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ABSTRACT

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PURPOSE: Delayed Onset Muscle Soreness (DOMS) is a common response to activities involving lengthening contractions. Muscle inflammation is associated with DOMS and may play an integral role in protecting a muscle from damage and soreness in response to subsequent bouts of lengthening contractions. Research in animals has shown that prior exposure to passive stretching and isometric contractions of a muscle resulting in muscle inflammation attenuates the muscle inflammatory response following subsequent bouts of lengthening contractions. The purpose of this study was to determine whether passive stretching and isometric contractions in humans would reduce DOMS following a typical bout of resistance exercise. METHODS: Thirty untrained male subjects were assigned to a control (C), stretching (S) or isometric (I) contraction group (n=10/group). In the week prior to the typical resistance training bout subjects in S and I were exposed to 3 separate sessions involving 5 minutes of passive stretching or maximal isometric contractions, respectively. Passive and active soreness, thigh girth, and relaxed knee angle were assessed prior to intervention and on days 1, 2, 4, and 8 following the bout of resistance exercise. RESULTS: Passive and active muscle soreness increased similarly in all groups. However, active soreness returned higher values than passive soreness at several time points following resistance exercise. Peak soreness occurred at 48h post exercise. Thigh girth and relaxed knee angle reached their highest values at 5 minutes following resistance exercise however there were no differences between the groups. CONCLUSIONS: The present study demonstrates that a typical bout of resistance exercise is sufficient to cause measurable levels of DOMS in untrained subjects and that subjects are more sensitive to active measures of DOMS compared to a passive assessment. Nevertheless the passive stretching and isometric contraction interventions did nothing to reduce DOMS in the current subjects.

Keywords: DOMS, isometric contraction, repeated bout effect, active stretching

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Thank you to my wife, Amy. She has been there throughout the whole process, spending long hours taking care of the kids and giving me the freedom to finish my degree.

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Introduction

For individuals beginning a new exercise program, pronounced muscle soreness is a common occurrence in the 24-48 hours following their first exercise bout. This is especially true when the exercise has a significant eccentric component (9-10, 33, 41, 43, 49, 57). This uncomfortable muscle sensation following the exercise session has been termed delayed onset muscle soreness (DOMS) due to the unique time course following the bout of exercise. DOMS is attributed to the muscle damage that accompanies an excessive load to the muscle fibers (12, 57, 61). The soreness associated with muscle damage is usually accompanied by a reduction in joint range of motion (ROM), a reduction in force production, a rise in plasma creatine kinase (CK) levels (10), and an inflammatory response (9, 38, 45, 50).

A single bout of exercise that results in DOMS has been shown to prevent or protect an individual from the same level of soreness and damage in a subsequent bout of exercise (16, 33, 39, 42-43). This phenomenon is known as the repeated bout effect (33). Research suggests that inflammation associated with muscle damage may be a trigger in eliciting the repeated bout effect (46). A decreased inflammatory response may be associated with reduced DOMS (50). Pizza and colleagues (46) examined the impact of prior passive stretching, and isometric contractions on inflammatory responses and muscle damage in mouse skeletal muscle following eccentric contractions. The interventions employed by Pizza et.al. (46) protected the muscles from damage when compared to control animals.

Historically, muscle loading protocols used to induce DOMS use very high exercise loads and volume with a large eccentric component compared to commonly prescribed progressive resistance training programs. Indeed, some studies have used up to 70 maximal eccentric contractions in a single bout of resistance exercise to induce DOMS (9, 39, 53). Therefore, the

muscle damage and DOMS reported by these researchers (9, 18) is likely more severe (21) than reported by the typical first time exerciser. The level of insult to the muscle caused by many eccentric contraction protocols may far exceed the capacity of any pre-exercise intervention to modify subsequent muscle damage and/or soreness. We propose that it may be possible to prescribe a simple intervention that will elicit a protective effect and provide some level of relief from DOMS following a typical resistance training session.

Discomfort associated with exercise training impacts an individual's adherence to an exercise program (15, 17, 35). Any intervention that reduces the intensity of DOMS following an initial bout of exercise should increase adherence to an exercise program in individuals who are in the process of initiating healthy exercise behaviors. Our rationale for this study is that if a simple intervention can be employed to reduce muscle soreness in first time exercise participants this may increase their satisfaction with the activity and improve compliance to prescribed exercise protocols. Compliance may result in positive fitness adaptations and an improved quality of life. The focus of this study was to determine the effect of passive stretching and isometric contraction interventions on DOMS in previously untrained human subjects following a typical bout of resistance exercise.

Methods

Subjects

Thirty untrained men between the ages of 18 and 35 participated in this study. Subjects were randomly assigned into either the control (C) or one of two experimental groups, passive stretching (S) and isometric contractions (I). Subjects were informed of the procedures and any possible risks of the study and signed an approved informed consent document. This study was approved by the Brigham Young University Human Subjects Institutional Review Board.

Study Design

This study used a randomized control design. There were two experimental groups, S underwent a passive stretching protocol of the quadriceps muscles on 3 consecutive days, ending 24 hours before the resistance exercise protocol. One day after the last stretching session the subjects returned to the lab to be tested and underwent the resistance exercise protocol. The second experimental group, I, underwent an isometric contraction protocol of the quadriceps muscles on 3 consecutive days preceding the resistance exercise protocol. Again, 1 day after the last isometric contraction protocol the subjects came back into the lab to undergo the resistance exercise protocol. Measurements were taken before the resistance exercise protocol; then again within 5 minutes post exercise, and then 1d, 2d, 4d and 8d post exercise. The control group (C) did not participate in the passive stretching or isometric contraction treatment protocols, but was tested and underwent the resistance exercise protocol. Measurements were also taken for the control group at the same post time points.

Pre-Exercise Measurements

On the first visit to the lab subjects were measured for height (m), weight (kg), muscle soreness, thigh girth, and relaxed knee angle of the right knee joint. On the day of the resistance exercise session each subject established their concentric one repetition maximum (1RM) for their right leg on a leg extension machine and leg press machine (Cybex).

Muscle soreness was measured subjectively (10) using a scale of 1 to 10. This was assessed with palpation at rest (passive) and during contraction (active) under a standard load representing 25% of their concentric 1 RM. Palpation soreness was determined at 50%, 70% and 90% of the distance between the anterior superior iliac spine (ASIS) and the patella. A mark was made on the subject's thigh, and soreness measurements were taken with the subjects lying flat

on their back with hips and knees extended and relaxed. Palpation soreness was determined using a pressure algometer that ensured that 2 kgs of palpation pressure was placed on the thigh with each test. The subjects determined their level of soreness on a scale of 1 to 10. One indicated no pain and 10 indicated extreme pain. To assess soreness during contraction the subjects performed a unilateral leg extension of the test leg using 25% of their concentric 1RM. Each subject raised and lowered the weight and described their level of soreness, as explained above, on a scale of 1 to 10.

Thigh girth of the right leg was measured by having each subject lie flat on their back with hips and knees extended and relaxed. The researcher then measured the length of the femur from the ASIS to the top of the patella. Circumference measurements were taken with a Gulick tape measure at three separate sites; 50%, 70%, and 90% of the distance between the ASIS and the top of the patella.

The relaxed knee angle of the right leg was determined using a goniometer by having each subject lie on their back with their hips at 90º of flexion. The subject's knee was then allowed to relax, allowing the heel to fall toward the buttocks.

The concentric only 1RM for the leg extension exercise was determined by having the subjects perform a unilateral leg extension exercise. The right leg of each subject was used to determine the 1RM. After a warm-up entailing 5 minutes of cycling on a stationary bike (Lode Groningen) at 95 watts, the subjects were seated in the leg extension machine to perform the test. The initial load was set at 50 kgs. Upon successful completion the next attempt was increased in weight determined based on the difficulty of the previous attempt. If the attempt was extremely easy, the resistance was increase by 25 kgs. When it became more difficult the increase in resistance was by 10 kgs, until to the subjects could not successfully complete the next attempt at

which point the increase in resistance went up by 5 kg increments. Upon the unsuccessful completion of an attempt, the subjects were allowed 5 minutes to rest before attempting to lift the weight again. This was repeated each time the subjects reached a weight they could not lift. The highest resistance successfully lifted was reported as the subjects 1RM. After each attempt the researchers lowered the weight for the subjects ensuring a maximal concentric attempt with no eccentric component.

The concentric only 1RM for the leg press exercise was determined by having the subjects perform a unilateral leg press exercise. The right leg of each subject was used to determine the 1RM. Subjects followed the same protocol for determining their 1RM as described above for the leg extension. Again, after each attempt the researchers lowered the weight for the subjects ensuring a maximal concentric attempt with no eccentric component.

Passive Stretching Protocol

Each subject in S underwent a supervised passive stretching treatment on 3 separate occasions prior to the resistance exercise bout. These stretching bouts occurred on a Monday, Tuesday, and Wednesday. The standing quadriceps stretch was completed by having each subject stand next to a wall or supporting object. Holding themselves stable with one arm they lifted the opposite leg by flexing the knee, bringing the heel towards the buttocks. They then grabbed the top of their foot with the hand on the same side of the body. They pulled their heel as close to their buttocks as they could and held the stretch. Each stretch was held for 5 seconds then relaxed for 1 second. This process was repeated for a duration of 5 minutes to mimic the same protocol used by Pizza el al (46).

Isometric Contraction Protocol

Each subject in group I participated in a supervised isometric contraction treatment. On 3 occasions during the week prior to the resistance exercise protocol each subject performed an isometric contraction treatment of the quadriceps muscles. These isometric contraction bouts occurred on a Monday, Tuesday, and Wednesday. To do this the subject sat on a leg extension machine with their knee at 60° of flexion (54). The weight was set at an amount that the subjects could not move with a maximal single leg effort. For the contraction protocol the subjects maximally contracted their quadriceps for 5 seconds then relaxed for 1 second. They did this for a duration of 5 minutes (46).

Resistance Exercise Protocol

Each subject in S, I, or C underwent the resistance exercise protocol. After the 1RM was determined for the subject's right leg, the subject then performed a leg extension and leg press workout on this leg. The resistance protocol included 4 sets of 12 repetitions of the single leg extension followed by the single leg press. All four sets of the single leg extension exercise were performed followed by a 2 minute rest between sets, and then 4 sets of 12 repetitions, with rests, of the single leg press exercise was performed. The subjects used a resistance that was 60%-65% of the 1RM determined for the right leg. Each subject followed a tempo of 4-2-2 where the subjects lowered the weight for 4 seconds, raised the weight for 2 seconds, and then held the weight at the top of the lift for 2 seconds. A metronome was set at 80 beats per minute, to ensure that the time of the eccentric and concentric stress was comparable between subjects. Between each set the subjects rested for 2 minutes then resumed the next set.

Post Exercise Measurements

At 5 minutes following the exercise protocol the subjects were assessed in the following order: passive and active soreness, thigh girth and relaxed knee angle of the knee joint. The measurements were again taken 1d, 2d, 4d, and 8d after the exercise protocol. Subjects were asked to refrain from any organized exercise activities during the entire study period.

Analysis

A repeated measures analysis of variance (ANOVA) was employed during this study. With four dependent variables including, ROM of the knee joint, thigh girth, passive soreness and active soreness. The independent variables were group and time in every area except when comparing passive and active soreness. With passive and active soreness a third independent variable was added; the type of soreness. For all statistical analysis in the study, a P-value <0.05 was used to establish significant differences. Results are reported as the means ± SE unless otherwise indicated.

Results

Subject Characteristics

Subjects completing the study ranged in age from 18 to 34 years (C=25±1 yr, S=24±1 yr, I=23± yr). The average subject height was C=176±3.5 cm, S=180±2.0 cm and I=182±1.6 cm. Subjects body mass averaged C=82.6±3.8 kg, S=77.5±5.1 kg, and I=81.0±8.3 kg.

Muscle Soreness

There was no difference in passive soreness among groups at the pre measurement (C=1.1$\pm$0.1, S=1.6$\pm$0.5, and I=1.0$\pm$0.0). Passive muscle soreness changed over time ($P<0.05$) following the resistance exercise bout however there was no interaction among groups (Figure 1). In all three groups the highest passive soreness value recorded occurred at the 48 hour time-

point (C=3.0±0.4, S=3.8±0.8, and I=2.5±0.4) and appeared to be resolved by the 192 hour post measure.

Results for active soreness also demonstrated a change over time ($P<0.05$) in the absence of group interaction. Peak levels of soreness with the active measure were reported at the 48 hour time-point (C=4.0±0.5, S=6±0.9, and I=5±0.6) and recovered back to baseline by 192 hours post exercise.

Passive and active soreness measures from each group were combined into a passive or active soreness aggregate value and were compared to examine the level of soreness experienced with palpation pressure versus active sub maximal muscle contraction. Soreness values for the active measure were significantly greater that those for passive measures at 5 min, 24 and 48h post exercise (3.2± 0.08 vs. 2.1±0.10 , 4.6±0.20vs. 2.4±0.06 , 5.0±0.24 vs. 3.0±0.22, and 2.1±0.17 vs. 1.5±0.09, active vs. passive, respectively) ($P<0.05$) (Figure 2). Values were similar and back to baseline by 192 hour post exercise.

Thigh Girth

Although thigh girth values increased over time ($P<0.05$) following the exercise bout there were no differences in thigh girth among the three groups (Figure 3). The greatest values for each of the groups occurred at the 5 min post exercise time point (C=53.4±1.2 cm to 54.4±1.3 cm, S=50.8±1.9 to 52.2±2.1 cm, and I=50.3±2.8 to 51.2±2.8 pre vs. 5 min post)

Relaxed Knee Angle

Relaxed knee angle did not differ among the various treatment groups however it did appear to vary over time ($P<0.05$) (Figure 4). Relaxed knee angle increased to its greatest value at the 5 min post exercise time point (C=48±2.4˚ to 50±1.7, S=43±1.8˚ to 49±2.0˚, and I=45±2.9 to 48±3.2˚, pre vs. 5 min post) and was returned to pre values by the end of the study.

Discussion

Delayed onset muscle soreness (DOMS) is a common response in the 24-48 hours following novel exercise especially when the exercise activity has an eccentric contraction component (12, 50, 57, 61). The ability to prevent or offset this type of muscle soreness might increase satisfaction from and adherence to exercise programs in the first-time exerciser (15, 17, 35). In the present study we employed isometric muscle contractions and passive stretching interventions that have been shown to reduce inflammatory responses in rodent hind limb tissue (46). Inasmuch as muscle inflammation has been associated with muscle damage and soreness we hypothesized that the protocol that reduced inflammatory responses in rodent tissue may, in turn, reduce muscle soreness when employed in humans. In the present subjects a typical bout of resistance exercise resulted in significant soreness (passive and active), increased thigh girth and reduced knee range of motion. However neither isometric contractions nor passive stretches prior to exercise influenced any of the dependent variables when compared to control.

The primary hypothesis of this study was that exposing the muscle to either isometric contractions or passive stretching would reduce DOMS in untrained individuals. This hypothesis was based on the concept of the repeated bout effect (5, 23, 31, 33, 39-40, 43). It has been demonstrated that exercise resulting in muscle damage, soreness and/or muscle inflammation is associated with reduced levels of muscle damage, soreness and/or inflammation in a subsequent bout of exercise (33, 39, 43). We modeled our treatment intervention after that used by Pizza et al (46) with the intent that producing minor levels of inflammation may elicit a repeated bout effect in our subjects. Indeed, in the study by Pizza et al they found that by using isometric contractions and passive stretching they were able to reduce the level of inflammation in mouse skeletal muscle after an initial bout of exercise that elicited a small inflammatory response but no

overt signs of muscle damage. Smith et al (50) found a correlation between the inflammatory response and muscle soreness. As inflammation was decreased so was soreness.

From these findings we assumed that by using these interventions we would decrease inflammation and subsequently decrease soreness. Contrary to our hypothesis we saw no reduction in DOMS after applying either of our treatment protocols. From the current data we can conclude that the passive stretching and isometric contraction protocols do not protect an individual from soreness following a typical bout of resistance exercise. Nevertheless, having not measured inflammation or other cellular markers of muscle damage we are unable to speculate as to the reason for the lack of protection from soreness.

A review of the literature related to muscle damage and soreness reveals myriad protocols for measuring delayed onset muscle soreness (1, 3-4, 9, 18, 20, 40, 49, 55, 61). Two hallmarks of DOMS are that soreness is only experienced during palpation and contraction of the muscle (31-33, 39-40, 47). Therefore we decided to use measures of passive palpation and active contraction in evaluating soreness level. We not only observed both measures of soreness to increase following the contraction intervention but that active soreness measure returned higher values than passive palpation assessment (Fig. 3). One interesting observation of this study was that of the difference between passive and active soreness. Though seemingly intuitive that active soreness measurement may produce higher pain scores than passive palpation assessment we would submit that active soreness evaluation may be a more representative measurement for assessing DOMS in human subjects. Passive soreness evaluation using a pressure algometer as in this study is assessing pressure sensations in a very limited area of the muscle whereas the active soreness measurement likely includes a much greater number of involved muscle fibers. In addition the greatest level of pain occurs during contraction, which

would be of issue in subsequent exercise bouts performed by an individual. We would propose that at the very least both active and passive measures of soreness be included in DOMS research.

During review of research previously done on DOMS and the protective effect, exercise protocols were extreme in nature when compared to what might be considered normal or typical weight lifting regimens (9-10). In some instances subjects performed as many as 70 maximal eccentric contractions (10). The subjects' responses to some of the extreme eccentric contraction research interventions resembled severe DOMS and even symptoms associated with a clinical diagnosis of rhabdomyolysis (21). We chose to demonstrate that a typical bout of exercise could be used to elicit DOMS in untrained individuals (Fig. 1,2,3). We defined typical to be a bout of resistance exercise that a novice exerciser would be likely to perform when attending a weight lifting session for the first time. In the current study the bout of resistance exercise used to elicit DOMS more closely resembled a normal exercise program. Of the many studies that have attempted to access the effectiveness of a pre-exercise intervention in reducing or preventing DOMS (1, 7, 13-14, 18, 22-23, 27, 40) it would seem logical to utilize a training protocol similar to that used by a typical exercise. In this way the results would be more generalizable to the population at large. In the current study we demonstrate that a typical bout of resistance exercise causes measurable and significant soreness in previously untrained subjects.

We report that a common resistance exercise protocol employed in untrained subjects may be used to produce and assess muscle soreness. Furthermore, active contraction measures of muscle soreness may be more robust than passive muscle palpation. However, pre-exercise isometric contraction and passive stretching protocols previously shown to be effective at

reducing inflammation in an animal model do not result in protection from muscle soreness when employed in untrained human subjects.

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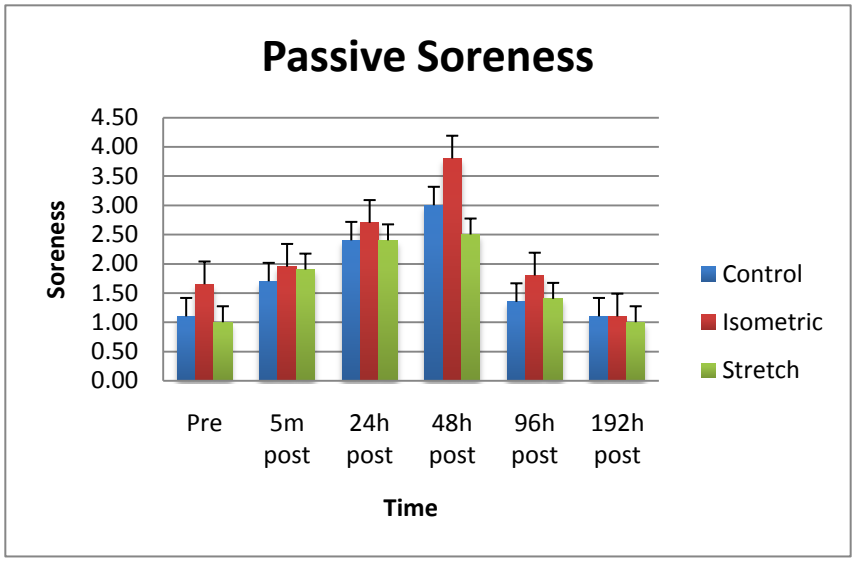

Figure 1. A comparison of passive soreness between groups. There was no significant difference between groups. Significance was seen when comparing the level of passive soreness over time. Soreness increased in all groups from pre-measurements to post measurements.

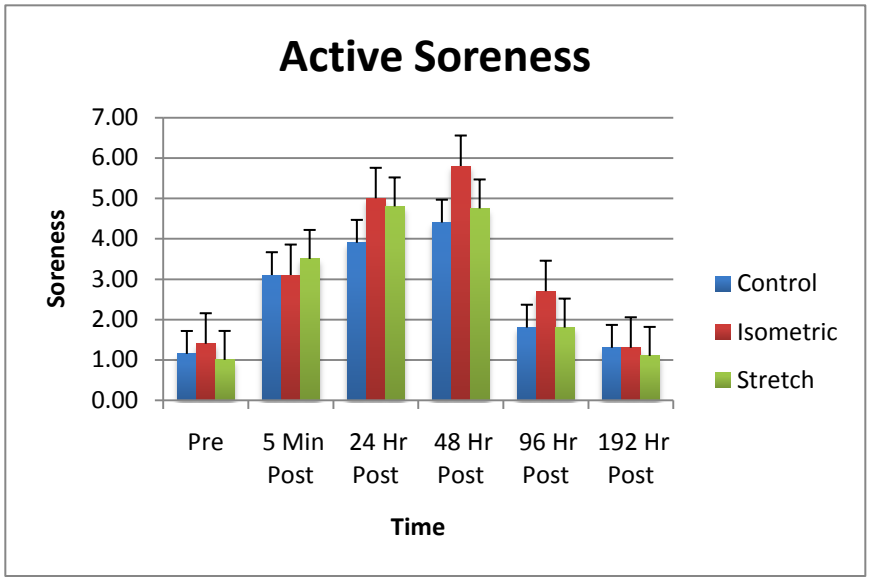

Figure 2. A comparison of Active Soreness between groups. There was no significant difference between groups. Significance was seen when comparing the level of active soreness over time. Soreness increased in all groups from pre-measurements to post measurements

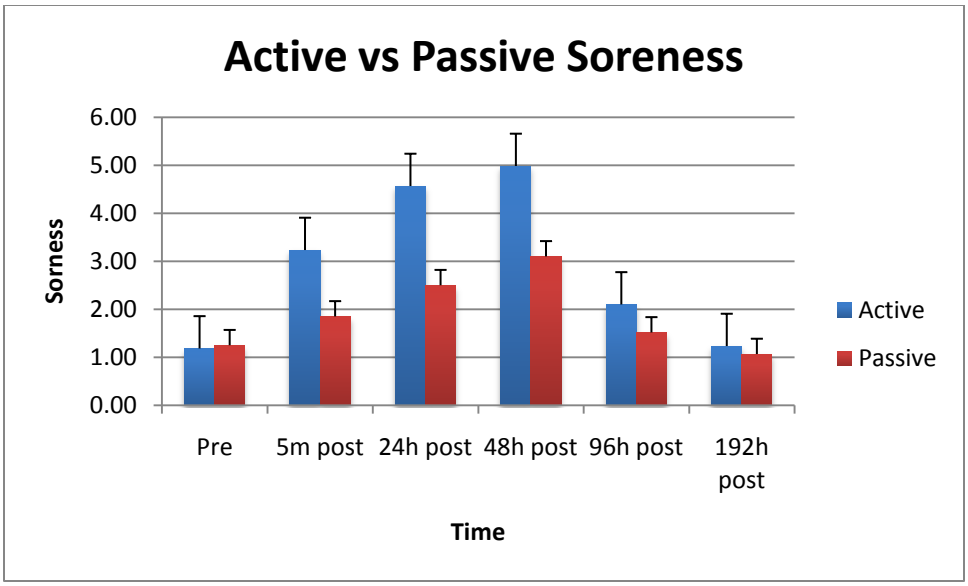

Figure 3. A comparison of active and passive soreness. Significant difference was seen at 5 m post exercise, 24h post exercise, 48h post exercise and 96h post exercise.

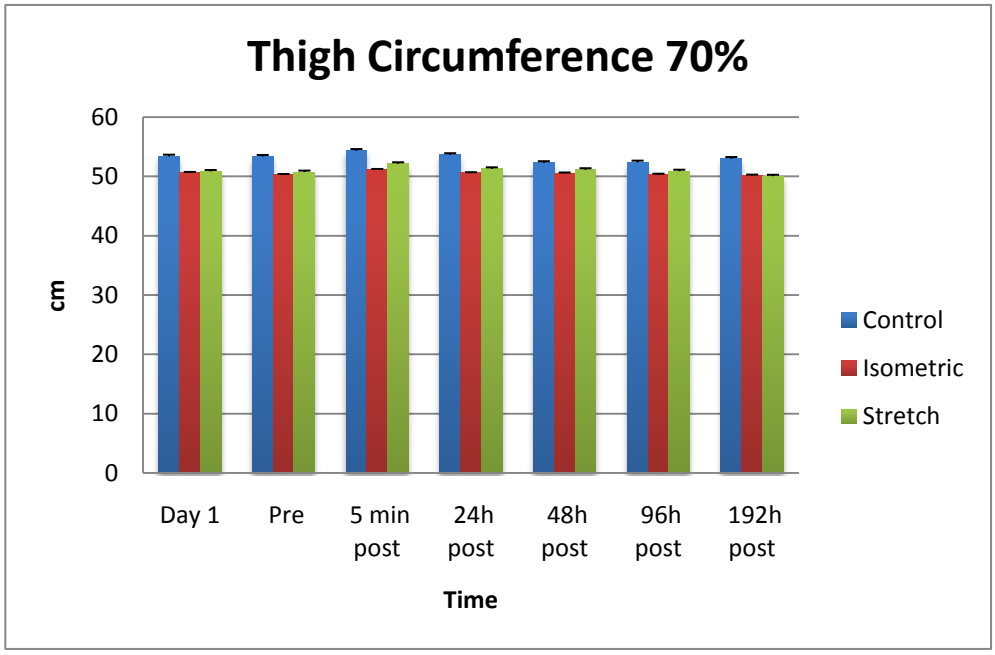

Figure 4. A comparison of thigh circumference at 70% of the distance from the ASIS to the top of the patella. No significant difference was seen between groups or over time.

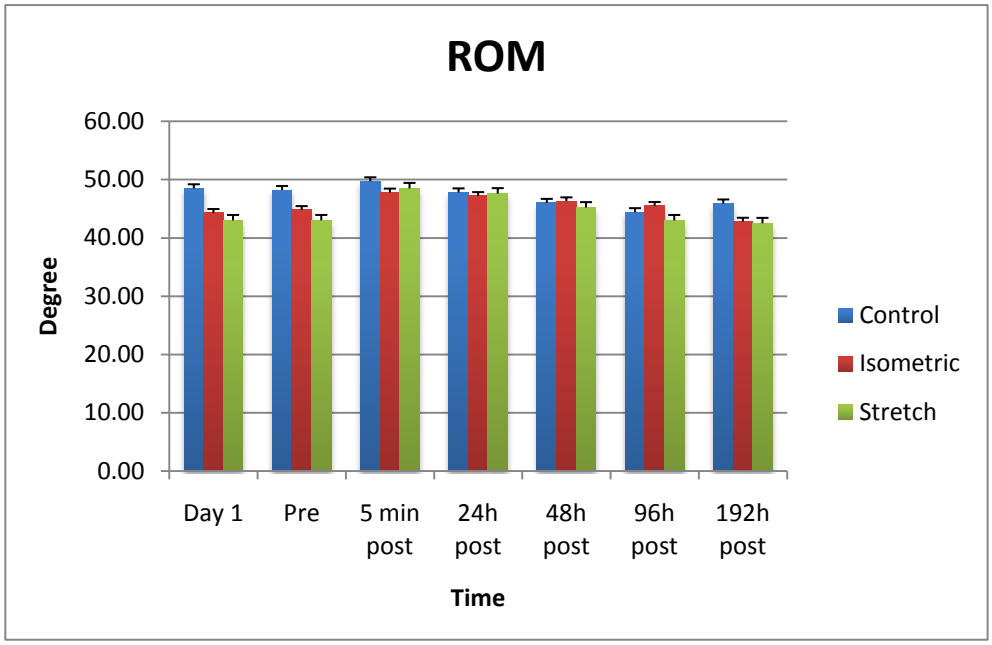

Figure 5. A comparison of Range of Motion (ROM) between groups. No significant difference was seen between groups or over time.

Prospectus

Chapter 1

Introduction

For individuals beginning a brand new exercise program, pronounced muscle soreness is a common occurrence in the 24-48 hours following their first exercise bout. This is especially true when the exercise has a significant eccentric contraction component such as weightlifting or running (9-10, 33, 41, 43, 49, 57). This uncomfortable muscle sensation following the first exercise session has been termed delayed onset muscle soreness (DOMS) due to the unique time course following the bout of exercise. DOMS is attributed to the muscle damage that accompanies eccentric contractions (12, 57, 61). The soreness associated with muscle damage is usually accompanied by changes in joint range of motion (ROM), force production, plasma creatine kinase (CK) levels (10) inflammation (9, 38, 45, 50) and Z-line streaming (19). It has been demonstrated that the discomfort involved in exercise training may impact an individual's adherence to an exercise program (15, 17, 35). An intervention that resulted in the reduction of DOMS following an initial bout of exercise would be advantageous to increasing adherence to an exercise program in individuals who are in the process of initiating healthy exercise behaviors.

A single bout of exercise that results in muscle soreness and damage has been shown to prevent or protect an individual from the same level of soreness and damage in a subsequent bout of exercise (16, 33, 39, 42-43). This phenomenon is known as the repeated bout effect (33). There is research that suggests that the inflammation associated with muscle damage may be a factor in eliciting the repeated bout effect (46). Specifically, Pizza and colleagues (46) examined the impact of prior passive stretching, and isometric contractions on inflammatory responses in mouse skeletal muscle following eccentric contractions. They reported that passive stretching or isometric contractions caused small increases in markers of inflammation in the effected skeletal muscle in the absence of marked muscle damage. Interestingly, when the

treated muscles were later exposed to lengthening contractions the measured inflammatory response in the treated muscles was lower compared to muscles that had not been treated prior to lengthening contractions. This demonstrated a protection from muscle inflammation without the prior presence of marked muscle damage. A decreased inflammatory response may be associated with decreased DOMS (50). The fact that the interventions by Pizza (46) produced components of the protective effect without overt signs of muscle damage beg the question, can a protective effect be initiated without damage to the muscle?

Generally, muscle damage protocols used to study muscle soreness are excessive in their exercise intensity and volume when compared to commonly prescribed resistance training programs. Indeed, some researchers have had their untrained subject do as many as 70 maximal eccentric contractions in a single bout of resistance exercise (9, 39, 53). Therefore, the damage and soreness responses seen by some researchers (9, 18) is likely more severe (21) compared to the level of DOMS encountered by the typical first time exerciser. The level of insult to the muscle caused by many eccentric contraction protocols may far exceed the capacity of pre-exercise interventions or the repeated bout effect to reduce muscle damage and soreness. We hypothesize that a simple intervention known to elicit protective effect responses may provide some level of protection against muscle damage and DOMS following a typical resistance training session.

Our rationale for this study is that if a simple intervention can be employed to reduce muscle soreness in first time exercise participants this may increase their satisfaction with the activity and improve compliance to prescribed exercise protocols. This compliance may result in positive fitness adaptations and an improved quality of life. The focus of this study will be to

determine the effect of passive stretching and isometric contraction interventions on DOMS in previously untrained subjects following a typical bout of resistance exercise.

Hypotheses

The following hypotheses are proposed as a result of 3 sessions of a stretching, or isometric contraction protocol prior to a typical weight training protocol:

1. DOMS will be reduced in the treated groups compared to controls, after an initial bout of exercise
2. Quadriceps ROM will remain closer to pre-exercise values
3. Increases in thigh girth will be reduced

Null Hypothesis

No affect will be seen in response to 3 sessions of stretching or isometric contraction protocols prior to a typical weight training protocol.

Definition of Terms

Acetylcholine (ACh) – A neurotransmitter released at the motor end plate of a motor neuron. It opens sodium channels on the muscle to induce an action potential, which leads to a muscle contraction.

Delayed Onset Muscle Soreness (DOMS) – Muscle soreness that appears 24 – 48 hours after an eccentric bout of exercise.

Heat Shock Proteins (HSP) – A group of small proteins which respond to stressors, on being heat. They are shown to aid in protection and recovery of muscle tissue.

One Repetition Maximum (1RM) – The maximum amount of force that an individual can produce within one repetition.

Range of Motion (ROM) – The range through which a joint can be extended and flexed.

Assumptions

1. Subjects will be untrained individuals who have not participated in an exercise regimen of the quadriceps muscle within the past 6 months
2. Subjects will complete all aspects of the treatment and exercise protocols
3. Subjects will not participate in any other exercise during the period of treatment and data collection.
4. Subjects will give their best effort during the experimental period.

Limitations

1. 1RM will be determined by using the left leg, while the exercise protocol will be performed by the left leg.
2. Only the right leg will be measured for markers of damage.
3. Changes in 1RM will not be determined.

Delimitations

The study will be composed of untrained individuals between the ages of 18 and 30. The groups will be randomly assigned.

Purpose of The Study

Our rationale for this study is that if a simple intervention can be employed to reduce muscle soreness in first time exercise participants this may increase their satisfaction with the activity and improve compliance to prescribed exercise protocols. This compliance may result in positive fitness adaptations and an improved quality of life. The focus of this study will be to determine the effect of passive stretching and isometric contraction interventions on DOMS in previously untrained subjects following a typical bout of resistance exercise.

Chapter 2

Review of Literature

Muscle Structure

The sarcomere is the smallest functional unit of the skeletal muscle. The sarcomere is defined on either side by the Z-line. From either Z-line of a sarcomere extend fibrous strands of actin myofilaments. These actin filaments interact with myosin myofilaments which are anchored to the center of the sarcomere at the M-line. Actin and myosin interaction produce a muscle contraction. Actin and myosin's interaction is regulated by two smaller myofilaments, troponin and tropomyosin.

Tropomyosin extends along the actin myofilament, covering each of its active sites. When covered, actin is unable to interact with myosin. Troponin is bound to tropomyosin. Troponin regulates when tropomyosin covers the actin active sites. Troponin acts in response to calcium (Ca++). When Ca++ is present troponin undergoes a conformational change which moves tropomyosin away from the actin active sites allowing myosin to bind. When Ca++ leaves troponin conforms so that tropomyosin covers the active sites, hindering myosin's binding.

Sarcomeres are arranged in a longitudinal series which make up a myofibril. Myofibrils are connected to adjacent myofibrils at their respective Z-lines, producing a regular striated pattern (8). With the shortening of a series of sarcomeres the myofibril will shorten. A large group of myofibrils encased within the sarcoplasm makes up a muscle cell. The muscle cell itself is encased within connective tissue, called the endomysium.

Muscle cells are held in bundles called fascicles by another connective tissue layer called perimysium. A bundle of fascicles, which make up the whole muscle, are then encased in the epimysium. The endomysium, perimysium, and epimysium are all connected producing an

extensive encasement of the muscle. This connective tissue then inserts into the tendons and later the bones.

The muscle itself is formed and stabilized by many proteins that form the cytoskeleton. The cytoskeleton has important roles in force transmission and tension development during muscle contraction (48). An example is desmin in knockout mice that lack the cytoskeletal protein desmin (48) they were unable to produce the same level of isometric force when compared to wild type mice. When tension is developed within the muscle to produce a muscle contraction, that tension is transmitted from the actin and myosin myofilaments through the muscle cytoskeleton and on to the outer surface of the muscle where eventually it reaches the bones and causes movement.

Muscle Function

In the arms and legs the purpose of muscle is to move the body about a joint, either by decreasing or increasing the joint angle. When a muscle contracts it undertakes an extremely complex process in a fraction of a second. A nervous impulse is sent to the nerve terminal releasing acetylcholine (ACh) into the synaptic cleft. The ACh then binds to receptors on the muscle membrane. Upon activation the channels open allowing sodium (Na+) to rush into the cell. This activates voltage gated Na+ channels, allowing the action potential to continue down the membrane. As the action potential travels it comes to the T-tubules. The tubules take the action potential deep into the center of the cell where it can activate the release of Ca++.

When Ca++ enters the cell it interacts with troponin, which eventually frees the actin active sites. Myosin binds to the active site where it will stay until ATP binds. The binding of ATP causes myosin to undergo a power stroke. This action pulls the actin myofilament along the myosin, drawing the Z-lines closer together, shortening the sarcomere. With the shortening of millions of sarcomeres the entire muscle is allowed to contract.

When the sarcomeres shorten, they need a way to transfer that force produced by the actin and myosin out to the periphery of the muscle then to the bones, allowing the joint to move. This is done by a series of cytoskeletal proteins. Myosin is stabilized at the M-line to keep it from moving. Actin interacts with α-actinin to bind it to the Z-disk. α-actinin is the primary protein of the Z-disk. There it interacts with other intermediate filaments. One in particular is desmin. Desmin links the Z-disk to other Z-disks in adjacent myofibrils. At the periphery of the muscle cell desmin connects the Z-disk to the sarcolemma. From there it connects to the connective tissue which then transfers the force to the tendons and bones.

A second way observed to transfer force to the tendon is through adjacent muscle fibers. Street (51) demonstrated that muscle force can be transmitted laterally between muscle fibers. She dissected a single muscle fiber from one end of the muscle and left the surrounding fibers intact on the other end. While securing the bare end of the single muscle fiber, she measured the force generated by the single fiber. She then released the bare end of the single fiber and secured the remaining muscle fibers adjacent to the single fiber at the opposite end. Again, she activated the bare single fiber and measured an isometric force nearly equivalent to that measured when the bare end of the single fiber was secured. This experiment showed that the physical interaction between adjacent fibers was sufficient so that the activated single fiber transmitted its force radially to adjacent fibers that were secured, and that force was transmitted to the force-measuring end via these adjacent fibers. It is important to note that this result was obtained in amphibian muscle, which has a relatively poorly developed endomysial connective network. The situation for mammalian muscle should be more impressive where the endomysial connective tissue matrix is much more highly developed (44).

Muscle Contraction Types

There are three types of muscle contraction. These are concentric, isometric and eccentric. Concentric contractions induce muscle shortening. This will cause the joint angle to become smaller. This type of contraction is seen when lifting an object, as in the elbow flexors when doing an arm curl. Isometric contraction happens when the muscle contracts but elicits no movement in the joint or lengthening or shortening of the muscle. This is done to stabilize a joint against movement, as when the shoulder musculature stabilizes the shoulder girdle while the elbow flexors lift an object. Eccentric contraction occurs when the muscle lengthens as it contracts. This is seen when lowering a weight, and in decelerating the body when running and jumping.

Eccentric Damage and DOMS

Eccentric contractions induce the greatest amount of damage to a muscle cell, when compared to the other forms of contraction (57, 60). Sarcomeres within a muscle cell have many differences (34, 36). They differ in their lengths, and tensile strength (47). Muscle contraction occurs with the interaction of actin and myosin myofilament, where they slide over each other to shorten the muscle cell. Without the interaction of actin and myosin the sarcomere cannot shorten. When the muscle undergoes a lengthening contraction, some sarcomeres may be overstretched to the point where the actin and myosin within that sarcomere no longer overlap. This would require the surrounding structures and connective tissue to take the stress of the contraction (9). This is one hypothesis that might explain the damage caused to the cytoskeleton.

Indicators of Muscle Damage

Indicators of muscle damage include; decreased flexion and relaxation angles or range of motion, decreased force generation in both isometric and dynamic actions, soreness, increased

blood serum CK levels (11) and Z-line streaming (19). The decreased range of motion is thought to be due to an increase in passive tension (57). The cause for this increase in passive tension is not completely understood. One hypothesis is that there is a rise in intracellular calcium concentration. This may be due to damage to the sarcoplasmic reticulum of the muscle cell (58). The damaged reticulum would allow calcium to enter the cell and cause interactions between the actin and myosin myofilaments, thus producing an increased level of tension within the relaxed muscle.

Some hypotheses about the decrease in force generation include damage to the sarcomeres (38), or overextension of these sarcomeres (9). When the muscles undergo a lengthening contraction, the actin and myosin may reach a point where they no longer overlap in selected sarcomeres. A hypothesis is that after some of the sarcomeres are overstretched they do not recover to their original overlapping structure after relaxation. Therefore, when the muscle contracts at a later time, the muscle cannot produce the same amount of force as the muscle was previously able to, because not all sarcomeres of each fiber are involved in the contraction. Others hypothesized that the strength loss may be due to damage to the excitation-contraction coupling process (56).

The pain following a damaging bout of exercise has been postulated to be due to the inflammatory process (9). After a damaging bout of exercise there is an increase in circulating neutrophils (9). Smith (50) has postulated that these neutrophils migrate to the site of injury, followed by monocytes. The monocytes peak at about the same time as muscle soreness, 48 hours post exercise. The monocytes synthesize a large amount of prostaglandins, which in turn sensitizes the afferent nerve endings causing the sensation of soreness (9). Another hypothesis is

that the soreness is due to inflammation of the surrounding connective tissue which may put pressure on the sensory nerve endings causing it to become sore (9).

As for increases in serum CK levels, damage to the muscle cell membrane is thought to allow the release of the CK into the blood stream (47). Z-line streaming can be seen when looking at a cross-sectional view of a damaged muscle cell. This may be due to damage or loss of some very important structural proteins, namely desmin and α-actinin (2, 19). As explained earlier the muscle cell is set up in a very organized, striated pattern. Desmin acts at the Z-line to hold the muscle cell in a very organized pattern. After a damaging bout of exercise desmin almost seems to disappear, only to reappear a few days later (2). With the loss of desmin there is a loss of an important structural protein to hold the cell in order, thus Z-line streaming appears, which is the term used for the disorganized sarcomere pattern.

Repeated Bout Effect

After a damaging bout of eccentric exercise, a second bout does not elicit damage to the same extent as the first. This is known as the repeated bout effect (16, 39, 47). The mechanism of the repeated bout effect is unknown. Some theories include neural adaptations, cellular adaptations, and cytoskeletal adaptations (31). In the McHugh (31) review he explained the neural adaptation as a possible increase in motor unit recruitment and/or motor unit synchronization. But this area of adaptation seems to play a very small part in the repeated bout effect (31).

Another area of interest is the cellular adaptations. McHugh (31) describes three main areas of interest regarding the cellular adaptations: the strain on the sarcomere, adaptations to the excitation-contracting coupling process, and the inflammatory response. Under the idea of the sarcomere strain theory, there is more damage seen to the cell when eccentric contractions take the cell to longer lengths. After a damaging bout, the muscle cell seems to add sarcomeres in

series, shifting the length tension curve to the right and allowing the muscle to move at longer lengths. With the addition of sarcomeres, at any given length there would be less tension within the muscle. This idea looks promising, but there is lack of evidence for this hypothesis (59). While there is a shift in the length tension curve following eccentric exercise, this shift has been shown to return to normal after only a few days (59). Also sub maximal eccentric training has been shown to result in longitudinal addition of sarcomeres in rats (29), sub maximal eccentric training did not, however, elicit a protective effect from subsequent maximal contractions in humans (41).

Strength loss associated with eccentric exercise damage may be attributed to disruption in the excitation-contraction coupling process. Strength loss is thought to be due to a combination of physical disruption and an impairment of the excitation-contraction coupling (56). Excitation-contraction coupling is the sequence of events starting from the release of acetylcholine from the nerve terminal into the synaptic cleft, to the subsequent release of calcium from the sarcoplasmic reticulum. Impaired excitation-contraction coupling has been estimated to account for 50%-75% of strength loss in the first 5 days after a damaging bout of eccentric exercise (56). This area is not very well understood. One suggestion is that strengthening of the sarcoplasmic reticulum would prevent impairment of the excitation-contraction coupling (31), but there is no evidence to support this claim. There is evidence, however, against the theory of damage along the excitation-contraction coupling process (10, 16). With an initial bout of eccentric exercise we see a decrease in strength; there is a similar decrease after additional bouts of exercise (10, 16). If there were an adaptation to this process, strength loss would not be reduced to the same extent on the second bout (31).

With the inflammatory response, we see a decrease in its response after a second bout. With the initial injury of eccentric damage a local inflammatory response is triggered, which leads to an aggravation of the damage prior to signs of recovery (45). Following a second bout the inflammatory response has been shown to be blunted (45-46). It is difficult to determine the cause of the decrease in the inflammatory response. It could be a reflection of less myofibrillar disruption during the actual repeated bout, a decrease in the secondary proliferation of damage or a combination of both (31).

In regards to the mechanical adaptations McHugh (31) looked at cytoskeletal adaptations and adaptations to the intramuscular connective tissue. Desmin has been shown to increase in response to damaging contractions (2). Others (25) have seen increases in intramuscular connective tissue following damaging contractions. They believed that this increase in connective tissue helps support the muscle cell. The belief is that an increase in cytoskeletal content and connective tissue will protect the muscle from recurring damage. Adaptations in these two areas would also increase the passive stiffness in these tissues (31). There is evidence that increased passive muscle stiffness increases the potential for damage (32). Subjects with "stiff" hamstrings showed a higher response in the indicators of muscle damage compared to subjects with more compliant hamstrings (33).

Prevention of DOMS

DOMS is an indicator of muscle damage. It is experienced by novice as well as trained individuals when doing an unaccustomed exercise. But as explained earlier, by the protective effect, the amount of DOMS felt by trained individuals is less than that of an untrained individual. Researchers have worked to try to prevent DOMS and to further understand how it occurs.

Role of Nutrition

Nelson et al (37) looked at nutrition's role in preventing DOMS. Their hypothesis stemmed from the understanding that fast-twitch muscle fibers experience a greater degree of muscle damage when compared to slow-twitch fibers. They looked at a proposed idea from Lieber and Friden(26), "fast-twitch fibers, which lack the higher oxidative capacities of slow-twitch fibers, fatigue early in an exercise period and become unable to generate enough ATP to support the contractile mechanism. According to the current theories of muscle contraction, the release of the myosin head from the actin filament requires the addition of a new ATP. Insufficient ATP causes this release mechanism to fail, and the fibers enter a ridged or highly stiff state" (37). In this rigid state the muscle fibers would encounter disruption to the sarcolemma, sarcoplasmic reticulum, and myofilaments. This would result in the observed damage to the cytoskeleton and myofibrils, causing an inflammatory response, followed by DOMS (37). Nelson et al (37) wanted to compare individuals who were glycogen depleted to those with glycogen in the muscles. Two groups underwent a glycogen depletion protocol. One group fasted for 12 hours after glycogen depletion then underwent an eccentric exercise protocol. The next group, after the glycogen depletion immediately consumed a meal designed to replenish glycogen stores. After a 12-hour fast they also underwent the eccentric exercise protocol. The results of this study showed no significant difference between groups for all areas that were measured. These included: maximal isometric strength, thigh circumference, relaxed knee angle, and DOMS.

Role of Warm-Up

Evans et al (18) looked at the effects of warm-up on DOMS. To do this they passively and actively warmed-up the muscle to be worked. They had three different treatment protocols with different groups. The first used low heat passive warm-up, the next group used high-heat

passive warm-up and the last group used active warm-up. Each group then immediately followed the treatment with a bout of eccentric exercise. To passively warm-up the muscle they heated the muscle using a Magnatherm Diathermy unit. They assumed the muscle temperature would increase 1ºC in the low heat group and 3.5ºC in the high heat group after 10 minutes of exposure with different levels of intensity from the diathermy unit. This assumption was based on earlier pilot work they performed. For active warm-up they actively flexed and extended the muscle of interest. This was done using a Boidex machine to actively take the arm through the range of motion. They determined this would increase muscle temp by 1ºC. Within 10-15 seconds after the warm-up activity the subjects underwent the eccentric exercise protocol. They found in this study that warm-up immediately before a damaging bout of eccentric exercise had no protective effect against DOMS and other indicators of muscle damage (18).

Nosaka et al (40) used microwave diathermy to warm the muscle 1 day prior to an eccentric bout of exercise. The basis for his research was a group of proteins called heat shock proteins (HSP). The idea was that with heating the muscle, they would be able to induce and increase the amount of HSP within the muscle, which may play a protective role in the muscle cell. Heat Shock Proteins have been shown to translocate to the cytoskeleton in response to eccentric damage of the muscle (24). Nosaka et al (40) hypothesized that by heating the muscle 1 day prior to eccentric exercise, there would be an increase in HSP, which would attenuate muscle damage. Doing this they were able to decrease many of the markers of muscle damage, including maximal isometric strength, ROM, and soreness. All of these indicators, except soreness, were protected to the same extent as prior eccentric training. The effect they had on soreness was small and was only seen in extension of the muscle; both flexion and palpation were not different than the control group (40).

Role of Contraction

Concentric Contraction

Performing a single bout of eccentric exercise has been shown to protect from damage against a subsequent bout (16, 39, 47). But what effect do concentric contractions have on the protective effect? Whitehead et al (57) tried to answer that question. The subjects performed concentric only heel raises with one leg for a period of 5 days. A few days later both legs were exposed to an eccentric exercise protocol. The researchers then compared the level of soreness and damage between each leg. DOMS was not affected by the concentric training protocol. Their results showed no protection from eccentric exercise.

Isometric Contraction

Isometric contractions have been shown to affect the inflammatory response. Pizza et al (46), though he did not look directly at DOMS, looked at the response of the inflammatory response to eccentric and isometric contractions, and passive stretching. They found a decreased inflammatory response to all three treatments in a subsequent eccentric bout. There was a difference in the response of each, with prior eccentric contractions eliciting the greatest response. Though eccentric contractions had the greatest response, isometric contractions and passive stretching had a response very close to that of eccentric damage. The onset of DOMS and the rise in the inflammatory response have a very strong correlation (50), where both DOMS and the inflammatory response peak at about 2-3 days and dissipate in a similar pattern (50). Although Pizza et al (46) did not directly measure DOMS, there is a strong possibility that with a decreased inflammatory response there may be a decrease in the level of DOMS experienced (50).

Eccentric Contraction

These are some examples of trials that have not worked to prevent DOMS. Researchers have successfully prevented DOMS. This has occurred in areas that may be categorized under the repeated bout effect. Prior eccentric exercise reduces the amount of DOMS experienced (42-43). This reduction in DOMS was experienced with as little as 2 eccentric contractions 2 weeks prior to a damaging bout of eccentric exercise (43). With prior eccentric work, reduction in DOMS is experienced in extension, flexion, as well as palpation (42-43). The effects of the protection against DOMS from eccentric work can last up to 6 weeks and possibly more for some individuals (39).

Role of Vibration

Bakhtiary et al (1) exposed subjects to vibrations to see its effect on DOMS. Their goal was to determine if vibration training before eccentric exercise may prevent DOMS by improving muscular strength and power development strategy, improving kinesthetic awareness, and providing insight into the effects of fatigue. Their protocol included using a 50 Hz vibrator apparatus to apply vibration to the middle line of the quadriceps, hamstrings, and calves on both the left and right sides. This was done for one minute immediately before the eccentric exercise protocol. To damage the muscle the subjects walked downhill on a 10º decline treadmill at 4 km per hour for 30 minutes. In this study they saw positive results in all areas measured. Soreness levels were greatly reduced in the treatment group. The other areas they measured included: plasma CK and maximum isometric voluntary contraction force. All measurements were significantly different in the treatment group compared to controls. Plasma CK levels were lower in the treatment group, showing less damage to the muscle tissue. With maximum isometric voluntary contraction force the researchers actually saw a significant increase in force production

for the treatment group. This phenomenon has not been seen in other research and should be pursued for further research.

Role of Stretching

Active eccentric contractions of a muscle are not the only way to prevent a muscle from experiencing DOMS. Passive stretching or isometric contractions also elicit a response that may play a protective roll against DOMS (46). As explained earlier, Pizza et al (46) looked at the response of the inflammatory response to eccentric and isometric contractions, and passive stretching. He observed a positive result in that passive stretching reduced the level of inflammation in a bout of exercise.

On the flipside, some researchers (22, 28) have also looked at stretching's role on DOMS. In these articles the researchers had their subjects undergo a stretching protocol immediately before an eccentric bout of exercise. In these articles the researchers saw no difference in DOMS between groups. Some reasons why these protocols may not have worked will be explained later.

Role of Antioxidants

Phenolic compounds have been shown to be effective in alleviating symptoms in inflammatory conditions (52). Smith (50) was able to show a relationship between DOMS and the inflammatory response. Phenolic compounds are found in cherries (13), therefore there may be a possibility that the antioxidant properties of tart cherries will help reduce DOMS due to eccentric training. Connolly et al (13) gave a group of subjects a mixture of tart cherry juice, which they took morning and night for four days. After the four days they performed a bout of eccentric exercise. Then for four more days they consumed the cherry drink. The researchers were able to reduce DOMS in flexion and extension but not in palpation. They were also able to reduce the amount of strength loss experienced by the treatment group. Because the cherry juice

did not prevent DOMS in all aspects the researchers state that they are unable to conclude that cherries had a protective effect. But it was able to maintain functionality by reducing the amount of strength loss.

Difference in Research Protocol

In both Evans et al (18) and Nosaka et al (40) they looked at the effect of heating or warming the muscle, but with regards to DOMS they both had differing results. Both used microwave diathermy as a means of heating the muscle. The difference was in the timing of the exercise protocol after the treatment. Evans et al (18) performed the exercise bout within seconds after the heating protocol. Nosaka et al (40) allowed the treatment to take effect for 1 day. Both researchers (18, 40) may have been able to elicit a response. But the muscle may need time to adapt to the treatment. The timing between the treatment and the exercise bout may not be long enough for an adaptation to take place within the muscle to aid in the protection from eccentric damage. In the articles that have seen the protective effect, it appears several days after the protocol. Whether it was an eccentric bout (10), isometric bout, passive stretching (46), or heating (40) the response was seen with at least 1 day given for adaptations to be seen.

Another area that may be overlooked in the prevention of DOMS and muscle damage is the volume of work being done by each subject. Some researchers have had their subjects do as many as 70 eccentric contractions (9, 39, 53). This volume of contractions is not what would be typically seen in a real-world setting. Though protection may have occurred it may not have been seen to the extent that it really worked, due to the high volume of repetitions that these researchers had their subjects undergo. By doing a more realistic model of what would be seen in the real world, we could possibly show a greater amount of protection against muscle damage and DOMS. Also, the damage seen by many of these researchers (9, 18) was characteristic of a condition called rhabdomyolysis (21). Symptoms of rhabdomyolysis include severe muscle

soreness, reduction in range of motion, decreased muscle strength, and compartmental syndrome among others (21). Compartmental syndrome is swelling of the musculature and other tissue within a connective tissue compartment (6). One recommendation for avoiding rhabdomyolysis is to avoid "extreme eccentric exercise" (21). To better determine if DOMS can be prevented or reduced in the average person, a protocol of realistic weight, reps, and sets would be more appropriate.

Conclusion

The prevention of DOMS may be advantageous for the adherence to an exercise protocol by novice exercisers. To date, the best way to prevent DOMS has been to produce DOMS with a prior bout of eccentric exercise (9, 31). There has been other ways of preventing DOMS demonstrated (40, 46). Heating the muscle, as demonstrated by Nosaka et al (40) may not be realistic to the average population. Also this treatment protocol did not decrease DOMS to a great extent (40). Passive stretching and isometric contractions, as demonstrated by Pizza et al (46), blunted the inflammatory response. Though they did not measure DOMS directly, DOMS and the inflammatory response are correlated (50). The question now is whether a stretching protocol would prevent DOMS in an untrained individual. By using a treatment and exercise protocol comparable to one that may be seen in a local gym, we may be able to produce a regimen for preventing DOMS that may be immediately taken into the gym by an untrained individual.

Chapter 3

Methods

Subjects

Thirty untrained individuals between the ages of 18 and 30 will participate in this study. Subjects will not have been involved in a regular resistance or running exercise program for at least 6 months prior to enrolling in the study. Subjects will be randomly assigned into either the control (C) or one of two experimental groups. Subjects will be informed of the procedures and any possible risks of the study before signing an approved informed consent document. This study will be approved by the Brigham Young University Human Subjects Institutional Review Board.

Study Design

This study will use a randomized control design. There will be two experimental groups the first group (S) will undergo a passive stretching protocol of the quadriceps muscles on 3 separate occasions during the week preceding the resistance exercise protocol. One day after the last stretching session the subjects will return to the lab to be tested and undergo the resistance exercise protocol. The second experimental group (I) will undergo an isometric contraction protocol of the quadriceps muscles on 3 separate occasions during the week preceding the resistance exercise protocol. Again, 1 day after the last isometric contraction protocol the subjects will come back into the lab to undergo the resistance exercise protocol. Measurements will be taken before the resistance exercise protocol; then again within 5 minutes post exercise, and then 1d, 2d, 4d and 8d post exercise. The control group will not participate in the passive stretching or isometric contraction treatment protocols, but will be tested and undergo the resistance exercise protocol. Measurements will also be taken for the control group at the same post time points.

Pre-Exercise Measurements

On the first visit to the lab subjects will be measured for height (m), weight (kg), range of motion (ROM) of the knee, thigh girth (cm), and muscle soreness. On the day of the resistance exercise session each subject will establish their concentric one repetition maximum (1RM) for their right leg on both the leg extension machine and the leg press machine.

The ROM of the knee of the right leg will be determined by having each subject lie on their back with their hips at 90º of flexion. The researcher will then passively move the subject's heel toward the buttocks. The measurement will be taken at the knee joint with a goniometer.

Thigh girth of the right leg will be measured by having each subject lie flat on their backs with hips and knees extended and relaxed. The researcher will measure from the anterior superior iliac spine (ASIS) to the top of the patella. The researcher will measure the girth of the thigh using a Gulick tape measure. Measurements will be taken at three separate sights; 50%, 70%, and 90% the distance between the ASIS and the top of the patella. Three measurements will be taken at each site. The average of the three measurements will be used for analyses.

Muscle soreness will be measured subjectively (10) using a scale of 1 to 10. This will be assessed with palpation at rest and during contraction under a standard load representing 25% of their pretesting concentric only 1 RM. Palpation soreness will be determined at 3 separate sights; 50%, 70%, and 90% the distance between the ASIS and the patella. Marks will be made on the subject's thigh, and soreness measurements will be taken with the subject sitting upright. Palpation soreness will be determined using a pressure algometer that ensures the same amount of palpation pressure is placed on the thigh with each test. The subjects will determine on a scale of 1 to 10 where their level of soreness is. One will indicate no pain, 10 will indicate extreme pain. The measurement will be taken 3 times. The average will be used for analyses. To assess soreness during contraction the subject will perform a unilateral leg extension of the test leg

using 25% of their 1RM. Determination of the subjects 1RM will be explained in the following paragraph. Each subject will extend their knee and describe their level of soreness, as explained above, on a scale of 1 to 10. The subject will then lower the leg and again describe the level of soreness. This way we can determine the level of soreness for both concentric and eccentric contractions.

The concentric only 1RM for the leg extension exercise will be determined by having the subjects perform a unilateral leg extension exercise. The right leg of each subject will be used to determine the 1RM. After a warm-up entailing 5 minutes of cycling at 90 to 100 watts, the subjects will be seated in the leg extension machine to perform the test. The initial load will begin at 50 kgs. Upon successful completion the next attempt will increase in weight determined by the difficulty of the previous attempt. If the attempt was extremely easy, the resistance will increase by 25 kgs. When it becomes more difficult the increase in resistance will be by 10 kgs, until to the subject cannot successfully complete the next attempt at which point the increase in resistance will go up by 5 kgs increments. Upon the unsuccessful completion of an attempt, the weight will be decreased by 5 kgs. Between each attempt the subject will be given a minimum of 5 minutes of rest (30), before the next attempt. The highest resistance successfully lifted will be the subjects 1RM. For a successful attempt the leg must start at a rested position with the weight stack completely lowered, the attempt will finish with the leg completely extended. If either of these requirements is not met the repetition will be considered failed. After each attempt the researchers will lower the weight for the subject ensuring a maximal concentric attempt with no eccentric component and reducing the amount of damage to the exercised muscle.

The concentric only 1RM for the leg press exercise will be determined by having the subjects perform a unilateral leg press exercise. The right leg of each subject will be used to

determine the 1RM. Subjects will follow the same protocol for determining their 1RM as described above for the leg extension. Again, after each attempt the researchers will lower the weight for the subject ensuring a maximal concentric attempt with no eccentric component and reducing the amount of damage to the exercised muscle.

Passive Stretching Protocol

Each subject in S will undergo a passive stretching treatment on 3 separate occasions prior to the resistance exercise bout. The subjects will be stretched by using the standing quadriceps stretch. Each stretch will be held for 4 seconds then relaxed for 1 second. This process will be repeated for a duration of 5 minutes to mimic the same protocol used by Pizza el al (46) in their study in which they observed inflammatory responses to passive stretching and isometric contraction. The subjects will then perform the same protocol on the other leg.

The standing quadriceps stretch will be done by having each subject stand next to a wall or supporting object. Holding themselves stable with one arm they will lift the opposite leg by flexing the knee, bringing the heel towards the buttocks. They will then grab the top of their foot with the hand on the same side of the body. They will pull their heel as close to their buttocks as they can and hold the stretch.

Isometric Contraction Protocol

Each subject in group I will participate in an isometric contraction treatment. On three occasions during the week prior to the resistance exercise protocol each subject will perform an isometric contraction treatment of the quadriceps muscles. To do this they will be seated in a leg extension machine with their knee at 60° of flexion (54). The weight will be set at an amount that the subject cannot move with a maximal single leg effort. For the contraction protocol the subjects will maximally contract their quadriceps for 5 seconds then relax for 1 second. They will do this for a duration of 5 minutes (46).

Resistance Exercise Protocol

Each subject in S, I, or C will undergo the resistance exercise protocol. After the 1RM is determined for the subject's right leg, the subject will then perform a leg press and leg extension workout on this leg. The resistance protocol will include 4 sets of 12 repetitions of the single leg press and the single leg extension. All four sets of the single leg press exercise will be performed in the manner indicated below, followed by a 2 minute rest, then the single leg extension exercise. The subjects will use a resistance that is 60%-65% of the 1RM determined for the right leg. Each subject will follow a tempo of 4-2-2 where the subject will lower the weight within 4 seconds, relax at the bottom for 2 seconds, and then raise the weight within 2 seconds. This will ensure that the eccentric and concentric stress is comparable among subjects. Between each set the subjects will rest for 2 minutes then resume the next set.

Post Exercise Measurements

At 5 minutes following the exercise protocol the subject will be assessed in the following order: thigh girth, passive soreness, ROM of the knee joint, active soreness. The measurements will again be taken 1d, 2d, 4d, and 8d after the exercise protocol. Subjects will refrain from any organized exercise activities during the entire study period.

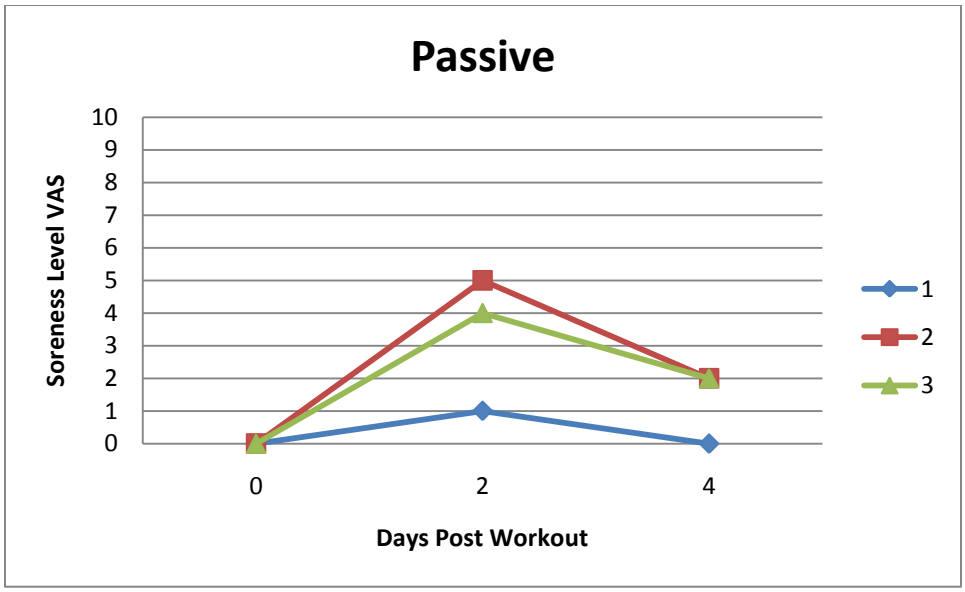

Figure 1a

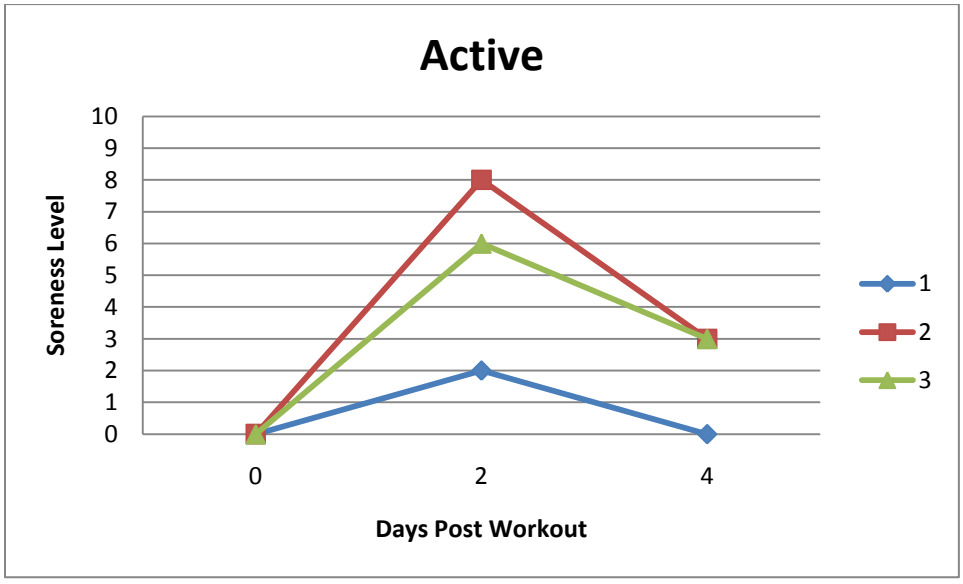

Figure 1b

Figure 1. (a) Depicts soreness levels with 2 kgs of pressure placed directly over the rectus femoris muscle at 0 d, 2 d and 4 d post exercise. (b) Depicts soreness levels with active leg extension performed with 25% of subjects 1RM at 0 d, 2 d, and 4 d post exercise.

Pilot Study

To determine if the protocol selected would elicit muscle soreness, a pilot study was performed on three subjects. The average age of the subjects was 25. Each subject was verbally informed of risks associated with the pilot study. Upon entering the lab each subject pedaled on a cycle ergometer for a period of 5 minutes at an intensity of 100 watts.

After the initial warm up, each subject then sat at the leg extension machine. At this point the subjects attempted a 1RM leg extension. If the subject was able to complete the repetition they were given 2 minutes to recover before attempting a higher weight. The subjects would concentrically extend the knee were upon the investigator would lower the weight for the subject, ensuring that the subject did not perform the eccentric action of the leg extension. When a weight could not be completed the subject was then given 5 minutes to recover before attempting the same weight again. If they were unsuccessful at extending the knee after the 5 minute rest the last weight they were able to extend was used as their 1RM. If they were able to fully extend the knee they were given 5 minutes more rest before another attempt.

After the 1RM was determined each subject was asked to perform the exercise protocol as outlined using 60-65% of their 1RM. Soreness levels were determined before the 1RM was determined then 2 days and 4 days after. After completion of the study subject 1 revealed that he was an avid cyclist, thus explaining why the subject's level of soreness didn't reach levels similar to the other subjects. The pilot study indicates that this exercise protocol is sufficient to induce muscle soreness and thus may be used for research.

Statistical Analysis

A 3 X 6 repeated measures design with four dependent variables will be employed. The factors of treatment (passive stretching, isometric contraction, control) and time (pre, immediate post, 1d, 2d, 4d, 8d) are the independent variables with the dependent variables being ROM,

thigh girth, level of soreness, and 1RM.

In the event of interaction a Tukey's post-hoc analysis will be used to make all pair wise comparisons to locate significant differences. For all statistical analysis in this study, a P-value < 0.05 will be used to establish significant differences. Results will be reported as the means ± standard deviation (SD) unless otherwise indicated.

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