (78). Altered communication patterns can be detrimental to the perception of joint position, joint movement, and muscle tension (63). Losses in proprioceptive functioning may significantly alter the ability to perform everyday motor tasks, ultimately impairing an individuals neuromuscular recruitment patterns and potential increasing risk of injury. Functional impairments caused by EIMD range from decreased joint ROM, to decreased force production, to altered muscle activation and recruitment patterns. Changes induced by EIMD cause damage to the soft-tissue of the body, resulting in soft-tissue restrictions, hindering an individual's ability to perform an activity or everyday motor task to the best of their ability or within what is considered to be their normal range (18). Running kinematics (45), vertical jump height (92), broad jump length (76), timed shuttle runs (61), sprint speed (76), and agility (76) have all been measured in past studies to assess functional impairments following EIMD.

In the assessment of joint kinematics following a bout of 30 minutes of downhill running, Hamill et al. (45) found significant differences in maximal ankle dorsiflexion and plantar flexion during the support phase of the stride, a reduction in maximum knee joint flexion in both swing and support phases, and a reduction in maximum hip flexion at touch down. Changes within the stride kinematics may be attributed to a compensatory response as a result of EIMD inflicted on the quadriceps muscles. EIMD likely causes the connective tissue to shorten as a result of the large number of eccentric muscle contractions involved when downhill running.

Willems et al. (92) looked at changes in one-legged vertical jump height following 20 minutes of downhill walking carrying 10% of their body mass. The control limb had significant deficits in vertical jump height measures at 24, 48, and 72 hours post-exercise. In comparison to pre-test measurements, there was a 21% decrease at 24 hours, a 16% decrease at 48 hours, and a 12% decrease at 72 hours in vertical jump height. Similar to Willems et al. (92) findings regarding vertical jump height, Pearcey et al. (76) found substantial deficits in broad jump performance post-exercise from EIMD in comparison to pre-test measures, with a 0.15 meter decrease at 24 hours, 0.19 meter decrease at 48 hours, and a 0.17 meter decrease in broad jump length at 72 hours post-exercise.

Shuttle run times have been analyzed by Mancinelli et al. (61) to assess the effects EIMD has on female collegiate basketball players performance during a 4 day training camp. Mancinelli et al. (61) showed EIMD to negatively effect shuttle run performance, with subjects averaging 7.92 seconds pre-test and 8.22 seconds post-test, demonstrating that EIMD significantly increases shuttle run times. When further analyzing alternative performance measures Pearcey et al. (76) found EIMD to have substantial negative effects on both 30m sprint time and agility at 24, 48, and 72 hours post exercise. 30m sprint time increased by 0.17 seconds at 24 hours, 0.16 seconds at 48 hours, and 0.13 seconds at 72 hours post-exercise. When assessing agility, test times increased by 0.23 seconds at 24 hours, 0.31 seconds at 48 hours, and 0.19 seconds at 72 hours post exercise.

2.2.2.5 INCREASED INJURY RISK:

With EIMD commonly occurring in individuals who are physically active, it is common for individuals who live an active lifestyle or are employed in a job requiring physical labor to continue to workout or go to work, even at times when they have feelings of intense muscle soreness from EIMD (18). Commonly individuals will work through the pain, potentially putting themselves at increased risk of injury, due to unaccustomed stress placed on the already damaged soft-tissue of the body. This stress can cause individuals to take on altered movement and muscle sequencing patterns to compensate and protect areas in the body recovering from EIMD (83). Changes in movement and muscle sequencing patterns (31) can lead to increased activation of muscles unaccustomed to the demand of the given workload, causing them to work at an intensity greater than what they are familiar to, potentially putting an individual at greater risk of injury. With EIMD having the potential to pose as a significant barrier to an individual's daily routine and participation in daily physical activity, we must first look to understand the underlying mechanism resulting in EIMD before we can look at potential methods to prevent EIMD or help aid in the recovery process following EIMD.

2.2.3 MECHANISMS OF EIMD:

2.2.3.1 INTRODUCTION:

With EIMD being so profound in recreational to elite athletes and with so many unanswered questions regarding the underlying mechanisms of EIMD, a number of theories have been proposed to debunk the underlying cause of EIMD resulting in DOMS. As many as six theories have been proposed and discussed in the literature within the past decade. Each theory has outlined different mechanisms as the root cause of EIMD subsequently resulting in the onset of

DOMS. The six major theories proposed include the: lactic acid, muscle spasm, enzyme efflux, inflammation, muscle tissue damage, and connective tissue damage theories.

2.2.3.2 INCREASED LACTIC ACID ACCUMULATION:

The lactic acid accumulation theory has largely been excluded as the root cause of DOMS. Increased lactic acid accumulation may cause acute pain during or immediately following an intense bout of exercise, but it cannot be established as the underlying cause of EIMD resulting in DOMS. The lactic acid theory has been rejected as muscle lactic acid accumulation has shown to return to pre-test measures within 60 minutes following exercise (80) with blood lactate levels also failing to show any correlations with muscle soreness levels over a 72 hour period post exercise. In addition to these findings, the lactic acid theory, along with other metabolic muscle damage theories have been largely rejected (3, 30). Exercise requiring increased metabolic demands do not always yield greater muscle damage, as running downhill in comparison to running uphill has shown to have greater EIMD even though downhill running requires a lower metabolic cost. Increased EIMD with downhill running is contributed to the substantially greater eccentric component required when running downhill in comparison to uphill running (4). Beltman et al. (11) research supports these finding, outlining the reduced metabolic costs associated with electrically stimulated lengthening contractions when compared with concentric and isometric contractions. Although metabolic demands may have the potential to intensify damage caused from

EIMD, Armstong et al. (4), Beltman et al. (11), and Schwane et al. (80) findings prove that metabolic costs seem highly unlikely to be the main cause of EIMD and the subsequent onset of DOMS. With EIMD unlikely being caused by metabolic stress, it seems likely that mechanical stress may be the root cause of EIMD.

2.2.3.3 MUSCLE SPASMS:

The muscle spasm theory proposes that hyperactivity in the resting muscle post-exercise (13, 22, 43) results in tonic muscle spasms which compress local blood vessels, decreasing blood flow to the muscle, causing ischemia and the accumulation of waste products and enzymes within and around the muscle. The accumulation of waste products and enzymes simulates pain receptors at the site of EIMD. These findings have been controversial, as past research has shown mixed results when looking at muscle activity using both bipolar (2) and unipolar (27) electrodes when measuring electromyography. Some studies have shown no increase in EMG activity in sore muscles, while others have shown increased EMG activity in sore muscles (2, 68). Even with an increase in EMG activity post-exercise, no relationship has been seen between EMG magnitude and muscle soreness (13). Along with the lactic acid theory, the muscle spasm theory has largely been rejected due to the inconsistency in results along with a number of studies showing an increase in resting joint stiffness, but no increase in EMG activity following EIMD (52, 55).

2.2.3.4 MUSCLE TISSUE DAMAGE:

The muscle damage theory was first proposed by Hough (48) in 1902, attributing EIMD to the disruption of the contractile components of the muscle, focusing particularly on disruption seen at the z-line following eccentric contractions (3, 37, 38, 56, 68). The z-line is commonly referred to as the weak link in the contractile structure of the muscle (37). Z-line disruption is the result of myofibrillar and sarcomere architectural damage (18). With eccentric muscle activity commonly known to recruit a larger number of type II fibers (34), and with type II fibers showing the greatest disruption at the z-line as a result of their z-lines being weaker and narrower (70), these findings can be used to explain Newham et al. (70) muscle biopsy findings. Newham et al. (70) biopsy findings showed greater z-line disruption following eccentric muscle activity, mostly likely due to increased type II fiber damage, resulting in greater EIMD. Along with Newham et al. (70) muscle biopsy findings, blood enzymes markers have also supported the muscle tissue theory. Newham et al. (68) displayed that following eccentric contractions, circulating plasma creatine kinase (CK) levels have been shown to rise up to 40,00 IU/L, with normal resting plasma CK levels being around 100 IU/L, Although increased circulating CK levels is an indicator of muscle damage, peak CK levels and peak muscle soreness levels do not correlate with each other (19, 20). It is believed that muscle damage may lead to the stimulation of nociceptors located within the connective tissue and vasculature surrounding the muscle, along with nocieptors at the musculotendinous junction, leading to the sensation of pain (18)

2.2.3.5 CONNECTIVE TISSUE DAMAGE:

The connective tissue theory focuses on the series of elastic components surrounding the muscle, in particular the connective tissue referred to as myofascia that forms sheaths around the bundles of muscle fibers (18). With EIMD, the main site of muscle soreness can be isolated to the distal portion of the muscle, at the myotendinous junction (57), which has a high ratio of connective tissue to muscle tissue in comparison to the rest of the muscle. As with the muscle damage theory and eccentric contractions causing great muscle damage to type II fibers, type II muscle fibers not only differ from type I fibers in regards to z-line properties but connective tissue properties as well. Type I fibers have a more robust connective tissue composition surrounding muscle fiber bundles in comparison to type II fibers. Eccentric muscle activity favors type II muscle fiber recruitment. Since type II fibers demonstrate an increased susceptibility to stretched-induced connective tissue damage (48, 86), the increased stress placed on the connective tissue during an eccentric contraction has a greater potential to cause connective tissue damage (43), stimulating mechanoreceptors (muscle spindles and Golgi tendon organs), subsequently leading to the sensation of pain (55).

Muscle soreness ratings can be directly correlated with hydroxyproline (OHP) levels, a marker of connective tissue breakdown. Maximal OHP levels have been found to occur at 48 hours post-exercise (2), consistent with when DOMS peaks (18, 85). Sydney-Smith & Quigley (87) also investigated the effects of EIMD on connective tissue breakdown analyzing the secretion of OHP and hydroxylysine (OHL), demonstrating mature collagen degradation as a result of overuse or tissue strain, supporting Abraham (2) findings. EIMD to the connective tissue enveloping the muscle not only leads to the sensation of muscle soreness, but also leads to the shortening of the connective tissue, resulting in a loss in ROM (55, 78). Along with a loss in ROM, connective tissue damage can alter neuromuscular recruitment patterns between the central nervous system (CNS) and the afferent receptors located in the damaged tissue (78). Changes in recruitment patterns can alter perception of joint position, joint movement, and muscle tension (63), having significant effects on motor patterns following a bout of eccentric activity causing EIMD.

2.2.3.6 ENZYME EFFLUX:

During EMID a number of different collagen and protein metabolites are released into the extracellular space due to increase membrane permeability, as a result of increased fiber degradation (3). Two major markers of muscle damage, creatine phosphokinase and calcium have been found to accumulate in the muscle following eccentric muscle activity resulting in muscle damage. Creatine phosphokinase is an indirect marker of muscle damage, whereas calcium, which is normally stored in the sarcoplasmic reticulum of the muscle cell is believed to accumulate in the muscle following muscle damage, inhibiting cellular respiration and slowing the re-uptake of calcium back into the sarcoplasmic reticulum (3, 43). Calcium accumulation within the injured muscle is also thought to activate proteases and phospholipases, causing further protein degradation within the muscle, further weakening the z-lines (35). With increased muscle degradation and chemical stimulation, there is likely an increase in the number of nerve endings sensitized, likely increasing the sensation of pain.

2.2.3.7 INFLAMMATION:

During connective and muscle tissue damage, an efflux of enzymes are released from the damaged cells. The body responds to these changes in an attempt to return to a state of homeostasis within the body. The body reacts to these alterations through inflammatory cell infiltration resulting in edema (84). Proteolytic and lipolytic enzymes within the muscle fibers initiate protein and lipid degradation, increasing the turnover rate of damaged muscle and connective tissue. In addition, the accumulation of histamine, kinins, prostaglandins, and potassium (3) attracts monocytes and neutrophils to the area effected by EIMD (46). As a result of these events, fluid moving across the cell membrane into the extracellular space results in edema, increasing osmotic pressure, stimulating group IV sensory neurons, subsequently resulting in pain (36). Smith (84) and Armstrong (3) believe that along with the increase in osmotic pressure, the monocytes/macrophages that accumulate at the site of EIMD also secrete substances that sensitize type III neurons as well as type IV neurons, further elevating the sensation of pain, which generally peaks between 24 and 48 hours.

2.2.3.8 CONCLUSION:

There is no one simple explanation or theory to explain the methods of how the body reacts to a given stimulus or stress. Rather the body reacts as a system, generally consisting of a serious of events, with all the events being interrelated, and each having a subsequent effect on the other. It is unlikely that one theory can explain EIMD. Most researchers have amalgamated a number of the aforementioned theories to outline the sequence of events that occurs when the body recovers from and adapts to the stress placed on it following a given bout of physical activity resulting in EIMD. Researchers generally agree that the high tensile forces placed on the soft-tissue of the body, especially via eccentric contractions, causes muscle and connective tissue damage, which leads to an enzyme efflux into the extracellular space. The diffusion of intracellular enzymes into extracellular space activates protease and phospholipases, ultimately leading to protein degradation, resulting in the further breakdown of damaged muscle and connective tissue. The above outlined series of events along with the accumulation of histamine, kinins, prostaglandins, and potassium, attracts monocytes and neutrophils to the area, increasing extracellular osmotic pressure, resulting in edema followed by pain.

2.2.4 ALLEVIATING EIMD & DOMS:

2.2.4.1 INTRODUCTION:

As a result of the complex sequence of events that ensue following EIMD, it is no wonder why there has been no one treatment, therapy, or modality that has provided undisputable results when treating EIMD. A number of different modalities have been implemented to treat EIMD, including: massage, cryotherapy, light exercise, compression and stretching. The majority of these therapies have provided varying results as a result of the heterogeneity in the methodologies used to investigate the effects of a given intervention. One therapy that has shown promising results is massage. A number of studies have demonstrated the potential benefits of the implementation of massage following EIMD. As foam rolling is considered a form of self-massage, and with the popularity of its use as a recovery tool following an intense bout of physical activity, it is of importance to utilize the wealth of knowledge obtained through the analysis of massage to help understand the potential effects the implementation of foam rolling may have as a recovery tool following an intense bout of physical activity.

2.2.4.2 MASSAGE:

The effects of massage in the recovery process following EIMD have been researched quite extensively. The large variance in methodologies regarding the type, time, and duration of the massage intervention, along with the: exercise modality used to induce EIMD, subjects utilized for the study, and measurements analyzed, it has become difficult to draw conclusions as to the effects of massage in the recovery process following EIMD. The theory behind the recovery effects of massage on EIMD is that massage can increase blood flow, therefore increasing the amount of oxygen and nutrients being able to reach the damaged tissue effected by EIMD (3). Increased oxygenated blood flow may potentially hinder the migration of neutrophils to the site of injury (85) while also restoring mitochondrial regeneration of ATP, therefore restoring the active transport of calcium back into the sarcoplasmic reticulum (3). Decreased neutrophil counts are believed to reduce prostaglandin production, subsequently attenuating damage associated with the inflammatory process. A number of variables have been analyzed to gain further insight into the effects of massage on EIMD, including: muscle strength (1, 33, 47, 91, 93), inflammation (24), mitochondrial biogenesis (24), neutrophil counts (47, 85), creatine kinase levels (33, 85), blood flow (15, 49, 88), limb girth (1, 93), muscle soreness (1, 33, 47, 61, 85, 92, 93), range of motion (1, 47, 93), and vertical jump (33, 61, 92).

EIMD is the direct result of unaccustomed stress placed on the muscle resulting in muscle and connective tissue damage. One of the subsequent results of EIMD is the attenuation of muscular strength and force production (39, 77). Abad et al. (1) found that the implementation of massage following an EIMD protocol had no effect in helping to reduce 1RM strength deficits following EIMD at post-48 hours and post-96 hours. Farr et al. (33) found that 40 minutes of downhill walking with a load of 10% of the subjects body weight caused a significant difference from pre-test measures in muscle strength at post-1 hour for both limbs, with massage having no effect on improving muscle strength. Even with varying EIMD and massage protocols, Hilbert et al. (47), Zainuddin et al. (93), and Weber et al. (91) all found that massage had no significant effects in attenuating strength deficits seen following EIMD. It can be concluded that massage may not be beneficial in directly treating the damaged inflicted on the muscle tissue of the body, but may help in the treatment of secondary responses following muscle tissue damage.

Following EIMD, the mechanical disruption to sarcomeres subsequently proliferates into a secondary inflammatory response (41). Crane's et al. (24) research is one of the first studies to provide supporting cellular and mechanistic evidence to support the application of massage following EIMD. Crane et al. (24) demonstrated that massage therapy post-EIMD is clinically beneficial in reducing signs of inflammation. It is believed that massage activates mechanotransductional signaling, attenuating the rise of several pathways indicative of muscle inflammation. The attenuated production of inflammatory cytokines following massage may help in reducing the sensation of DOMS. On top of attenuating inflammation, Crane et al. (24) demonstrated that massage therapy post EIMD is clinically beneficial in promoting mitochondrial biogenesis, due to massage causing an increase in nuclear abundance of PGC-1α at 2.5 hours post massage. PGC-1α is an important mediator of tissue repair by enhancing metabolism and increasing mitochondrial content. Crane's et al. (24) research demonstrates mechanistically how a massage intervention can be beneficial in treating EIMD, attenuating the first stages of the secondary response to muscle tissue damage (i.e. inflammation).

Inflammation is first initiated by the migration or increased accumulation of neutrophils at the site of the injury. Smith's et al. (85) research has shown that following EIMD resulting in DOMS, massage causes a slight increase in circulating neutrophils levels, with the control group showing the contrary. Smith et al. (85) attributes these findings to massage not allowing neutrophils to be able to migrate from circulation into the tissue space, causing elevated neutrophil levels in the blood. In contrast Hilbert et al. (47) found with the implementation of massage, no significant changes in neutrophil counts were seen between the massage and control group.

Once the inflammatory response has been initiated by an increase in neutrophils at the site of injury, increased neutrophil levels generally initiates an increase in serum creatine kinase (CK) levels. Smith et al. (85) showed massage to significantly reduce serum CK levels following a DOMS inducing EIMD protocol. They believed that massage may interfere with neutrophil accumulation at the injury site causing a reduction in CK efflux following eccentric exercise. Zainuddin et al. (93) supports Smith et al. (85) findings, showing that following 10 sets of 6 repetitions of eccentric biceps curls, plasma CK levels were significantly lower following a massage intervention in comparison to the CON group at post-96 hours.

As part of the inflammatory process, blood begins to pool at the site of injury, increasing white blood cell counts to reduce muscle and tissue damage. One proposed mechanism to aid in the recovery process following EIMD is to increase blood flow to the injury site, increasing oxygen and nutrients to damaged tissue cells, while removing waste products and debris. Although no research to the author's knowledge has looked at the effects of massage on blood flow following EIMD, Hovind & Nielsen (49) and Cafarelli & Flint (15) found that following a massage intervention subjects vascular bed blood flow increased, whereas Tiidus (88) found no changes in arterial or venous blood flow following massage. These findings may be the result of massage not having a significant effect on total systemic blood flow, but having a more minute effect at the vascular bed level, increasing the diffusion of oxygen and nutrients into the damaged cells, along with the diffusion of carbon dioxide and other waste products out of the cells.

Increased blood flow and fluid accumulating at the site of injury due to inflammation has also shown to cause an increase in limb girth following EIMD (52, 84). Zainuddin et al. (93) showed that following 10 sets of 6 repetitions of eccentric biceps curls, a massage intervention significantly reduced upper arm circumference at POST-72 and POST-96 in comparison to the control group. On the contrary, Abad et al. (1) found that the implementation of massage following an EIMD protocol had no effect on limb girth following EIMD, although no significant changes were seen in limb girth following the EIMD protocol at all time points (POST-24, 48, 72, & 96).

Nociceptors and other pain receptors are activated as a result of sarcomere damage, calcium accumulation, protein degradation, and osmotic pressure, resulting in the sensation of DOMS (18). Farr et al. (33) found massage to significantly attenuate muscle soreness and tenderness at POST-24 following an EIMD protocol consisting of 40 minutes of downhill walking with a load of 10% of the subjects body weight. Hilbert et al. (47), Willems et al. (92), and Mancinelli et al. (61) also found massage to have a significant effect in attenuating muscle soreness following EIMD at POST-48 in comparison to the control group, even with each study using a different protocol to elicit EIMD. Smith et al. (85) showed massage to significantly reduce muscle soreness following an EIMD protocol. The massage group peaked in muscle soreness at POST-24 and the control group peaked at POST-48. The control group also consistently reported higher muscle soreness readings from POST-24 to POST-96. Zainuddin et al. (93) had similar findings with a massage intervention following 10 sets of 6 repetitions of eccentric biceps curls, with massage significantly attenuating muscle soreness following EIMD, alleviating the severity of DOMS by 20-40%. Although a number of studies have shown a massage intervention to be beneficial in treating DOMS following an EIMD protocol, Abad et al. (1) and Weber et al. (91) found no significant differences in muscle soreness readings between massage and control groups following an EIMD protocol at all time points (POST-24, 48, 72, & 96).

Mechanical disruption to muscle fibers, inflammation, and the chronic activation of pain receptors generally results in a reduction in ROM. Abad et al. (1) found that the implementation of massage following an EIMD protocol had no effect in helping to reduce ROM deficits seen following EIMD at all time points (POST-24, 48, 72, & 96). Hilbert et al. (47) and Zainuddin et al. (93) also found that massage had no significant effects in decreasing ROM deficits seen following EIMD.

As a result of the changes inflicted by EIMD outlined in the previous paragraphs, functional movement is generally impaired. A commonly administered test to analyze functional impairments following EIMD is vertical jump (VJ). Mancinelli et al. (61) found that following an intense day of a pre-season training camp involving strength training and drills, VJ height had significantly improved at POST-48 following a massage intervention, where the control group VJ height performances did not. Willems et al. (92) found that following a EIMD protocol consisting of 20 minutes of downhill walking with a load equivalent to 10% of your bodyweight, massage was effective in improving VJ height performance at POST-48 in comparison to a control group. In contrast to Mancinelli et al. (61) and Willems et al. (92) findings, Farr et al. (33) found a significant decrease in VJ height for 1-legged VJs at POST-01 and POST-24 for the massage limb in comparison to the contralateral control limb following 40 minutes of downhill walking with subjects loaded with 10% of their body weight. Since the VJ protocols implemented between studies varied, further research must be administered to gain further insight into the effects of massage on VJ.

It can be concluded that there are a number of potential benefits to the implementation of massage following EIMD. Although massage has not been shown to be an effective method in improving range of motion (90, 93) or muscular strength (33, 90, 93) following EIMD, massage has been shown to be beneficial in treating EIMD by increasing mitochondrial biogenesis (24), restoring blood flow (85), and vertical jump height (61, 92), while decreasing muscle soreness (24, 33, 85, 93), cellular stress (24), and inflammation (24). Massage has also shown varying results in reducing limb circumference (90, 93) and creatine kinase levels (85, 90) while potentially increase circulating neutrophil counts (23, 85, 90).

2.2.4.3 CRYOTHERAPY:

Cryotherapy is a commonly used treatment in recovery from EIMD. Cryotherapy is implemented to decrease inflammation, swelling and intravascular pressure following soft-tissue injury (43). Ascensão et al. (6), Bailey et al. (7), Eston & Peters (32) and Skurvydas et al. (82) all demonstrated the beneficial effects of the implementation of 10-15 minutes of cold-water immersion following EIMD. Cryotherapy demonstrated to be beneficial in reducing: muscle soreness (6, 7, 82), plasma creatine kinase levels (6, 32, 82), and blood myoglobin concentrations (6, 7) while also attenuating decrements in muscular strength (voluntary and evoked) (6, 7, 82), range of motion (32), and vertical jump performance (82). On the contrary, a number of other studies (42, 53, 73) have also assessed the effects of cryotherapy on recovery from EIMD and have found no significant differences when assessing the same dependent variables. The heterogeneity in methodological designs makes it hard to pool findings regarding the effects cryotherapy on treating EIMD. Evidence does point to cryotherapy applied repeatedly over time to be the most effective method in promoting muscle recovery (32).

2.2.4.4 COMPRESSION:

Compression therapy is a relatively new therapy implemented to treat EIMD. A number of studies addressing the effects of compression garments on EIMD have demonstrated that the use of graduated compression stockings attenuated blood lactate recovery time (12, 16) along with significantly improving post-recovery cycling performance (16). In a recent study, Gill et al. (40) demonstrated that the use of a lower body compression garment for 12 hour duration post-game enhanced recovery from muscle damage in rugby players in comparison to a control group. A more in-depth analysis conducted by Kraemer et al. (59) revealed that wearing a compression sleeve garment following eccentric elbow flexion contractions was effective in reducing strength losses, muscle soreness, swelling and joint stiffness. Presently, the small body of compression research indicates that compression may be beneficial for treating EIMD. Only one study to date has shown that the use of a compression garment had no effect on post-exercise recovery when analyzing blood lactate concentrations, oxygen consumption, and heart rate. More research needs to be conducted to increase or understanding of how compression garments aid in the recovery process, especially over a 72 hour plus recovery period.

2.2.4.5 LIGHT EXERCISE:

Low intensity exercise is another recovery method commonly implemented following EIMD. Low intensity exercise is believed to help flush out any left over waste products and debris resulting from EIMD. Similar to cryotherapy research, the varying methodologies implemented in past studies makes it difficulty to draw any concrete conclusions regarding the effects low-intensity exercise has on EIMD. Donnelly et al. (28), Saxton & Donnelly (79), and Zainuddin et al. (94) all showed light exercise to be beneficial in helping to treat EIMD. Low-intensity exercise has been shown to reduce plasma creatine kinase levels (28, 79), muscle soreness (79), and muscle tenderness (94). On the contrary, a number of other studies (17, 26, 91) have also assessed the effects of light exercise on EIMD recovery and have found no significant differences when assessing the same dependent variables.

2.2.4.6 STRETCHING:

Static stretching is commonly implemented prior to or following any type of intense physical activity. Static stretching is believed to help attenuate losses in joint ROM, while decreasing muscle soreness and stiffness following an intense bout of physical activity. McGlynn et al. (64) and Torres et al. (89) have found static stretching to decrease EMG activity and attenuate reductions in joint ROM, respectively. Gulick et al. (44) contradicts Torres et al. (89) findings, demonstrating static stretching to have no effect on joint ROM recovery following EIMD. Although static stretching has shown potential benefits in attenuating EMG activity and losses in joint ROM, static stretching has shown no effects in reducing muscle soreness (44, 54, 64) and tenderness (54), muscle strength deficits (44, 54), and limb girth (44), all of which are common symptoms of EIMD.

2.2.4.7 CONCLUSION:

Existing therapies have provided inconclusive results for treating EIMD. To date, massage therapy seems to be the most effective method of intervention. With FR being termed a form of self-massage and with FR becoming an increasingly popular therapy technique used by therapists, fitness professionals, and active populations, it seems prudent that we analyze the efficacy of FR as a recovery tool following EIMD. Furthermore, physiotherapists have estimated that ~45% of their time spent with athletes is devoted to massage during major national and international athletic events (5). Therapist and fitness professionals have reported that the main limitation in the implementation of massage is the time commitment required, as many therapists/fitness professional must deal with high-volume caseloads, making massage too time-consuming to implement (75). Therapists and fitness professionals who can effectively implement FR into a patient, client, or athlete's program, may be able to more effectively address soft-tissue problems that have the potential to manifest into pain and joint dysfunction.

However, only one practical study (76) has addressed the effectiveness of foam rolling as a recovery tool following EIMD.

2.2.5 ALLEVIATING EIMD & DOMS VIA FOAM ROLLING (FR):

Currently there is only one published article by Pearcey et al. (76) pertaining to the effects of FR on EIMD. Pearcey et al. (76) analyzed the effects of FR on functional measures such as sprint speed, agility, broad jump, squat strength and pain threshold. Their findings showed that FR substantially improved quadriceps muscle tenderness in the days following EIMD (d=0.59 to 0.84) while also substantially improving sprint time (d=0.68 to 0.77), power (d=0.48 to 0.87) and dynamic strength-endurance (d=0.54). They concluded that FR is effective in reducing DOMS and associated decrements in most dynamic performance measures. Since Pearcey et al. (76) took an applied research approach, the next progression is to analyze the possible underlying mechanisms regarding how FR improves the recovery process following EIMD. No research to date has analyzed the underlying mechanisms on how FR improves the recovery process from EIMD.

2.3 CONCLUSION:

Through the analysis of the effects, mechanisms, and methods of treating EIMD, along with a thorough review of the effectiveness of a number of common interventions implemented to help treat symptoms of EIMD, a vast knowledge base has been established. From this knowledge base, the wealth of information obtained persisting to

EIMD and FR can be applied to provide insight into the potential effectiveness of the implementation of FR as a recovery tool following EIMD. With an understanding how EIMD is induced, the effects of EIMD, and the variety of treatment methods implemented, and their benefits, detriments, or trivial effect in aiding in the recovery process from EIMD, hypotheses can be drawn regarding the potential effects of FR in treating EIMD.

With FR being in the exploratory phase from a research stand point, but commonly being implemented in therapeutic, rehabilitation, and performance settings, knowledge needs to be gained to grasp a better understanding regarding the effectiveness of FR. With few articles published persisting to FR, FR research to date has looked at FR density (25), the acute effects of FR on neuromuscular performance (60), and the effects of FR on applied performance measures following EIMD (76). With FR research being rudimentary, a number of avenues are open to explore. Of interest, the analysis of the neuromuscular mechanism regarding how the implementation of an FR protocol following EIMD affects the recovery process. Gaining insight into the neuromuscular mechanism of FR will give a better understanding of how FR effects muscle tissue, connective tissue, and central nervous system responses following EIMD. With this knowledge, we hope to be able to explain why one see the effects/changes demonstrate in previous studies published (76), following the implementation of FR. With this knowledge, we will be better able to prescribe, implement and utilize FR in therapeutic, rehabilitation, and performance settings.

2.4 REFERNCES:

1. Abad CCC, Ito LT, Barroso R, Ugrinowitsch C, and Tricoli V. Effect of Classical Massage on Subjective Perceived Soreness, Edema, Range of Motion and Maximum Strength after Delayed Onset Muscle Soreness Induced by Exercise. *Revista Brasileira De Medicina Do Esporte*. 2010;16(1):36-40.

2. Abraham WM. Factors in delayed muscle soreness. *Medicine and Science in Sports and Exercise*. 1977;9(1):11-20.

3. Armstrong RB. Mechanisms of Exercise-Induced Delayed Onset Muscular Soreness. *Medicine and Science in Sports and Exercise*. 1984;16(6):529-38.

4. Armstrong RB, Ogilvie RW, and Schwane JA. Eccentric exercise-induced injury to rat skeletal muscle. *Journal of applied physiology: respiratory, environmental and exercise physiology*. 1983;54(1):80-93.

5. Arroyo-Morales M, Olea N, Martinez M, Moreno-Lorenzo C, Diaz-Rodriguez L, and Hidalgo-Lozano A. Effects of myofascial release after high-intensity exercise: a randomized clinical trial. *Journal of manipulative and physiological therapeutics*. 2008;31(3):217-23.

6. Ascensao A, Leite M, Rebelo AN, Magalhaes S, and Magalhaes J. Effects of cold water immersion on the recovery of physical performance and muscle damage following a one-off soccer match. *Journal of Sports Sciences*. 2011;29(3):217-25.

7. Bailey DM, Erith SJ, Griffin PJ, Dowson A, Brewer DS, Gant N, and Williams C. Influence of cold-water immersion on indices of muscle damage following prolonged intermittent shuttle running. *Journal of Sports Sciences*. 2007;25(11):1163-70.

8. Barnes J. Myofascial Release. In: MI Hammer editor. *Functional Soft Tissue Examination and Treatment by Manual Methods: New Perspectives*. Aspen: Gaitherburg; 2005, pp. 533-88.

9. Barnes MF. The basic science of myofascial release: morphologic change in connective tissue. *Journal of Bodywork & Movement Therapies*. 1997;1(4):231-8.

10. Barnes MF. The basic science of myofascial release: morphologic change in connective tissue. *Journal of Bodywork and Movement Therapies*. 1997;1(4):231-8.

11. Beltman JG, van der Vliet MR, Sargeant AJ, and de Haan A. Metabolic cost of lengthening, isometric and shortening contractions in maximally stimulated rat skeletal muscle. *Acta physiologica Scandinavica*. 2004;182(2):179-87.

12. Berry MJ, and Mcmurray RG. Effects of Graduated Compression Stockings on Blood Lactate Following an Exhaustive Bout of Exercise. *American Journal of Physical Medicine & Rehabilitation.* 1987;66(3):121-32.

13. Bobbert MF, Hollander AP, and Huijing PA. Factors in delayed onset muscular soreness of man. *Medicine and Science in Sports and Exercise.* 1986;18(1):75-81.

14. Brown SJ, Child RB, Day SH, and Donnelly AE. Indices of skeletal muscle damage and connective tissue breakdown following eccentric muscle contractions. *European Journal of Applied Physiology and Occupational Physiology.* 1997;75(4):369-74.

15. Cafarelli E, and Flint F. The role of massage in preparation for and recovery from exercise. An overview. *Sports medicine.* 1992;14(1):1-9.

16. Chatard JC, Atlaoui D, Farjanel J, Louisy F, Rastel D, and Guezennec CY. Elastic stockings, performance and leg pain recovery in 63-year-old sportsmen. *European Journal of Applied Physiology.* 2004;93(3):347-52.

17. Chen TC, Nosaka K, and Wu CC. Effects of a 30-min running performed daily after downhill running on recovery of muscle function and running economy. *Journal of Science and Medicine in Sport.* 2008;11(3):271-9.

18. Cheung K, Hume P, and Maxwell L. Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine.* 2003;33(2):145-64.

19. Clarkson PM, Byrnes WC, McCormick KM, Turcotte LP, and White JS. Muscle soreness and serum creatine kinase activity following isometric, eccentric, and concentric exercise. *International journal of sports medicine.* 1986;7(3):152-5.

20. Clarkson PM, and Ebbeling C. Investigation of serum creatine kinase variability after muscle-damaging exercise. *Clinical science.* 1988;75(3):257-61.

21. Clarkson PM, Nosaka K, and Braun B. Muscle Function after Exercise-Induced Muscle Damage and Rapid Adaptation. *Medicine and Science in Sports and Exercise.* 1992;24(5):512-20.

22. Cleak MJ, and Eston RG. Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Medicine.* 1992;26(4):267-72.

23. Connolly DAJ, Sayers SP, and McHugh MP. Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research.* 2003;17(1):197-208.

24. Crane JD, Ogborn DI, Cupido C, Melov S, Hubbard A, Bourgeois JM, and Tarnopolsky MA. Massage therapy attenuates inflammatory signaling after exercise-induced muscle damage. *Science Translational Medicine.* 2012;4(119):119ra13.

25. Curran PF, Fiore RD, and Crisco JJ. A comparison of the pressure exerted on soft tissue by 2 myofascial rollers. *Journal of Sport Rehabilitation*. 2008;17(4):432-42.

26. Dannecker EA, Koltyn KF, Riley JL, and Robinson ME. The influence of endurance exercise on delayed onset muscle soreness. *Journal of Sports Medicine and Physical Fitness*. 2002;42(4):458-65.

27. Devries HA. Prevention of muscular distress after exercise. *Research Quarterly*. 1961;32(2):177-85.

28. Donnelly AE, Clarkson P, and Maughan RJ. Exercise-induced muscle damage: effects of light exercise on damaged muscle. *European Journal of Applied Physiology and Occupational Physiology*. 1992;64(4):350-3.

29. Dorpat TL, and Holmes TH. Mechanisms of skeletal muscle pain and fatigue. *A.M.A. archives of neurology and psychiatry*. 1955;74(6):628-40.

30. Ebbeling CB, and Clarkson PM. Exercise-induced muscle damage and adaptation. *Sports medicine*. 1989;7(4):207-34.

31. Edgerton VR, Wolf SL, Levendowski DJ, and Roy RR. Theoretical basis for patterning EMG amplitudes to assess muscle dysfunction. *Medicine and science in sports and exercise*. 1996;28(6):744-51.

32. Eston R, and Peters D. Effects of cold water immersion on the symptoms of exercise-induced muscle damage. *Journal of Sports Sciences*. 1999;17(3):231-8.

33. Farr T, Nottle C, Nosaka K, and Sacco P. The effects of therapeutic massage on delayed onset muscle soreness and muscle function following downhill walking. *Journal of Science and Medicine in Sport*. 2002;5(4):297-306.

34. Friden J. Muscle soreness after exercise: implications of morphological changes. *International journal of sports medicine*. 1984;5(2):57-66.

35. Friden J, Seger J, Sjostrom M, and Ekblom B. Adaptive response in human skeletal muscle subjected to prolonged eccentric training. *International journal of sports medicine*. 1983;4(3):177-83.

36. Friden J, Sfakianos PN, and Hargens AR. Muscle Soreness and Intramuscular Fluid Pressure - Comparison between Eccentric and Concentric Load. *Journal of Applied Physiology*. 1986;61(6):2175-9.

37. Friden J, Sjostrom M, and Ekblom B. A Morphological-Study of Delayed Muscle Soreness. *Experientia*. 1981;37(5):506-7.

38. Friden J, Sjostrom M, and Ekblom B. Myofibrillar Damage Following Intense Eccentric Exercise in Man. *International Journal of Sports Medicine.* 1983;4(3):170-6.

39. Gibala MJ, Macdougall JD, Tarnopolsky MA, Stauber WT, and Elorriaga A. Changes in human skeletal-muscle ultrastructure and force production after acute resistance exercise. *Journal of Applied Physiology.* 1995;78(2):702-8.

40. Gill ND, Beaven CM, and Cook C. Effectiveness of post-match recovery strategies in rugby players. *British Journal of Sports Medicine.* 2006;40(3):260-3.

41. Gleeson M, Almey J, Brooks S, Cave R, Lewis A, and Griffiths H. Haematological and acute-phase responses associated with delayed-onset muscle soreness in humans. *European journal of applied physiology and occupational physiology.* 1995;71(2-3):137-42.

42. Goodall S, and Howatson G. The effects of multiple cold water immersions on indices of muscle damage. *Journal of Sports Science and Medicine.* 2008;7(2):235-41.

43. Gulick DT, and Kimura IF. Delayed onset muscle soreness: what is it and how do we treat it? *Journal of Sport Rehabilitation.* 1996;5:234-43.

44. Gulick DT, Kimura IF, Sitler M, Paolone A, and Kelly JD. Various treatment techniques on signs and symptoms of delayed onset muscle soreness. *Journal of Athletic Training.* 1996;31(2):8.

45. Hamill J, Freedson PS, Clarkson PM, and Braun B. Muscle Soreness during Running - Biomechanical and Physiological Considerations. *International Journal of Sport Biomechanics.* 1991;7(2):125-37.

46. Hasson S, Daniels J, Divine J, Niebuhr B, Richmond S, Stein P, and al. e. Effect of ibuprofen use on muscle soreness, damage, and performance: A preliminary investigation. *Medicine and Science in Sport and Exercise.* 1993;25:9-17.

47. Hilbert JE, Sforzo GA, and Swensen T. The effects of massage on delayed onset muscle soreness. *British Journal of Sports Medicine.* 2003;37(1):72-5.

48. Hough T. Ergographic studies in muscular soreness. *American Journal of Physiology.* 1902;7(1):76-92.

49. Hovind H, and Nielsen SL. Effect of massage on blood flow in skeletal muscle. *Scandinavian journal of rehabilitation medicine.* 1974;6(2):74-7.

50. Howatson G. The impact of damaging exercise on electromechanical delay in biceps brachii. *Journal of Electromyography and Kinesiology.* 2010;20(3):477-81.

51. Howatson G, and van Someren KA. The prevention and treatment of exercise-induced muscle damage. *Sports Medicine*. 2008;38(6):483-503.

52. Howell JN, Chila AG, Ford G, David D, and Gates T. An Electromyographic Study of Elbow Motion during Postexercise Muscle Soreness. *Journal of Applied Physiology*. 1985;58(5):1713-8.

53. Jakeman JR, Macrae R, and Eston R. A single 10-min bout of cold-water immersion therapy after strenuous plyometric exercise has no beneficial effect on recovery from the symptoms of exercise-induced muscle damage. *Ergonomics*. 2009;52(4):456-60.

54. Johansson PH, Lindstrom L, Sundelin G, and Lindstrom B. The effects of preexercise stretching on muscular soreness, tenderness and force loss following heavy eccentric exercise. *Scandinavian journal of medicine & science in sports*. 1999;9(4):219-25.

55. Jones DA, Newham DJ, and Clarkson PM. Skeletal-muscle stiffness and pain following eccentric exercise of the elbow flexors. *Pain*. 1987;30(2):233-42.

56. Jones DA, Newham DJ, Round JM, and Tolfree SEJ. Experimental Human-Muscle Damage - Morphological-Changes in Relation to Other Indexes of Damage. *Journal of Physiology-London*. 1986;375:435-48.

57. Komi PV, and Buskirk ER. Effect of eccentric and concentric muscle conditioning on tension and electrical-activity of human muscle. *Ergonomics*. 1972;15(4):417.

58. Komi PV, and Viitasalo JT. Changes in Motor Unit-Activity and Metabolism in Human Skeletal-Muscle during and after Repeated Eccentric and Concentric Contractions. *Acta Physiologica Scandinavica*. 1977;100(2):246-54.

59. Kraemer W, Bush J, Wickham R, Denegar C, Gomez A, Gotshalk L, and al. e. Influence of compression therapy on symptoms following soft tissue injury from maximal eccentric exercise. *Journal of Orthopedic and Sports Physical Therapy*. 2001;31(6):282-90.

60. MacDonald G, Penney M, Mullaley M, Cuconato A, Drake C, Behm DG, and Button DC. An acute bout of self myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *Journal of Strength and Conditioning Research*. 2013;27(3):812-21.

61. Mancinelli CA, Davis DS, Aboulhosn L, Brady M, Eisenhofer J, and Foutty S. The effects of massage on delayed onset muscle soreness and physical performance in female collegiate athletes. *Physical Therapy in Sport*. 2006;7(1):5-13.

62. Martin V, Millet GY, Lattier G, and Perrod L. Effects of recovery modes after knee extensor muscles eccentric contractions. *Medicine and Science in Sports and Exercise*. 2004;36(11):1907-15.

63. McCloskey DI. Kinesthetic sensibility. *Physiological reviews*. 1978;58(4):763-820.

64. Mcglynn GH, Laughlin NT, and Rowe V. Effect of electro-myographic feedback and static stretching on artificially induced muscle soreness. *American Journal of Physical Medicine & Rehabilitation*. 1979;58(3):139-48.

65. Mills PC, Ng JC, Thornton J, Seawright AA, and Auer DE. Exercise-induced connective-tissue turnover and lipid-peroxidation in horses. *British Veterinary Journal*. 1994;150(1):53-63.

66. Morgan DL, and Proske U. Popping sarcomere hypothesis explains stretch-induced muscle damage. *Clinical and Experimental Pharmacology & Physiology*. 2004;31(8):541-5.

67. Newham DJ, Jones DA, and Clarkson PM. Repeated high-force eccentric exercise: effects on muscle pain and damage. *Journal of applied physiology*. 1987;63(4):1381-6.

68. Newham DJ, Jones DA, and Edwards RH. Large delayed plasma creatine kinase changes after stepping exercise. *Muscle & nerve*. 1983;6(5):380-5.

69. Newham DJ, Jones DA, Tolfree SEJ, and Edwards RHT. Skeletal-Muscle Damage - a Study of Isotope Uptake, Enzyme Efflux and Pain after Stepping. *European Journal of Applied Physiology and Occupational Physiology*. 1986;55(1):106-12.

70. Newham DJ, McPhail G, Mills KR, and Edwards RH. Ultrastructural changes after concentric and eccentric contractions of human muscle. *Journal of the neurological sciences*. 1983;61(1):109-22.

71. Newham DJ, Mills KR, Quigley BM, and Edwards RHT. Pain and Fatigue after Concentric and Eccentric Muscle Contractions. *Clinical Science*. 1983;64(1):55-62.

72. Nosaka K, and Clarkson PM. Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. *International journal of sports medicine*. 1996;17(2):120-7.

73. Paddon-Jones DJ, and Quigley BM. Effect of cryotherapy on muscle soreness and strength following eccentric exercise. *International Journal of Sports Medicine*. 1997;18(8):588-93.

74. Paolini J. Review of myofascial release as an effective massage therapy technique. *Athletic Therapy Today*. 2009;15(5):30-4.

75. Paolini J. Review of Myofascial Release as an Effective Massage Therapy Technique. *Athletic Therapy Today*. 2009;14(5):30-4.

76. Pearcey GEP, Bradbury-Squires DJ, Kawamoto JE, Drinkwater EJ, Behm DG, and Button DC. Effects of foam rolling on delayed onset muscle soreness and recovery of dynamic performance measures. *Journal of Athletic Training [In Press]*. 2013.

77. Prasartwuth O, Taylor JL, and Gandevia SC. Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. *Journal of Physiology-London*. 2005;567(1):337-48.

78. Saxton JM, Clarkson PM, James R, Miles M, Westerfer M, Clark S, and Donnelly AE. *Neuromuscular dysfunction following eccentric exercise*. 1995, 1185-93 p.

79. Saxton JM, and Donnelly AE. Light concentric exercise during recovery from exercise-induced muscle damage. *International journal of sports medicine*. 1995;16(6):347-51.

80. Schwane JA, Hatrous BG, and Johnson SR. Is lactic acid related to delayed-onset muscle soreness? *Physician and sportsmedicine*. 1983;11(3):124-27.

81. Sefton J. Myofascial Release for Athletic Trainers, Part 1: Theory and Session Guidelines. *Athletic Therapy Today*. 2004;9(1):48-9.

82. Skurvydas A, Sipaviciene S, Krutulyte G, Gailiuniene A, Stasiulis A, and Mamkus G. Cooling leg muscles affects dynamics of indirect indicators of skeletal muscle damage. *Journal of Back and Musculoskeletal Rehabilitation*. 2006;19:141-51.

83. Smith L. Causes of delayed onset muscle soreness and the impact on athletic performance: A review. *The Journal of Applied Sport Science Research*. 1992;6(3):135-41.

84. Smith LL. Acute-Inflammation - the Underlying Mechanism in Delayed Onset Muscle Soreness. *Medicine and Science in Sports and Exercise*. 1991;23(5):542-51.

85. Smith LL, Keating MN, Holbert D, Spratt DJ, Mccammon MR, Smith SS, and Israel RG. *The effects of athletic massage on delayed-onset muscle soreness, creatine-kinase, and neutrophil count - a preliminary report*. 1994, 93-9 p.

86. Stauber WT. Eccentric Action of Muscles - Physiology, Injury, and Adaptation. *Exercise and Sport Sciences Reviews/Series*. 1989;17:157-85.

87. Sydney-Smith M, and Quigley B. *Delayed onset muscle soreness: evidence of connective tissue damage, liquid peroxidation and altered renal function after exercise [dissertation]*. Canberra: Australian Sports Commission; 1992. Available from: Australian Sports Commission.

88. Tiidus PM. Manual massage and recovery of muscle function following exercise: a literature review. *The Journal of orthopaedic and sports physical therapy*. 1997;25(2):107-12.

89. Torres R, Appell HJ, and Duarte JA. Acute effects of stretching on muscle stiffness after a bout of exhaustive eccentric exercise. *International Journal of Sports Medicine*. 2007;28(7):590-4.

90. Torres R, Ribeiro F, Duarte JA, and Cabri JMH. *Evidence of the physiotherapeutic interventions used currently after exercise-induced muscle damage: systematic review and meta-analysis* 2012. 101-14 p.

91. Weber MD, Servedio FJ, and Woodall WR. The Effects of 3 Modalities on Delayed-Onset Muscle Soreness. *Journal of Orthopaedic & Sports Physical Therapy*. 1994;20(5):236-42.

92. Willems M, Hale T, and Wilkinson C. Effects of manual massage on muscle-specific soreness and single leg jump performance after downhill treadmill walking. *Medicina Sportiva*. 2009;13(2):6.

93. Zainuddin Z, Newton M, Sacco P, and Nosaka K. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. *Journal of Athletic Training*. 2005;40(3):174-80.

94. Zainuddin Z, Sacco P, Newton M, and Nosaka K. Light concentric exercise has a temporarily analgesic effect on delayed-onset muscle soreness, but no effect on recovery from eccentric exercise. *Applied Physiology, Nutrition, and Metabolism*. 2006;31(2):126-34.

95. Zhou S. Acute effect of repeated maximal isometric contraction on electromechanical delay of knee extensor muscle. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*. 1996;6(2):117-27.

FOAM ROLLING AS A RECOVERY TOOL FOLLOWING AN INTENSE BOUT OF PHYSICAL ACTIVITY

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CHAPTER 3: FOAM ROLLING AS A RECOVERY TOOL FOLLOWING AN INTENSE BOUT OF PHYSICAL ACTIVITY

3.1 ABSTRACT:

3.1.1 PURPOSE:

Understand the effectiveness of foam rolling as a recovery tool following exercise induced muscle damage (EIMD), analyzing: muscle soreness, dynamic and passive range of motion (ROM), along with evoked and voluntary neuromuscular properties.

3.1.2 METHODS:

20 male subjects with 3+ years of strength training experience were randomly divided into either the control (CON) (n=10) or foam rolling (FR) (n=10) group. All subjects followed the same testing protocol. The only between group difference was that the FR group performed a 20-minute foam rolling exercise protocol at the end of the testing session at post-test 0, 24, and 48 hours (POST-0, POST-24, POST-48). Subjects participated in 5 testing sessions: [1] orientation and 1 repetition maximum (1RM) back squat, [2] pre-test measurements (PRE), 10 x 10 squat protocol (weight: 60% 1RM, tempo: 4,1,1,1) with 2 minutes rest between sets, and post-test measurements (POST-0), along with measurements at: [3] POST-24, [4] POST-48, and [5] POST-72. Test measurements included: thigh girth, muscle soreness, range of motion (ROM), evoked and voluntary contractile properties, vertical jump, along with perceived pain (FR-pain) and reaction forces (FR-force) while foam rolling.

3.1.3 RESULTS:

Thigh girth showed no substantial between group differences at all time points. FR substantially reduced muscle soreness at all time points while substantially improving ROM. FR negatively affected evoked contractile properties (twitch force, rate of force development, and potentiated twitch force) with the exception of half-relaxation time (½RT) and electromechanical delay (EMD). ½RT showed no substantial between group differences at all time points, while FR substantially improved EMD. Voluntary contractile properties showed no substantial between group differences for all measurements besides voluntary muscle activation, with FR substantially improving muscle activation at all time points. FR improved functional movement, with substantial between group differences in vertical jump height. When performing the five FR exercises at the three time points (POST-0, POST-24, POST-48), subjects FR-force ranged between 26-46kg (32-55% of subjects' body weight) with FR-pain measurements (based on NRS) ranging between 2.5-7.5 pts.

3.1.4 CONCLUSION:

The most important findings of the present study were that FR was beneficial in attenuating muscle soreness while improving vertical jump height, muscle activation, and passive and dynamic ROM in comparison to CON. FR negatively impacted a number of evoked contractile properties of the muscle, except for ½ RT and EMD, indicating that FR benefits are primarily accrued through neural responses and connective tissue.

3.1.5 KEY WORDS:

self myofascial release, exercise-induced muscle damage, muscle activation, perceived pain, muscle soreness, recovery

3.2 INTRODUCTION:

Foam rolling (FR) is commonly used as a recovery tool following a bout of physical activity with advocates (3, 15) claiming that FR: corrects muscular imbalances, alleviates muscle soreness, relieves joint stress, improves neuromuscular efficiency, and improves range of motion (ROM). FR has been implemented into a number of different rehabilitation and training programs to help promote soft-tissue extensibility, enhance joint ROM, and promote optimal skeletal muscle functioning (3, 15, 25). Although FR has been strongly advocated and is commonly used, there have only been three peer-reviewed research articles published to date. Pearcey et al. (32) examined the effects of FR after an intense exercise protocol on pressure pain threshold and dynamic performance measures, concluding that FR is an effective method in reducing delayed onset muscle soreness (DOMS) and associated performance decrements in sprint time, power, and dynamic strength-endurance. MacDonald et al. (25) investigated the effects of acute FR prior to physical activity and demonstrated that FR had no effects on neuromuscular performance, while significantly increasing ROM at 2 and 10 minutes post-FR by 10 and 8%, respectively. Curran et al. (15) determined that a higher density foam roller significantly increased soft tissue pressure and isolated the soft tissue contact area, potentially increasing the effects foam rolling has on improving soft tissue health. Quantifiable scientific evidence to validate the use of foam rollers and understand the effectiveness of FR as a recovery tool from physical activity is rudimentary, thus it would be prudent to further investigate its effectiveness and mechanisms.

From the recreationally active to the elite athlete, many individuals commonly experience exercise induced muscle damage (EIMD) resulting in DOMS following an intense bout of physical activity. EIMD is characterized by muscle soreness, muscle swelling, temporary muscle damage, an increase in intramuscular protein and passive muscle tension, and a decrease in muscular strength and ROM (10, 37). In addition to these responses, EIMD can affect neuromuscular performance by reducing shock attenuation and altering muscle sequencing and recruitment patterns, potentially placing unaccustomed stress on muscle tendons and ligaments (10). There are a number of proposed theories regarding the mechanisms of DOMS, with the bulk of the literature reporting that high mechanical stress placed on the myofibrils, most commonly seen during eccentric exercise, damages the muscle tissue and connective tissue, triggering an acute inflammatory response consisting of edema and inflammatory cell infiltration that leads to a loss of cellular homeostasis, particularly due to high intracellular calcium concentrations (37). Sarcomere damage, calcium accumulation, protein degradation, and osmotic pressure all combine to sensitize nociceptors and other pain receptors, causing the sensation of DOMS (10). Through the analysis of a number of review articles, treatments that have shown potential benefits in treating symptoms of EIMD include: cryotherapy (12, 20, 37), light exercise (12, 37), and compression (10, 12). Although these therapies have shown to be beneficial in treating EIMD symptoms, the contrary has also been demonstrated in the literature (10, 12, 20, 37). On top of this, although these methods have shown to be beneficial in treating EIMD symptoms, no one therapy has proven to be beneficial in treating the full array of symptoms often present with EIMD.

Varying results demonstrated in the literature can be attributed to the varying EIMD protocols, along with the heterogeneity amongst studies assessing a given therapy in relation to the: dose, frequency, and intensity of the intervention.

Currently there is only one published article by Pearcey et al. (32) pertaining to the effects of FR on EIMD. Pearcey et al (32) analyzed the effects of FR on functional measures such as sprint speed, agility, broad jump, squat strength and pain threshold but did not examine the possible underlying mechanisms. FR has been referred to as a method of self-myofascial release or self-massage (25, 31, 32). Although there is little research on myofascial release, massage research may allow us to gain insight into the mechanisms and effects of FR on EIMD. Although massage has not been shown to be an effective method in improving range of motion (37, 40) or muscular strength (18, 37, 40) following EIMD, massage has been shown to be beneficial in treating EIMD by increasing mitochondrial biogenesis (13), restoring blood flow (35), and vertical jump height (26, 39), while decreasing muscle soreness (13, 18, 35, 40), cellular stress (13), and inflammation (13). Massage has also shown varying results in reducing limb circumference (37, 40) and creatine kinase levels (35, 37) while potentially increase circulating neutrophil counts (12, 35, 37).

With a number of studies showing the benefits of massage when treating EIMD, the purpose of our research was to substantiate if FR was an effective tool to aid in the recovery from an intense bout of physical activity that induces DOMS and identify potential mechanisms. We specifically addressed the effects of FR on: muscle soreness, voluntary and evoked contractile properties, vertical jump, and ROM. This investigation also explored the general characteristics of FR relating to force application and perceived pain during five different lower body FR exercises.

3.3 METHODS:

3.3.1 SUBJECTS:

Twenty physically active resistance trained male subjects volunteered for the study. All subjects regularly resistance trained 3+ times a week (1RM squat: 129.2 ± 26.7 kg, 1RM as % bodyweight: 152.2 ± 24.5%). Subjects were randomly assigned to an experimental "foam rolling" (FR) (n=10, height: 180.9 ± 5.5 cm, weight: 82.4 ± 9.4 kg, age: 25.1 ± 3.6 yrs., 1RM squat: 130.0 ± 20.6 kg) or "control" (CON) (n=10, height: 179.4 ± 4.0 cm, weight: 86.9 ± 8.6 kg, age: 24.0 ± 2.8 yrs., 1RM squat: 128.4 ± 32.9 kg) group. Written informed consent was obtained from all subjects. The Memorial University of Newfoundland Human Investigation Committee approved the study.

3.3.2 EXPERIMENTAL DESIGN:

All subjects were required to participate in 5 testing sessions, all occurring at the same time of day for each participant. The organization of the 5 testing sessions was: [1] Orientation & 1 repetition maximum (1RM) testing, [2] pre-test measurements (PRE), 10 x 10 squat protocol, post-test 0 (POST-0), [3] post-test 24 (POST-24), [4] post-test 48 (POST-48), and [5] post-test 72 (POST-72) hours. All testing sessions were separated by 24 hours, except sessions 1 and 2, which were separated by at least 96 hours, to ensure that subjects had recovered from the 1RM protocol.

In session 1, subjects were provided with a verbal explanation of the study, and read and signed an informed-consent form. Participants: age, height, weight, and thigh girth were recorded. Subjects were randomly assigned to one of two test groups: foam rolling (FR), or control (CON).

Once subjects were assigned to their test group, subjects' 1RM for a free-weight back squat was determined. The 1RM protocol consisted of a general warm-up on a stationary cycle ergometer with the resistance set at 1kp, cycling at a cadence of 70 revolutions per minute (rpm) for 5 minutes. The squat protocol required subjects to perform a full ROM (thighs parallel to floor) plate-loaded barbell back squat. Subjects were instructed to position the plate-loaded barbell above the posterior deltoids at the base of the neck.

Before attempting a 1RM lift, subjects performed a series of submaximal sets of 8, 5, and 2 repetitions with increasing loads. Subjects rested for 2 minutes between submaximal lift trials and for 3 minutes between each 1RM trial. If subjects successfully performed a squat with proper form, the weight was increased by approximately 1-10 kg, and the subject would attempt another squat at the new set weight. A failed lift was defined as a squat falling short of full ROM, or failure to complete the squat repetition. If a subject failed on two consecutive attempts, at a set weight, or felt that they had reached their 1RM, the previous successfully lifted weight was considered the subjects 1RM.

Once the subject's 1RM was determined, all subjects were familiarized with the EIMD protocol, along with the test measures and instruments used to conduct the experiment. Subjects in the FR group were also oriented with the FR exercise protocol (see FR section), instructed on how to perform the experimental exercise techniques, and given time to ask any questions they had regarding the FR exercise protocol.

In session 2, all subjects were required to complete a EIMD protocol consisting of 10 sets of 10 repetitions (reps) of back squats, with 2-minutes of rest between each set. The weight was set at 60% of their 1RM weight. Three subjects from each group failed to complete the EIMD protocol, implementing the 2-minute rest period between each set. Adequate rest was given to subjects, allowing them to complete all 100 reps outlined in the protocol. Two subjects were exempt for the study as they were unable to complete the required 100 reps. Prior to completing the EIMD protocol, subjects performed the same previously described 5 min cycle ergometer warm-up, followed by 2 sets of 5 reps, at 50% of their 1RM. Squats were performed at a tempo of 4s eccentric-1s pause at bottom-1s concentric-1s pause at top of lift, to focus on the eccentric phase of the lift for greater EIMD. Tempo was controlled using an interval timer, with the investigator signaling the subject regarding the changes in the lifting phase.

Testing sessions 2-5 had similar sequences. Upon entering the lab, subjects would have their thigh girth measured and perceived pain assessed. Subjects then performed the previously described 5 min stationary cycle ergometer warm-up. Subjects completed the vertical jump trials followed by a randomized allocation of the assessment of their maximal voluntary contractile (MVC) force, quadriceps and hamstrings ROM measurements. Vertical jump was not randomized and was tested prior to MVCs and ROM measurements as it is a bilateral movement, whereas MVCs were measured with the subjects' right leg, and ROM was measured with the subjects' left leg. Subjects performed 3 trials for each test measurement, with the best result being recorded. Session

2 differed slightly, as it included the EIMD protocol following the pre-test measurements, along with post-test (POST-0) measurements immediately following the EIMD protocol. Subjects in the FR group were required to perform the FR exercise protocol upon completion of the testing protocol at POST-0, POST-24, and POST-48 (FIGURE 3.7.1).

3.3.3 INDEPENDENT VARIABLES:

3.3.3.1 FOAM ROLLING:

Subjects in the foam rolling (FR) group performed 5 different foam rolling exercises, targeting the major muscle groups of the anterior, lateral, posterior, and medial aspect of the thigh, along with the gluteal muscles. A custom made foam roller that was constructed of a polyvinyl chloride pipe (10.16 cm outer diameter and 0.5 cm thickness) surrounded by neoprene foam (1 cm thickness) was used for all exercises, as greater pressure can be placed on the soft tissues of the body when using a high-density versus a low-density foam roller (15, 25). Subjects performed each of the 5 exercises on both the right and left leg, for two, 60-second bouts each. For exercises targeting the thigh (anterior, lateral, posterior, and medial), subjects were instructed to place their body weight on the foam roller, starting at the proximal aspect of the thigh and rolling down the thigh, using small undulating movements, gradually working their way towards the knee. Once the foam roller reached the distal aspect of the thigh, subjects were instructed to return the roller to the starting position in one fluid motion and continue the sequence for the remainder of the 60-second trial. For the fifth exercise, targeting the gluteal muscles, subjects were instructed to sit on top of the foam roller, placing both of their hands on the floor behind the foam roller. Subjects then crossed their right/left leg over their left/right knee, positioning their body so their right/left gluteal muscles were in contact with the roller and their body weight was placed on the foam roller. Subjects were instructed to undulate back and forth with the foam roller running inline with the origin to insertion point of the gluteus maximus muscle. Subjects completed all five exercises on one side of the body, and then switched to the other side of the body and repeated all five exercises.

3.3.4 DEPENDENT VARIABLES:

3.3.4.1 THIGH GIRTH:

Thigh girth (TG) was defined as the circumference at mid-thigh. Mid-thigh was defined as the halfway point between the anterior superior iliac spine and the proximal aspect of the patella. All measurements were taken when the subject was standing erect, with their right thigh muscles relaxed. A line was permanently marked around the circumference of the right thigh on the first day of testing to ensure measurements were reliable between testing sessions.

3.3.4.2 MUSCLE SORENESS:

Muscle soreness was measured using the BS-11 Numerical Rating Scale (NRS). The NRS allowed subjects to express the amount of pain, in reference to muscle soreness they perceived. The NRS is an 11-point scale ranging 0-10, with "0" being defined as "absolutely no muscle soreness", and "10" being defined as "the worst muscle soreness you have ever felt". Muscle soreness measurements were taken prior to each testing session. Subjects performed a squat using only body weight (no external resistance), squatting down until their thighs were parallel with the floor. Once subjects had assumed the squat position with their thighs parallel with the floor, subjects were then asked to rate their perceived pain based on muscle soreness.

3.3.4.3 RANGE OF MOTION:

To assess hamstrings and quadriceps range of motion (ROM), three measurements were taken at the knee and hip of the left leg. The three measurements included: quadriceps passive ROM (QP-ROM), hamstrings passive ROM (HP-ROM), and hamstrings dynamic ROM (HD-ROM). QP-ROM was measured by having subjects perform a modified kneeling lunge and measuring passive knee flexion angle using a manual goniometer (accurate to 1°), as outlined in a previous study (25). All landmark sites were marked with permanent marker and remained marked for all testing sessions to ensure reliability across trials. A decrease in the angle between the posterior aspect of the shank and thigh indicated an increase in quadriceps ROM.

Hamstrings ROM measurements were taken using a custom made electronic goniometer (Memorial University Technical Services, St. John's, Newfoundland) and analyzed using a software program (AcqKnowledge 4.1, BioPac Systems Inc. Hilliston, MA), measuring changes in ROM at the hip. Subjects' left leg was equipped with a knee brace to prevent movement at the knee joint, and to isolate the hip. Subjects stood erect, and were strapped to a wooden platform harnessed to the wall. Three straps placed around the right ankle, right thigh, and across the chest harnessed the subject to the wooden platform. HP-ROM was assessed by having the investigator passively flex the subject's right hip until the subject reached a point of maximum discomfort. HP-ROM measurements have been reported to have a high intraclass correlation coefficient reliability (r=0.96) (30). HD-ROM was assessed with the same harnessing and knee brace on the custom made electronic goniometer by having subjects contract their hip flexors and kick up as high and as fast as possible.

3.3.4.4 EVOKED CONTRACTILE PROPERTIES:

Peak twitch force was evoked with electrodes connected to a high-voltage stimulator (Stimulator Model DS7AH+; Digitimer, Welwyn Garden City, Hertfordshire, UK), as outlined in a previous study (33). Stimulating electrodes were placed over the inguinal triangle (proximal) and directly above the patella (distal) of the right leg. To determine the peak twitch force, voltage was sequentially increased (100 - 300 volts) until a maximum twitch force was achieved. The amperage (1amp (A)) and duration (50 micro-seconds (μs)) was kept constant throughout. Once peak twitch was achieved, the voltage used to achieve the peak twitch force was maintained throughout the testing session.

An evoked twitch was administered 2 s prior to the subject's MVC, allowing peak twitch force (TF), electromechanical delay (EMD), rate of force development (RFD), and half relaxation time (½RT) to be analyzed. An evoked twitch was also administered 2 s post-MVC to analyze potentiated twitch characteristics. EMD was defined as the time period between the onset of muscle stimulation and the onset of twitch force production (>1% the twitch amplitude from baseline) (2). RFD was measured over a 50ms window, beginning at the onset of twitch force development, defined as force deviating >1% the twitch amplitude from baseline force measurements (2). ½RT was defined as the time required for the twitch force to decrease from peak twitch force to 50% of peak twitch force (38). Potentiated twitch force (PTF) was defined as the peak force produced from an evoked twitch elicited 2 s following the MVC.

3.3.4.5 VOLUNTARY CONTRACTILE PROPERTIES:

Maximal Voluntary Contractile Force (MVC force) was assessed via an isometric knee extension (knee angle: 90°) of the right leg. MVC force protocol followed methods outlined in a previous study (33). Button & Behm (7) reported that MVCs demonstrated excellent day-to-day reliability (r=0.99). Prior to attempting an MVC, subjects performed 2 submaximal contractions. Subjects performed three, 3-5 second MVCs, separated by 2 minutes each, with all forces detected by the strain gauge, amplified (Biopac Systems Inc. DA 150, and analog to digital (A/D) converter MP150WSW; Hilliston, MA), and displayed on a computer monitor. Data was sampled at 2000 Hz and analyzed using a software program (AcqKnowledge 4.1, BioPac Systems Inc. Hilliston, MA). In order to ensure subjects were performing to their maximal effort, subjects had to perform two MVCs with no greater than 5% variance in force outputs between trials (7).

Verbal encouragement was given to all subjects during the MVC to provide motivation.

Muscle Activation (VA) was assessed using the interpolated twitch technique (ITT). ITT was utilized to measure muscle activation and the central nervous systems (CNS) ability to fully activate the contracting muscle (5). The voltage used to elicit the peak twitch force was employed during the MVC to provide an interpolated or superimposed twitch. ITT methods administrated were similar to previous studies (4, 7, 33). ITT was performed using two evoked twitches: [1] an interpolated twitch once subjects reached their maximal force output, determined via visual inspection (once the subject's MVC force output plateaued), and a potentiated twitch approximately 2 s post-MVC. An interpolated twitch ratio was calculated comparing the amplitude of the interpolated twitch with the potentiated twitch to estimate the extent of activation during a voluntary contraction ([1 - (interpolated doublet force/potentiated doublet force)] x 100 = % of muscle activation) (4).

Integrated electromyography (iEMG) activity was used as a measure of peripheral muscle activation. Surface EMG recording electrodes (Tyco Healthcare Group LP, Meditrace 133, ECG Conductive Adhesive Electrodes, Mansfield, MA) were placed on the right leg, over the muscle belly of the rectus femoris. Electrodes were placed at half the distance between the anterior superior iliac spine of the pelvis and the patella. A ground electrode was secured on the fibular head. Thorough skin preparation for all electrodes was administered (33). EMG activity was sampled at 2000Hz, with a Blackman -92 dB band pass filter between 10-500 Hz, amplified (bi-polar differential amplifier, input impedance = 2 MΩ, common mode ejection ratio > 110 dB min (50/60 Hz), gain X 1000, noise > 5 μV) and was analog-to-digitally converted (12 bit) and stored on a personal computer for analysis. iEMG was measured for a one second period during subject's peak MVC force (0.5 s pre- and post-maximum force output).

3.3.4.6 VERTICAL JUMP HEIGHT:

Vertical Jump testing followed the vertical jump protocol outlined in the Canadian Physical Activity, Fitness and Lifestyle Approach (CPAFLA) manual (14). One modification was made to the protocol. Instead of pausing at the bottom of the squat, subjects were instructed to perform a countermovement jump (CMJ). Subjects were given the same instructions before performing each CMJ for the entirety of the study. Depth and speed of the countermovement was not controlled to allow the movement to be as natural as possible. Permanent marker was placed on the tip of the subject middle finger. The difference between subjects standing reach height and CMJ height was recorded as the subjects vertical jump height. Visual inspection of the marking as it lined up with the measuring tape was recorded to the nearest 0.1 cm.

3.3.4.7 FOAM ROLLING FORCE:

Force placed on the foam roller (FR-force) was measured using a force plate (Advanced Mechanical Technology, Inc. Biomechanics Force Platform model BP400600HF, Watertown, Massachusetts, USA). The force plate contains four load cells which measure the three orthogonal force and moment components along the X, Y and Z axis', producing a total of six outputs (Fx, Fy, Fz, Mx, My, Mz). Subjects foam rolled over the force plate, with the force plate being the only point of contact for the roller. Subjects were instructed to perform the FR exercise protocol, keeping the foam roller on the force plate, while keeping all body parts off the force plate. Force was analyzed using the force measurements recorded in the Fz-plane over each 60-second trial, collected at a sampling rate of 60 Hz.

3.3.4.8 FOAM ROLLING PAIN:

Perceived pain while foam rolling (FR-pain) was measured while subjects performed the FR exercise protocol using the NRS. The NRS 11-point scale ranged from 0-10, with "0" being defined as "absolutely no pain", and "10" being defined as "the worst pain you have ever felt". At 30-seconds into each 60-second FR trial, subjects rated their perceived pain for each of the five FR exercises.

3.3.5 STATISTICAL ANALYSIS:

To avoid the shortcomings in relation to the clinical significance of the present research based on null-hypothesis significance testing, magnitude-based inferences and precision of estimation were employed (21). Magnitude-based inferences on the interaction effects in the mean changes between the intervention trials (CON and FR) were determined. The interaction effect of time and FR was calculated from the mean difference between PRE and each time-point (PRE to POST-24, 48, and 72) for CON and FR. The two differences were then subtracted to estimate the effect of FR at each time point.

Qualitative descriptors of standardized effects were assessed using these criteria: trivial < 0.2, small 0.2-0.5, moderate 0.5-0.8, and large> 0.8 (11). Precision of estimates is indicated with mean difference ±95% confidence limits, which defines the range representing the uncertainty in the true value of the (unknown) population mean.

We were interested in practical differences between groups and time, so we based the smallest worthwhile change on a small effect size (>0.2). The likelihood that the observed effect size was larger than the smallest worthwhile change (i.e., was clinically meaningful) was calculated based on previous methods. Chances of clinically meaningful difference were interpreted qualitatively as follows: <1%, almost certainly not; <5%, very unlikely; <25%, unlikely; 25–75%, possible; >75%, likely; >95%, very likely; and >99% almost certain (21). Therefore, results are expressed by the percent change from pre-test (PRE) measurements (% Δ), percent likelihood that the observed between group difference was greater than a small effect size (% likelihood), and the effect size. Results with a >75% likelihood were considered to be substantial.

3.4 RESULTS:

3.4.1 FATIGUE: (EIMD)

The prescribed EIMD protocol induced substantial changes in all the dependent variables except HP-ROM (-1%, unclear, trivial, ***% Δ, % likelihood, effect size***) and HD-ROM (0%, unclear, trivial). The DOMS protocol induced a substantial increase in TG (2%, >99%, small), along with a number of performance decrements in the dependent variables measured: QP-ROM (-7%, 76%, small), twitch force (-40%, >99%, large), RFD

(-39%, >99%, large), potentiated twitch force (-41%, >99%, large), VJ (-13%, >99%, large), MVC force (-24%, >99%, large), muscle activation (-9%, >99%, large), and iEMG (-14%, 96%, small). EMD (-6%, 95%, moderate) and ½RT (-22%, >99%, moderate) were the only two dependent measures to show improvements immediately following the DOMS protocol (FIGURE 3.7.2).

3.4.2 THIGH GIRTH:

TG, with PRE measurements of, FR: 58.3 ± 2.7 cm and CON: 59.1 ± 4.2 cm, showed no substantial between group differences at: POST-24 (FR: 1%, CON: 1%, 0.03 ± 0.31 cm, ***FR: % Δ CON: % Δ, Mean Difference ± 95% CI)***, POST-48 (FR: 3%, CON: 3%, 0.03 ± 0.65 cm), and POST-72 (FR: -1%, CON: -2%, 0.03 ± 0.51 cm) with all time points showing "unclear" results regarding whether FR is beneficial or detrimental due to trivial effect sizes (POST-24: -0.09 ± 0.96, POST-48: -0.04 ± 0.96, POST-72: -0.06 ± 0.97, ***d ± 95% CI***).

3.4.3 MUSCLE SORENESS:

Muscle soreness, recorded prior to each testing session, with PRE measurements of, FR: 0.7 ± 1.0 points and CON: 0.7 ± 1.1 points, showed substantial between group differences at: POST-24 (FR: 543%, CON: 714%, ***% Δ***) with FR (85%, ***% likelihood***) having a substantial effect in reducing muscle soreness, demonstrating a "moderate" effect size, POST-48 (FR: 414%, CON: 807%), and POST-72 (FR: 243%, CON: 607%) with FR (48hrs: 98%, 72hrs: 97%) having a substantial effect in reducing muscle soreness, demonstrating a "large" effect size, based on NRS measurements (FIGURE 3.7.3).

3.4.4 RANGE OF MOTION:

Quadriceps passive ROM (QP-ROM) PRE measurements were, FR: 59.0 ± 12.8° and CON: 64.7 ± 14.6°. QP-ROM showed no substantial between group differences at POST-24 (FR: 8%, CON: 5%), but showed substantial differences at POST-48 (FR: 11%, CON: 0%) and POST-72 (FR: 13%, CON: 4%) with FR increasing QP-ROM, demonstrating a "moderate" effect size (TABLE 3.8.1).

Hamstrings passive ROM (HP-ROM) PRE measurements were, FR: 111.2 ± 6.9° and CON: 103.8 ± 14.8°. HP-ROM showed no substantial between group differences at POST-24 (FR: -1%, CON: -3%) and POST-48 (FR: 0%, CON: 0%), but showed substantial differences at POST-72 (FR: 3%, CON: 0%) with FR increasing HP-ROM, demonstrating a "moderate" effect size (TABLE 3.8.1).

Hamstring dynamic ROM (HD-ROM), with PRE measurements of FR: 105.5 ± 6.2° and CON: 98.0 ± 11.3° showed substantial between group differences at POST-24 (FR: 0%, CON: -4%) with FR increasing HD-ROM, demonstrating a "moderate" effect size, but showed no substantial differences at POST-48 (FR: 0%, CON: -3%) and POST-72 (FR: 1%, CON: -1%) (TABLE 3.8.1).

3.4.5 EVOKED CONTRACTILE PROPERTIES:

Twitch force (TF), with PRE measurements of FR: 153.4 ± 34.8 N and CON: 135.7 ± 27.8 N showed substantial between group differences at: POST-24 (FR: -14%, CON: -5.%), POST-48 (FR: -9%, CON: 8%), and POST-72 (FR: -10%, CON: -3%) with FR reducing TF, demonstrating a "moderate", "large", and "moderate" effect size, respectively (TABLE 3.8.2).

Electromechanical delay (EMD) PRE measurements were FR: 46.1 ± 4.7 ms, and CON: 46.4 ± 4.4 ms. EMD showed substantial between group differences at POST-24 (FR: -2%, CON: 7%) and POST-48 (FR: -1%, CON: 6%) with FR shortening EMD duration, demonstrating a "moderate" effect size, but showed no substantial differences at POST-72 (FR: 2%, CON: -2%) (TABLE 3.8.2).

Rate of force development (RFD), with PRE measurements of FR: 1852.6 ± 478.8 $N \bullet s^{-1}$ and CON: 1488.5 ± 460.5 $N \bullet s^{-1}$ showed substantial between group differences at POST-24 (FR: -23%, CON: 5%) and POST-48 (FR: -17%, CON: 15%) with FR reducing RFD, demonstrating a "large" effect size, but showed no substantial differences at POST-72 (FR: -10%, CON: -4%) (TABLE 3.8.2).

Half relaxation time (½RT) PRE measurements were FR: 68.4 ± 21.0 ms and CON: 64.6 ± 20.8 ms. ½RT showed no substantial between group differences at POST-24 (FR: 9%, CON: 1%), POST-48 (FR: 7%, CON: -5%), and POST-72 (FR: 6%, CON: -3%) (TABLE 3.8.2).

Potentiated twitch force (PTF), with PRE measurements of FR: 223.2 ± 31.4 N and CON: 183.6 ± 31.8 N showed no substantial differences at POST-24 (FR: -7%, CON: -6%), but showed substantial between group differences at POST-48 (FR: -5%, CON: 9%) and POST-72 (FR: -6%, CON: 1%) with FR decreasing PTF, demonstrating a "large" and "moderate effect size at POST-48 and POST-72, respectively (TABLE 3.8.2).

3.4.6 VOLUNTARY CONTRACTILE PROPERTIES:

Maximal voluntary contractile force (MVC force), with PRE measurements of, FR: 761.4 ± 126.3 N and CON: 621.9 ± 100.9 N, showed no substantial between group differences at: POST-24 (FR: -9%, CON: -12%), POST-48 (FR: -6%, CON: -6%), and POST-72 (FR: -7%, CON: -6%), demonstrating a "trivial" effect size at all time-points (TABLE 3.8.2).

Voluntary muscle activation (VA) PRE measurements were FR: 93.3 ± 4.2% and CON: 91.9 ± 4.0%. VA showed substantial between group differences at: POST-24 (FR: 0%, CON: -4%), POST-48 (FR: 1%, CON: -5%), and POST-72 (FR: 1%, CON: -2%) with FR increasing VA, demonstrating a "moderate", "large", and "moderate" effect size, respectively (TABLE 3.8.2).

Integrated electromyography (iEMG), with PRE measurements of FR: 0.43 ± 0.11 mV/s and CON: 0.42 ± 0.16 mV/s, showed no substantial between group differences for POST-24 (FR: -2%, CON: -7%), POST-48 (FR: -4%, CON: 0%), and POST-72 (FR: -9%, CON: -2%) (TABLE 3.8.2).

3.4.7 VERTICAL JUMP HEIGHT:

Vertical jump height PRE measurements were FR: 51.7 ± 7.9 cm and CON: 46.5 ± 7.0 cm. Vertical jump height approached substantial between group differences at POST-24 (FR: 0%, CON: -6%) with FR increasing vertical jump height, demonstrating a "small" effect size, and showed substantial between group differences for POST-48 (FR: 1%, CON: -5%) with FR increasing vertical jump height, demonstrating a "large" effect size, but showed no substantial differences at POST-72 (FR: 0%, CON: 0%) (TABLE 3.8.2).

3.4.8 FOAM ROLLING FORCE:

Foam roller force (FR-force) averaged between 28-46 kg or 35-55% of the subjects' body weight at POST-0, 26-44 kg or 32-53% of the subjects' body weight at POST-24 and 26-44 kg or 32-53% of the subjects' body weight at POST-48 (TABLE 3.8.3 & 3.8.4).

FR-force showed substantial time differences between POST-0 and POST-24 for the anterior (-10%, moderate, **% Δ, effect size**), lateral (-15%, large), medial (-7%, small), and gluteals (-8%, moderate) foam rolling exercises, with no substantial differences for the posterior (2%, trivial) exercise (TABLE 3.8.3A).

FR-force showed no substantial between time differences between POST-24 and POST-48 for the anterior (-6%, small), lateral (-1%, trivial), posterior (0%, trivial), medial (-2%, trivial), and gluteals (2%, trivial) foam rolling exercises (TABLE 3.8.3B).

3.4.9 FOAM ROLLING PAIN:

Foam roller perceived pain (FR-pain) ranged between 2.5-7.5 pts. at POST-0, 3-7.5 pts. at POST-24, and 2.5-6.5pts. at POST-48 on the NRS for the five different FR exercises (TABLE 3.8.3).

FR-pain showed substantial time differences between POST-0 and POST-24 for the medial (19%, moderate), and gluteals (24%, moderate) foam rolling exercises, with no substantial differences for the anterior (5%, small), lateral (2%, trivial), and posterior (11%, small) exercises (TABLE 3.8.3A).

FR-pain showed substantial time differences between POST-24 and POST-48 for the anterior (-16%, moderate), lateral (-12%, moderate), medial (-8%, small), and gluteals

(-15%, small) foam rolling exercises, with no substantial differences for the posterior (10%, small) (TABLE 3.8.3B).

3.5 DISCUSSION:

Pearcey et al. (32) is the only research article to analyze the effects of foam rolling on recovery from EIMD resulting in DOMS. No research to date has examined the potential physiological mechanisms regarding the recovery benefits seen with foam rolling that have been outlined in previous literature (32). The most important findings of the present study were that FR was beneficial in improving dynamic movement, percent muscle activation, and both passive and dynamic ROM in comparison to the CON group, while attenuating muscle soreness, although no benefits were seen at the muscular level when it was isolated.

3.5.1 EIMD PROTOCOL:

Similar to previous EIMD related studies (19, 34), substantial muscular fatigue and damage was inflicted by the EIMD protocol resulting in a substantial increase in thigh girth along with substantial decrements in: QP-ROM, TF, RFD, PTF, vertical jump height, MVC force, muscle activation, and iEMG. Only two muscle properties showed improvements immediately post-exercise, with a reduction in the duration of EMD and ½RT.

3.5.2 MUSCLE SORENESS: (DOMS)

In the FR group, muscle soreness peaked at POST-24, whereas the CON group peaked at POST-48. Results are parallel to Smith et al. (35), findings comparing a massage intervention group to a control group. The massage group reported peak muscle soreness at POST-24, whereas the control group peaked at POST-48. In the present study, substantially higher muscle soreness readings were recorded at all time points (pts.) for the CON group, showing the effectiveness of FR in reducing muscle soreness. Reductions in muscle soreness readings can be further supported by the force plate data collected from the FR exercise protocol (Table 4B) at POST-24 and POST-48. While the FR group showed no substantial changes in FR-force between POST-24 (26-44 kg) and POST-48 (26-44 kg) for all five foam roller exercises, substantial decreases in FR-pain (POST-24: 3-7.5 pts. and POST-48: 2.5-6.5 pts.) were seen while performing four out of the five exercises. This finding further supports that muscle soreness peaked at POST-24 for the FR group, and then began to return to baseline levels. The improved recovery rate in muscle soreness in the FR group signifies that FR is an effective tool to treat DOMS.

DOMS has been attributed to both muscle (8, 12, 29, 36) and connective (17, 23, 28, 36) tissue damage. Although DOMS is associated with muscle cell damage, it is unlikely that DOMS is the direct result of muscle cell damage (36) as muscle enzyme efflux and myofibrillar damage are not correlated with the actual sensation of muscle soreness (10, 23). It has been postulated that DOMS may be the result of connective tissue damage more so than muscle damage. Connolly et al. (12) stated that pain and stiffness may be more related to the inflammatory response (13), as a result of cells and fluid moving into the interstitial spaces rather than the actual muscle damage incurred. This can be supported by Mills et al. (28), who demonstrated the presence of muscle damage without the presence of muscle soreness, as well as the presence of muscle soreness without muscle damage. Jones et al. (23) suggested that changes in connective tissue properties to be the main cause of DOMS, with the myotendinous junction being the predominant area of soreness (24). Previous studies have reported connective tissue breakdown following eccentric exercise (1, 24), with damaged connective tissue stimulating mechanically sensitive receptors, giving rise to pain when stretched or pressed (23). This finding suggests that the benefits of foam rolling may be more predominant for the treatment of connective tissue rather than muscle tissue damage.

3.5.3 EVOKED CONTRACTILE PROPERTIES:

Evidence that foam rolling has a greater effect on connective tissue rather than muscle can be further strengthened by the greater decrement in evoked contractile properties with FR vs. CON. Decrements in TF, PTF, and RFD in the FR group may be a result of increased muscle damage from the foam rolling protocol. Callaghan (8) showed that after a vigorous massage protocol, an increase in lactate dehydrogenase and creatine kinase was reported, both being markers of muscle damage. Zainuddin et al. (40) and Crane et al. (13) both showed that massage was effective in alleviating DOMS, although Zainuddin et al. (40) found that massage had no effect on muscle function. The present findings suggest that although foam rolling may be beneficial in treating connective tissue damage, minor damage to muscle tissue may incur. Whether this is beneficial for the repair process occurring in the muscle, or only causing more damage to the muscle is unknown (10).

The only evoked contractile property to benefit from the FR protocol in comparison to the CON group was EMD, being substantially shorter at POST-24 (9%) and POST-48 (7%) when compared to CON. EMD has been shown to be influenced by a number of factors including: series elastic components, ability of the action potential to propagate, and excitation contraction coupling (22). Zhou et al. (41) considers EMD to reflect the elastic properties of the muscle with the major portion of the EMD representing the time required to stretch the series elastic components of the muscle (9). Following EIMD, elongation of EMD is believed to be due to mechanical stress placed on the muscle and increased passive tension on non-contractile structures in the myofibers (29), resulting in connective tissue damage. Based on these findings, with FR improving the recovery of EMD to baseline measurements following EIMD, potentially by restoring the passive non-contractile structures (series of elastic components) in the muscle, it is likely that FR provides a more clinically significant recovery effect upon connective versus muscle tissue following EIMD.

3.5.4 VOLUNTARY CONTRACTILE PROPERTIES:

Although foam rolling did not help in treating EIMD at the muscular level, the FR group showed no decrements in voluntary properties in comparison to the CON group and showed substantially greater muscle activation in comparison to the CON group from POST-24 to POST-72. The most substantial between group difference in muscle activation was seen at POST-48, occurring when muscle soreness was also at its most substantial between group difference of any of the three post-test time points. At POST-48, the FR group also showed its greatest decrement in TF and PTF loss in comparison to the CON group. This being said, FR group may have been able to maintain muscle activation where decrements were seen in the CON group, potentially due to a substantial reduction in DOMS and less neural inhibition as a result of healthier connective tissue allowing for appropriate afferent feedback from mechanical and sensory receptors located within the connective tissue enveloping the muscle (12, 19). To our knowledge, there is no information on the effects of massage on muscle activation, although the decrements seen in the CON group were similar to previous EIMD studies (19, 34).

There were no substantial iEMG between group differences. This may seem perplexing as muscle activation showed substantial between group differences. This finding may be the result of large inter-individual variations in EMG levels when analyzing sore muscles (6). McGlynn et al. (27) showed that EMG standard deviations increased by 25-fold from PRE to POST-72. Along with the above noted findings, Abraham (1) demonstrated no changes in EMG readings using bipolar electrodes, with de Vries (16) claiming that bipolar electrodes may not be sensitive enough to detect changes in EMG. Although not evident with iEMG measures, the improved muscle activation may be the result of decreased neural inhibition (12, 19) associated with less pain, due to a decrease in inflammation (13).

MVC force showed no substantial between group differences with both groups showing deficits (6-12%) at all time points, and not recovering to PRE measures by POST-72. MVC force deficits are similar to those reported in a previous EIMD study

(40). It is interesting to note that there was no substantial between group differences in MVC force even though the FR group showed substantial decrements in TF, demonstrating greater damage within the muscle (19). Although muscle fibers produced less force (as seen through TF measurement) in the FR group, most likely a result of greater individual muscle fibers damage (29), the subjects' ability to activate a greater number of muscle fibers (as seen through VA measurements) may have acted to counterbalance the decrement in force production per fiber (34).

3.5.5 RANGE OF MOTION:

FR was beneficial in improving both passive and dynamic ROM in comparison to the CON group. FR increased passive ROM (QP-ROM and HP-ROM) and maintained dynamic ROM (HD-ROM) in relation to PRE measures (TABLE 1). EIMD research (23, 34) attributes a loss in ROM to the shortening of non-contractile elements. Previously published research from our laboratory demonstrates that the application of FR increases ROM (25). Improved ROM was attributed to FR acting in a similar fashion to myofascial release techniques, potentially: reducing muscle soreness, decreasing inflammation, and/or reducing adhesions between layers of fascia (3, 15). Muscular manipulation has been shown to promote active blood flow and move interstitial fluid back into circulation, reducing inflammation and muscle soreness (8, 13).

3.5.6 VERTICAL JUMP HEIGHT:

Vertical jump performance incorporates all three (muscle, CNS, ROM) of the major properties analyzed in the present study. Similar to Pearcey et al. (32) findings, the

FR group showed substantial benefits in comparison to the CON group when assessing dynamic performance at POST-24 and POST-48. Willems et al. (39) and Mancinelli et al. (26) research supports these findings, with massage shown to improve vertical jump height at 48 hours post exercise by 3% and 4.5%, respectively. Farr et al. (18) contradicts the present findings showing decrements in vertical jump height at POST-24 in the massage group, although there were no differences between the massage and control limb. It must be noted that Willems et al. (39) and Farr et al. (18) vertical jump tests consisted of 1-legged vertical jumps, with the contralateral leg acting as the control. Since evoked contractile properties are not improved by FR, FR likely acts by reducing neural inhibition (12, 34) due to accelerated recovery of the connective tissue, as a result of decreased inflammation and increased mitochondria biogenesis (13), decreasing nociceptor activation (17), allowing for better communication from afferent receptors in the connective tissue (34). Better communication with afferent receptors, may possibly allow for the maintenance of natural muscle sequencing and recruitment patterns (34) maintaining vertical jump height.

3.6 CONCLUSION:

From the present findings, it is speculated that FR provides recovery benefits primarily through the treatment of connective tissue. As the present evidence is indirect, further research should be conducted.

The FR group displayed substantially less pain at all time points in comparison to the CON group. Since connective tissue (i.e. myotendinous junction) is the major site of EIMD disruption and pain (17, 23, 24, 36), FR can be considered to be beneficial in the recovery of connective tissue. Crane et al. (13) research supports this finding, reporting that massage decreased pain and inflammation, potentially by promoting blood flow to areas of low blood flow, such as the muscle tendon interface. The FR group recorded substantially less muscle soreness, while having substantially greater decrements in evoked contractile properties, ruling out improved muscle recovery as the determining factor. The reduction in pain with FR may have been an influential factor for the maintenance of muscle activation (i.e. less neural inhibition) (12, 34). When analyzing dynamic movements (vertical jump height, HD-ROM) or EMD, all heavily involving the series elastic components, FR proved to be beneficial. When comparing isometric (MVC Force) versus dynamic contraction (vertical jump height) results in the FR group, the greatest benefits from FR were displayed in the dynamic movement. It must be emphasized that the majority of benefits seen with FR following EIMD are the result of FR maintaining rather than an improving PRE measures (VA, EMD, HD-ROM, vertical jump height), where the CON group incurred substantial decrements.

3.7 FIGURES:

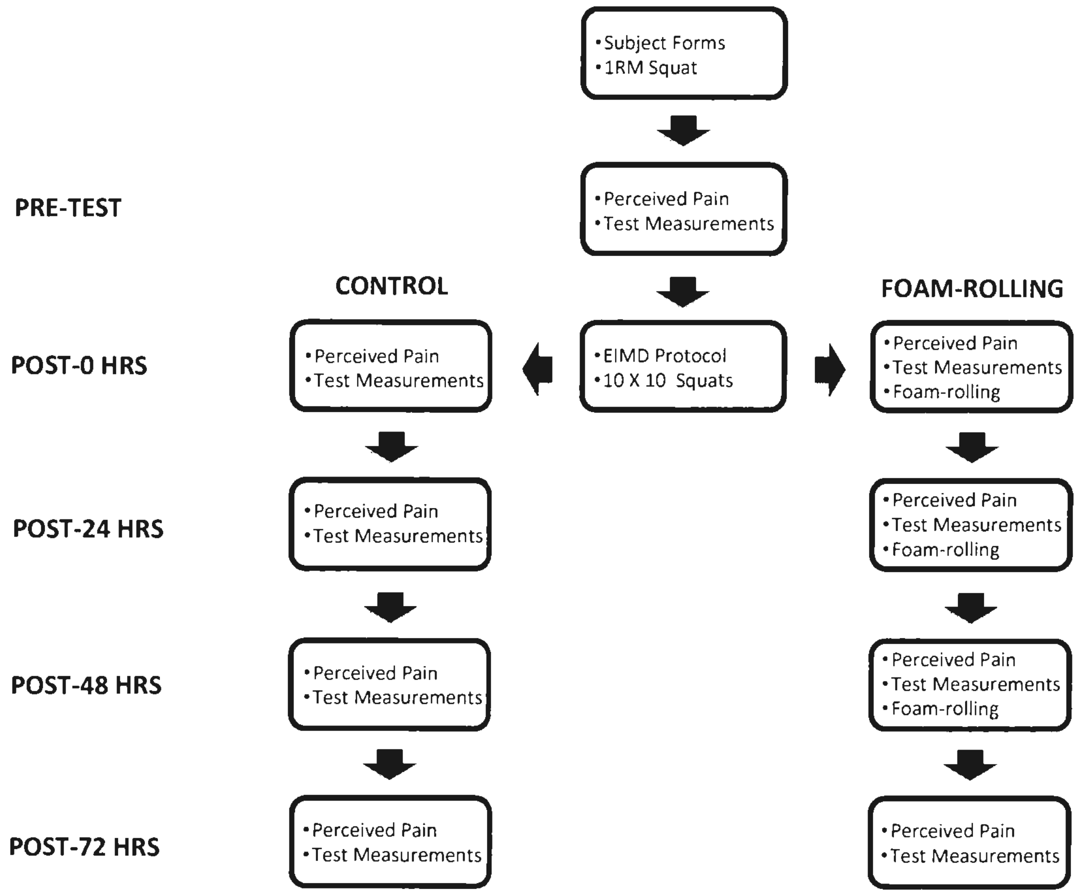

FIGURE 3.7.1: METHODOLOGY

Flow chart displays methodology along with the dependent variables assessed each time test measurements were taken.

FIGURE 3.7.2: CHANGES INDUCED BY EIMD PROTOCOL

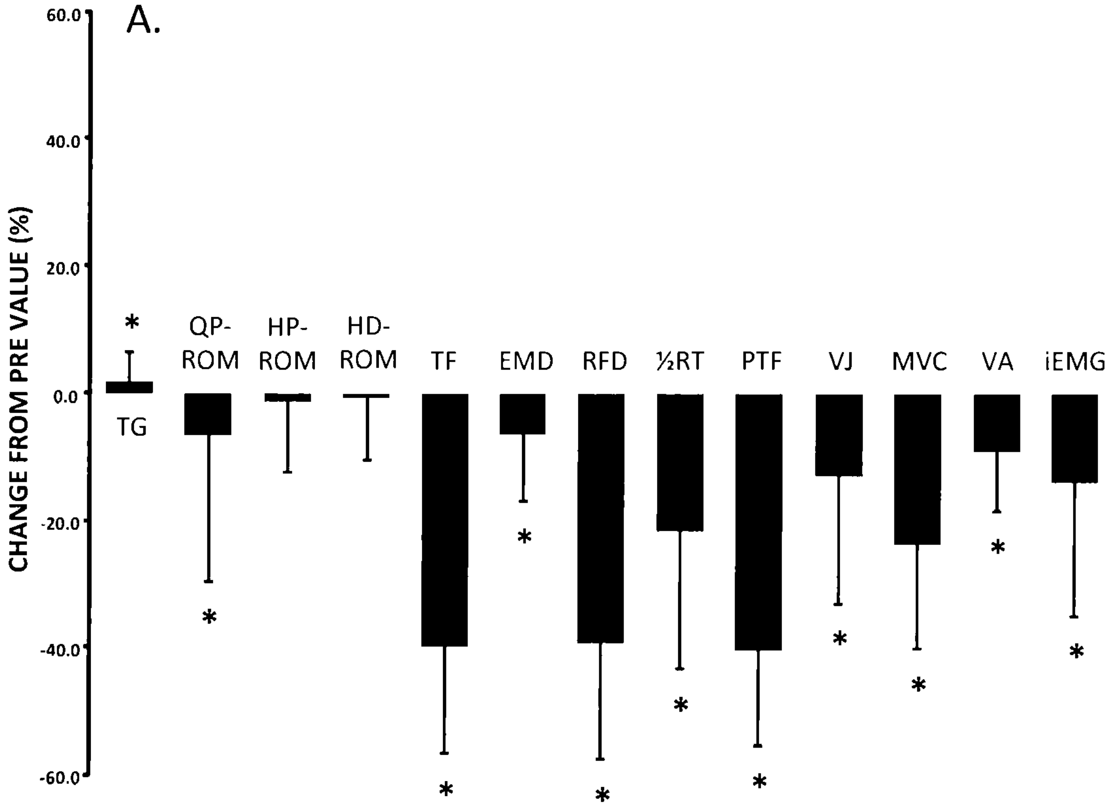

FIGURE 3.7.2A:

TG: Thigh Girth, **QPR:** Quadriceps Passive ROM, **HPR:** Hamstrings Passive ROM, **HDR:** Hamstrings Dynamic ROM, **TF:** Twitch Force, **EMD:** Electromechanical Delay, **RFD:** Rate of Force Development, **½RT:** Half-Relaxation Time, **PTF:** Potentiated Twitch Force, **VJ:** Vertical Jump, **MVC:** Maximal Voluntary Contractile Force, **MA:** Muscle Activation, **iEMG:** Integrated Electromyography.

The y-axis displays % Δ from pre-test measurements. The x-axis displays the dependent variables. Asterisks (*) indicate conditions with substantial change (>75% likelihood that the difference exceeds the smallest worthwhile difference).

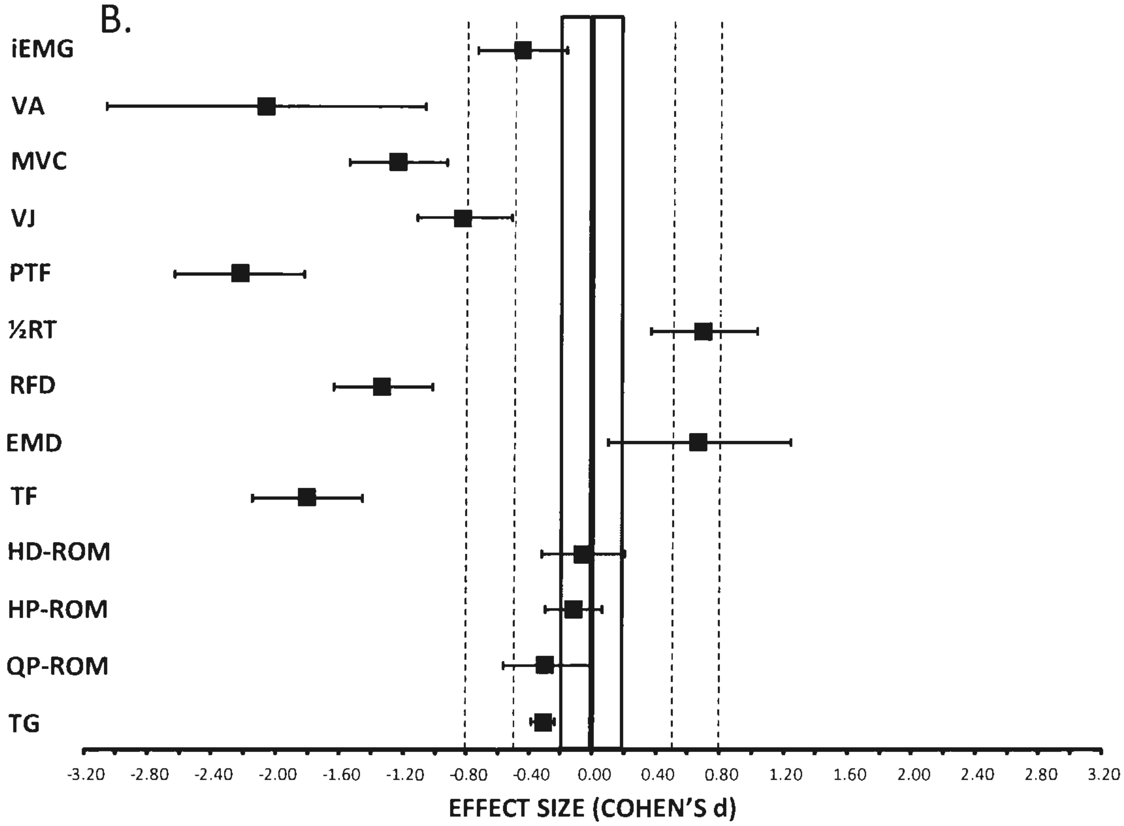

FIGURE 3.7.2B:

Graph plots standardized effect size differences between CON and FR groups. Plots represent the magnitude of difference between the two groups. Error bars indicate 95% confidence limits of the mean difference between groups. The shaded area of the graph indicates the region in which the difference between groups is trivial (i.e. between -0.20 and 0.20 standardized effect sizes).

FIGURE 3.7.3: MUSCLE SORENESS

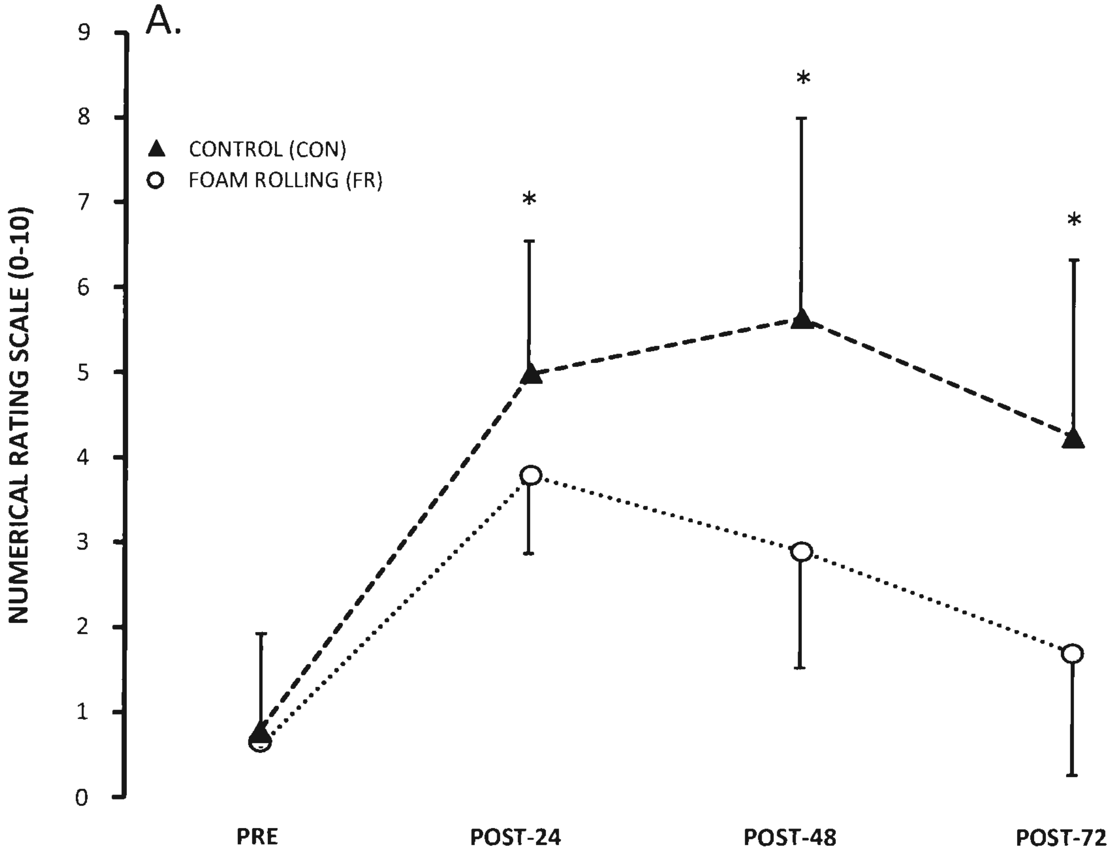

FIGURE 3.7.3A:

The y-axis displays muscle soreness based on the NRS. The x-axis displays pre-test and post-test measurements for the 4 different time points. Asterisks (*) indicate conditions with substantial change (>75% likelihood).

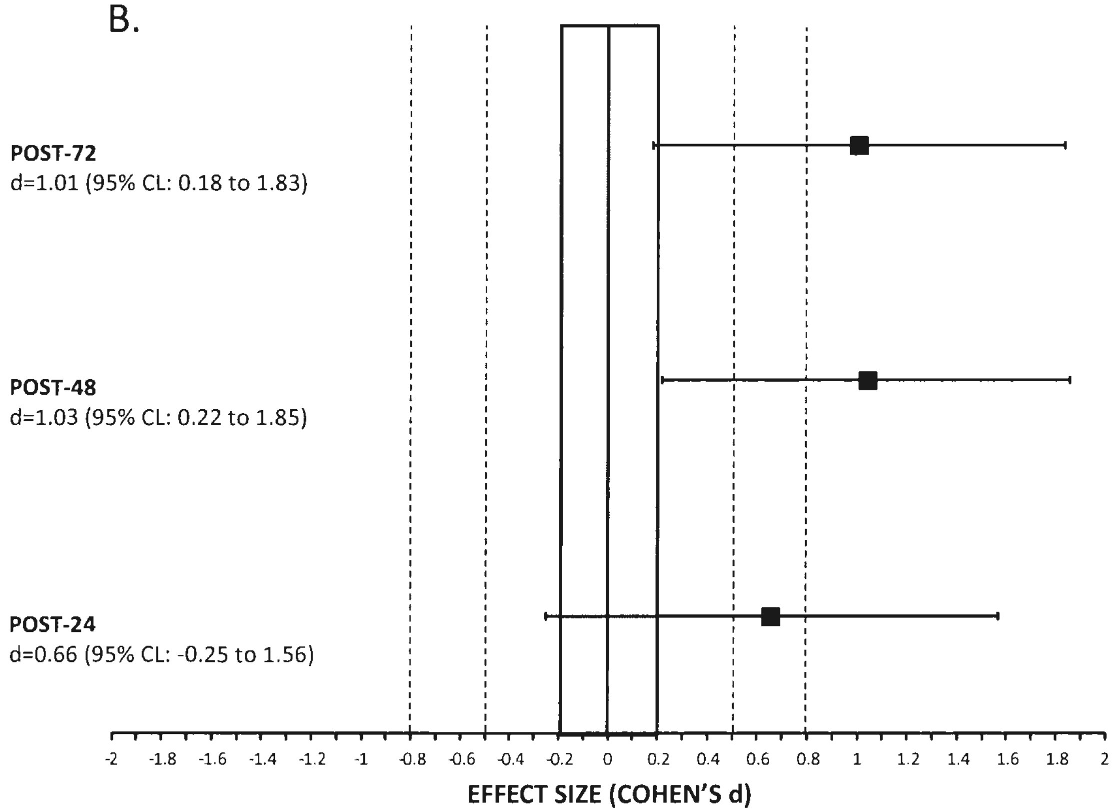

FIGURE 3.7.3B:

Please reference FIGURE 3.7.2B description.

3.8 TABLES:

TABLE 3.8.1: ROM

Measurement	Time	% Likelihood	↓ / ↑	Mean Diff	Lower 95% CL	Upper 95% CL	d	Lower d	Upper d
QP-ROM (°)	POST-24	UNCLEAR		1.3	-6.18	8.78	0.17	-0.79	1.1
	POST-48	**90**	↑	6.6	-0.96	14.16	0.77	-0.11	1.66
	POST-72	**79**	↑	4.95	-3.24	13.14	0.56	-0.37	1.48
HP-ROM (°)	POST-24	60	↑	1.5	-3.07	6.08	0.31	-0.6	1.27
	POST-48	UNCLEAR		0.04	-4.8	4.71	-0.01	-0.97	0.96
	POST-72	**83**	↑	3.4	-1.62	8.42	0.62	-0.3	1.53
HD-ROM (°)	POST-24	**79**	↑	3.9	-2.43	10.22	0.57	-0.4	1.49
	POST-48	65	↑	3.03	-4.68	10.75	0.37	-0.58	1.32
	POST-72	66	↑	2.75	-3.96	9.46	0.39	-0.56	1.33

QP-ROM: Quadriceps Passive ROM, **HP-ROM:** Hamstrings Passive ROM, **HD-ROM:** Hamstrings Dynamic ROM.

Table displays between group differences regarding percentage likelihood that the FR intervention had an effect on the recovery of the dependent variable, whether FR caused: an increase (↑), a decrease (↓), or an unclear change (UNCLEAR) in the dependent variable, the mean differences (Mean Diff) between CON and FR groups, and standardized effect size (d). 95% confidence limits are displayed for both mean difference and effect size. **Bold numbers** (% likelihood) indicate conditions with substantial change (>75% likelihood).

TABLE 3.8.2: CONTRACTILE PROPERTIES

Measurement	Time	% Likelihood	↓ / ↑	Mean Diff	Lower 95% CL	Upper 95% CL	d	Lower d	Upper d
VJ (cm)	**POST-24**	74	↑	2.38	-2.14	6.9	0.49	-0.4	1.43
	POST-48	**92**	↑	2.8	-0.22	5.82	0.81	-0.06	1.69
	POST-72	UNCLEAR		0.1	-2.82	2.62	-0.04	-1	0.93
MVC force (N)	**POST-24**	UNCLEAR		8.15	-62.66	78.96	0.11	-0.9	1.12
	POST-48	UNCLEAR		9.3	-93.24	74.64	-0.11	-1.07	0.86
	POST-72	UNCLEAR		11.32	-86.05	63.41	-0.15	-1.11	0.82
VA (%)	**POST-24**	**88**	↑	2.88	-0.76	6.52	0.71	-0.2	1.61
	POST-48	**97**	↑	5.34	0.89	9.79	1	0.17	1.83
	POST-72	**79**	↑	2.62	-1.63	6.87	0.57	-0.35	1.49
iEMG (mV/s)	**POST-24**	53	↑	0.02	-0.07	0.12	0.23	-0.72	1.19
	POST-48	53	↓	0.02	-0.12	0.07	-0.23	-1.19	0.72
	POST-72	62	↓	0.05	-0.19	0.09	-0.33	-1.29	0.62
TF (N)	**POST-24**	**88**	↓	15.03	-33.59	3.53	-0.73	-1.6	0.17
	POST-48	**98**	↓	25.6	-46.08	5.13	-1.03	-1.85	-0.21
	POST-72	**86**	↓	11.68	-27.01	3.64	-0.69	-1.59	0.21
EMD (ms)	**POST-24**	**85**	↓	4.4	-1.63	10.43	0.66	-0.25	1.66
	POST-48	**75**	↓	3.05	-2.63	8.73	0.5	-0.43	1.43
	POST-72	58	↑	1.7	-7.23	3.83	-0.29	-1.25	0.66
RFD (N•s-1)	**POST-24**	**> 99**	↓	489.84	-700.7	-279	-1.47	-2.1	-0.84
	POST-48	**> 99**	↓	544.7	-839.6	-249.8	-1.32	-2.03	-0.6
	POST-72	59	↓	125.84	-523.4	271.7	-0.3	-1.26	0.65
½RT (ms)	**POST-24**	61	↑	5.2	-9.92	20.32	-0.33	-1.28	0.6
	POST-48	67	↑	7.75	-10.5	26	-0.4	-1.35	0.54
	POST-72	59	↑	6.05	-12.58	24.68	-0.31	-1.26	0.64
PTF (N)	**POST-24**	UNCLEAR		5.85	-38.14	26.44	0.17	-1.1	0.79
	POST-48	**92**	↓	26.17	-54.82	2.47	0.8	-1.68	0.08
	POST-72	**84**	↓	15.7	-37.6	6.21	0.65	-1.56	0.26

VJ: Vertical Jump, **MVC Force:** Maximal Voluntary Contractile Force, **VA:** Muscle Activation, **iEMG:** Integrated Electromyography, **TF:** Twitch Force, **EMD:** Electromechanical Delay, **RFD:** Rate of Force Development, **½RT:** Half-Relaxation Time, **PTF:** Potentiated Twitch Force. Please reference TABLE 3.8.1 description.

TABLE 3.8.3: FOAM ROLLING PROPERTIES

TABLE 3.8.3A: POST-0 to POST-24

Exercise	Measurement	% Likelihood	↓ / = / ↑	Mean Diff	Lower 95% CL	Upper 95% CL	d	Lower d	Upper d
Anterior	FR-force (kg)	**> 99**	↓	4.29	-6.2	-2.83	-0.56	-0.81	-0.31
	FR-pain	63	↑	0.27	-0.11	0.66	0.26	-0.11	0.63
Lateral	FR-force (kg)	**> 99**	↓	6.39	-8.23	-4.55	-1.04	-1.33	-0.74
	FR-pain	73	=	0.14	-0.19	0.47	0.11	-0.16	0.38
Posterior	FR-force (kg)	78	=	0.82	-1.13	2.77	0.10	-0.14	0.35
	FR-pain	59	↑	0.3	-0.12	0.72	0.24	-0.09	0.57
Medial	FR force (kg)	**96**	↓	2.13	-3.33	-0.93	-0.41	-0.64	-0.18
	FR-pain	**99**	↑	0.84	0.3	1.37	0.70	0.26	1.15
Gluteals	FR force (kg)	**> 99**	↓	3.63	-4.98	-2.28	-0.44	-0.61	-0.28
	FR-pain	**98**	↑	0.9	0.37	1.43	0.56	0.23	0.90

TABLE 3.8.3B: POST-24 to POST-48

Exercise	Measurement	% Likelihood	↓ / = / ↑	Mean Diff	Lower 95% CL	Upper 95% CL	d	Lower d	Upper d
Anterior	FR-force (kg)	67	↓	2.01	-3.65	-0.37	-0.25	-0.45	-0.04
	FR-pain	**> 99**	↓	1.01	0.51	1.51	-0.64	-0.96	-0.32
Lateral	FR-force (kg)	94	=	0.51	-1.68	0.66	-0.07	-0.24	0.09
	FR-pain	**>99**	↓	0.89	0.54	1.23	-0.54	-0.74	-0.33
Posterior	FR-force (kg)	97	=	0.07	-1.5	1.36	-0.01	-0.19	0.17
	FR-pain	60	↓	0.3	0.04	0.56	-0.22	-0.42	-0.03
Medial	FR force (kg)	95	=	0.44	-1.12	0.24	-0.09	-0.22	0.05
	FR-pain	**86**	↓	0.45	0.12	0.78	-0.34	-0.58	-0.09
Gluteals	FR force (kg)	94	=	0.67	-0.57	1.91	0.08	-0.07	0.23
	FR-pain	**97**	↓	0.69	0.36	1.02	-0.38	-0.56	-0.20

Anterior: Quadriceps, **Lateral:** Iliotibial Band, **Posterior:** Hamstrings, **Medial:** Adductors, **Gluteals:** Gluteeals.

Referring to the muscles/area targeted for each foam rolling exercise.

Table displays between time difference for **FR-force** (average force placed on the foam roller) and **FR-pain** (perceived pain while foam rolling) regarding the percentage likelihood that there was a change/no change in the dependent variable, whether FR caused: an increase (↑), no substantial change (=), or a decrease (↓) in the dependent variable, the mean values for each time point, and the standardized effect size (d) with 95% confidence limits

TABLE 3.8.4: FOAM ROLLER FORCE

	POST-0			POST-24			POST-48		
Exercise	**Mean (% BW)**	**Mean (kg)**	**SD (kg)**	**Mean (% BW)**	**Mean (kg)**	**SD (kg)**	**Mean (% BW)**	**Mean (kg)**	**SD (kg)**
Anterior	52	42.34	6.99	47	38.05	7.62	44	36.05	8.19
Lateral	53	43.30	5.70	46	36.91	6.17	45	36.40	7.10
Posterior	52	42.39	7.58	53	43.22	7.97	53	43.15	7.99
Medial	35	28.72	4.78	32	26.59	5.17	32	26.15	5.01
Gluteals	55	45.39	8.20	51	41.76	8.21	52	42.43	8.27

Table displays FR-force for all five FR exercises. FR-force is displayed as the average force placed on the foam roller for each exercise. FR-force is displayed as a percentage of bodyweight (% BW) and kilograms (kg) of force.

3.9 REFERENCES:

1. Abraham WM. Factors in delayed muscle soreness. *Medicine and Science in Sports and Exercise*. 1977;9(1):11-20.

2. Andersen LL, and Aagaard P. Influence of maximal muscle strength and intrinsic muscle contractile properties on contractile rate of force development. *European Journal of Applied Physiology*. 2006;96(1):46-52.

3. Barnes MF. The basic science of myofascial release: morphologic change in connective tissue. *Journal of Bodywork & Movement Therapies*. 1997;1(4):231-8.

4. Behm DG, St-Pierre DM, and Perez D. Muscle inactivation: assessment of interpolated twitch technique. *Journal of Applied Physiology*. 1996;81(5):2267-73.

5. Behm DG, Whittle J, Button D, and Power K. Intermuscle differences in activation. *Muscle & Nerve*. 2002;25(2):236-43.

6. Bobbert MF, Hollander AP, and Huijing PA. Factors in delayed onset muscular soreness of man. *Medicine and Science in Sports and Exercise*. 1986;18(1):75-81.

7. Button DC, and Behm DG. The effect of stimulus anticipation on the interpolated twitch technique. *Journal of Sports Science and Medicine*. 2008;7(4):520-4.

8. Callaghan MJ. The role of massage in the management of the athlete: a review. *British Journal of Sports Medicine*. 1993;27(1):28-33.

9. Cavanagh PR, and Komi PV. Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. *European Journal of Applied Physiology*. 1979;42:159-63.

10. Cheung K, Hume P, and Maxwell L. Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine*. 2003;33(2):145-64.

11. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Hillsdale (NJ): Lawerence Earlbaum Associates; 1988.

12. Connolly DAJ, Sayers SP, and McHugh MP. Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research*. 2003;17(1):197-208.

13. Crane JD, Ogborn DI, Cupido C, Melov S, Hubbard A, Bourgeois JM, and Tarnopolsky MA. Massage therapy attenuates inflammatory signaling after exercise-induced muscle damage. *Science Translational Medicine*. 2012;4(119):119ra13.

14. CSEP. *The Canadian Physical Activity, Fitness and Lifestyle Approach (CPAFLA)*. 3rd ed. Ottawa: Canadian Society for Exercise Physiology; 2003.

15. Curran PF, Fiore RD, and Crisco JJ. A comparison of the pressure exerted on soft tissue by 2 myofascial rollers. *Journal of Sport Rehabilitation*. 2008;17(4):432-42.

16. Devries HA. Prevention of muscular distress after exercise. *Research Quarterly*. 1961;32(2):177-85.

17. Eston RG, Finney S, Baker S, and Baltzopoulos V. Muscle tenderness and peak torque changes after downhill running following a prior bout of isokinetic eccentric exercise. *Journal of Sports Sciences*. 1996;14(4):291-9.

18. Farr T, Nottle C, Nosaka K, and Sacco P. The effects of therapeutic massage on delayed onset muscle soreness and muscle function following downhill walking. *Journal of Science and Medicine in Sport*. 2002;5(4):297-306.

19. Gibala MJ, Macdougall JD, Tarnopolsky MA, Stauber WT, and Elorriaga A. Changes in human skeletal-muscle ultrastructure and force production after acute resistance exercise. *Journal of Applied Physiology*. 1995;78(2):702-8.

20. Gulick DT, and Kimura IF. Delayed onset muscle soreness: what is it and how do we treat it? *Journal of Sport Rehabilitation*. 1996;5:234-43.

21. Hopkins WG, Marshall SW, Batterham AM, and Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise*. 2009;41(1):3-12.

22. Howatson G. The impact of damaging exercise on electromechanical delay in biceps brachii. *Journal of Electromyography and Kinesiology*. 2010;20(3):477-81.

23. Jones DA, Newham DJ, and Clarkson PM. Skeletal-muscle stiffness and pain following eccentric exercise of the elbow flexors. *Pain*. 1987;30(2):233-42.

24. Komi PV, and Buskirk ER. Effect of eccentric and concentric muscle conditioning on tension and electrical-activity of human muscle. *Ergonomics*. 1972;15(4):417.

25. MacDonald G, Penney M, Mullaley M, Cuconato A, Drake C, Behm DG, and Button DC. An acute bout of self myofascial release increases range of motion without a subsequent decrease in muscle activation or force. *Journal of Strength and Conditioning Research*. 2013;27(3):812-21.

26. Mancinelli CA, Davis DS, Aboulhosn L, Brady M, Eisenhofer J, and Foutty S. The effects of massage on delayed onset muscle soreness and physical performance in female collegiate athletes. *Physical Therapy in Sport*. 2006;7(1):5-13.

27. Mcglynn GH, Laughlin NT, and Rowe V. Effect of electro-myographic feedback and static stretching on artificially induced muscle soreness. *American Journal of Physical Medicine & Rehabilitation*. 1979;58(3):139-48.

28. Mills PC, Ng JC, Thornton J, Seawright AA, and Auer DE. Exercise-induced connective-tissue turnover and lipid-peroxidation in horses. *British Veterinary Journal*. 1994;150(1):53-63.

29. Morgan DL, and Proske U. Popping sarcomere hypothesis explains stretch-induced muscle damage. *Clinical and Experimental Pharmacology & Physiology*. 2004;31(8):541-5.

30. Murphy JR, Di Santo MC, Alkanani T, and Behm DG. Aerobic activity before and following short-duration static stretching improves range of motion and performance vs. a traditional warm-up. *Applied Physiology, Nutrition, and Metabolism*. 2010;35(5):679-90.

31. Paolini J. Review of Myofascial Release as an Effective Massage Therapy Technique. *Athletic Therapy Today*. 2009;14(5):30-4.

32. Pearcey GEP, Bradbury-Squires DJ, Kawamoto JE, Drinkwater EJ, Behm DG, and Button DC. Effects of foam rolling on delayed onset muscle soreness and recovery of dynamic performance measures. *Journal of Athletic Training [In Press]*. 2013.

33. Power K, Behm D, Cahill F, Carroll M, and Young W. *An acute bout of static stretching: effects on force and jumping performance*. 2004, 1389-96 p.

34. Saxton JM, Clarkson PM, James R, Miles M, Westerfer M, Clark S, and Donnelly AE. *Neuromuscular dysfunction following eccentric exercise*. 1995, 1185-93 p.

35. Smith LL, Keating MN, Holbert D, Spratt DJ, Mccammon MR, Smith SS, and Israel RG. *The effects of athletic massage on delayed-onset muscle soreness, creatine-kinase, and neutrophil count - a preliminary report*. 1994, 93-9 p.

36. Sydney-Smith M, and Quigley B. *Delayed onset muscle soreness: evidence of connective tissue damage, liquid peroxidation and altered renal function after exercise [dissertation]*. Canberra: Australian Sports Commission; 1992. Available from: Australian Sports Commission.

37. Torres R, Ribeiro F, Duarte JA, and Cabri JMH. *Evidence of the physiotherapeutic interventions used currently after exercise-induced muscle damage: systematic review and meta-analysis* 2012. 101-14 p.

38. Verburg E, Thorud HM, Eriksen M, Vollestad NK, and Sejersted OM. Muscle contractile properties during intermittent nontetanic stimulation in rat skeletal muscle. *American Journal of Physiology*. 2001;281(6):1952-65.

39. Willems M, Hale T, and Wilkinson C. Effects of manual massage on muscle-specific soreness and single leg jump performance after downhill treadmill walking. *Medicina Sportiva*. 2009;13(2):6.

40. Zainuddin Z, Newton M, Sacco P, and Nosaka K. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. *Journal of Athletic Training*. 2005;40(3):174-80.

41. Zhou S, Carey MF, Snow RJ, Lawson DL, and Morrison WE. Effects of muscle fatigue and temperature on electromechanical delay. *Electromyography and Clinical Neurophysiology*. 1998;38(2):67-73.