THE EFFECTS OF CONTINUOUS COMPRESSION AS A THERAPEUTIC INTERVENTION ON DELAYED ONSET MUSCLE SORENESS FOLLOWING ECCENTRIC EXERCISE

by

Brent F. Fedorko

B.S., Pennsylvania State University, 1999

M.S., University of Pittsburgh, 2001

Submitted to the Graduate Faculty of

The School of Education in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy

University of Pittsburgh

2007

UNIVERSITY OF PITTSBURGH

SCHOOL OF EDUCATION

This dissertation was presented

by

Brent F. Fedorko

It was defended on

August 22, 2007

and approved by

Elizabeth Nagle-Stilley, PhD, Assistant Professor, Health and Physical Activity

Robert Robertson, PhD, Professor, Health and Physical Activity

Tony Salesi, MA, ATC, PT, Coordinator of Athletic Training for Olympic Sports

Dissertation Advisor: Fredric Goss, PhD, Associate Professor, Health and Physical Activity

THE EFFECTS OF CONTINUOUS COMPRESSION AS A THERAPEUTIC INTERVENTION ON DELAYED ONSET MUSCLE SORENESS FOLLOWING ECCENTRIC EXERCISE

Brent F. Fedorko, PhD

University of Pittsburgh, 2007

It has been established that exercise-induced muscle damage occurs following exercise in individuals who are not accustomed to rigorous physical activity. The damage results in a dull aching pain, known as delayed onset muscle soreness (DOMS). Other clinical signs associated with DOMS include a decreased range of motion, swelling, and strength loss; all of which can lead to an impaired ability to perform daily activities. **PURPOSE:** The purpose of this study was to determine the effect of a continuous compression treatment protocol on the clinical signs and pain associated with DOMS. **METHODS:** Twenty male subjects, aged 18-35, were randomly assigned to a continuous compression treatment protocol (CC) or a no treatment protocol (N). The induction of DOMS was accomplished by having the subjects perform repetitive eccentric contractions of the quadriceps muscle group. Subjects completed three sets of knee extension repetitions. Immediately following the eccentric contractions, subjects in the (CC) group applied a compression garment over the involved thigh. The (CC) subjects wore the compression garment continuously during the 3-day recovery period. The subjects in the control group did not receive any treatment. All subjects reported at 24, 48, and 72-hour post exercise for measurement of the dependent variables of passive muscle soreness, active muscle pain, rating of perceived exertion associated with the active muscle, swelling, extension and flexion angles, and strength measures. **RESULTS:** The primary findings of this investigation were that

continuous compression resulted in a significant difference in the overall perception of passive muscle soreness across time at 24 and 48-hours post exercise ($F(3, 54) = 3.75$, $p < 0.05$). In addition, regardless of treatment protocol, there were significant differences across time observed ($p < 0.05$) in active muscle pain, rating of perceived exertion associated with the active muscle, distal thigh circumference, supine knee flexion angle, and knee extension angle following the induction of DOMS. **CONCLUSIONS:** Results suggest that continuous compression is beneficial in reducing muscle soreness during the first 48-hours after unaccustomed eccentric exercise. By providing mechanical support to the tissues, a compression garment may decrease the detrimental effects associated with DOMS.

TABLE OF CONTENTS

AKNOWLEDGMENTS XIII

1.0 INTRODUCTION 1

1.1 RATIONALE 1

1.2 STATEMENT OF PROBLEM 4

1.3 HYPOTHESES 4

1.4 SIGNIFICANCE 5

2.0 LITERATURE REVIEW 6

2.1 INTRODUCTION 6

2.2 DELAYED ONSET MUSCLE SORENESS (DOMS) 6

2.3 ECCENTRIC CONTRACTIONS 8

2.4 CLINICAL SIGNS AND SYMPTOMS 9

2.4.1 Pain. 9

2.4.2 Swelling. 10

2.4.3 Range of Motion. 10

2.4.4 Strength. 11

2.4.5 Creatine Kinase. 11

2.5 THEORIZED MECHANISMS 12

2.5.1 Lactic Acid. 12

2.5.2 Muscle Spasm. 13

2.5.3 Enzyme Efflux Theory. 14

2.5.4 Muscle Damage / Torn Connective Tissue 14

2.5.5 Inflammation 15

2.5.6 Theory Sequence 16

2.6 PREVENTION OF DOMS 17

2.6.1 Pharmacological Treatments. 18

2.6.2 Nutritional Supplements 18

2.6.3 Therapeutic Treatments. 19

2.6.3.1 Cryotherapy. 19

2.6.3.2 Electrical Current Techniques. 19

2.6.3.3 Hyperbaric Oxygen. 20

2.6.3.4 Massage 20

2.6.3.5 Stretching 21

2.6.3.6 Ultrasound. 21

2.7 COMPRESSION 21

2.8 CONCLUSION 23

3.0 METHODS 24

3.1 EXPERIMENTAL DESIGN 24

3.2 VARIABLES. 24

3.3 SUBJECTS. 25

3.4 PROTOCOL. 26

3.4.1 Day O (Orientation): 26

3.4.2 Day 1: 28

3.5 INDUCTION OF DOMS 28

3.6 MEASUREMENT OF DEPENDENT VARIABLES 30

3.6.1 Muscle Soreness/Muscle Pain/RPE-AM. 30

3.6.2 Swelling. 31

3.6.3 Flexion Angle. 32

3.6.4 Extension Angle. 33

3.6.5 Strength. 34

3.7 DAYS 2 (24-HOURS), 3(48-HOURS) AND 4 (72-HOURS) – POST EXERCISE: 35

3.8 TREATMENT PROTOCOLS 35

3.8.1 Continuous compression. 35

3.8.2 Control. 35

3.9 STATISTICAL ANALYSIS 36

4.0 RESULTS 37

4.1 SUBJECT CHARACTERISTICS 37

4.2 PASSIVE MUSCLE SORENESS 38

4.3 ACTIVE MUSCLE PAIN 39

4.4 RATING OF PERCEIVED EXERTION ASSOCIATED WITH THE ACTIVE MUSCLE (RPE-AM) 42

4.5 CIRCUMFERENCE MEASURES 45

4.6 STRENGTH (ONE REPETITION MAXIMUM) 47

4.7 FLEXION ANGLES (PRONE AND SUPINE) 48

4.8 EXTENSION ANGLE 50

5.0 DISCUSSION 52

5.1 PASSIVE MUSCLE SORENESS 53

5.2 ACTIVE MUSCLE PAIN 54

5.3 RATING OF PERCEIVED EXERTION ASSOCIATED WITH THE ACTIVE MUSCLE 55

5.4 SWELLING 56

5.5 STRENGTH 57

5.6 RANGE OF MOTION 58

5.7 LIMITATIONS 59

5.8 CONCLUSIONS 60

5.9 RECOMMENDATIONS FOR FUTURE RESEARCH 60

APPENDIX A 63

APPENDIX B 65

APPENDIX C 73

APPENDIX D 75

APPENDIX E 77

APPENDIX F 79

APPENDIX G 81

APPENDIX H 84

BIBLIOGRAPHY 86

LIST OF TABLES

Table 1. Subject Descriptive Data 37

Table 2. Passive Muscle Soreness for Compression and No Treatment Subjects at Four..... 38

Table 3. Repeated Measures ANOVA Summary for Passive Muscle Soreness..... 38

Table 4. Active Muscle Pain Measured at the Midpoint of the Warm-Up Repetitions..... 40

Table 5. Repeated Measures ANOVA Summary for Active Muscle Pain Measured at the Midpoint of the Warm-Up Repetitions. 40

Table 6. Active Muscle Pain Measured Immediately Following the Warm-Up Repetitions. 41

Table 7. Repeated Measures ANOVA Summary for Active Muscle Pain Measured Immediately Following the Warm-Up Repetitions. 42

Table 8. RPE-AM taken at the Midpoint of the One Repetition Maximum Warm-Up..... 43

Table 9. Repeated Measures ANOVA for Rating of Perceived Exertion Associated with the Active Muscle at the Midpoint of the Warm-Up Repetitions 43

Table 10. RPE-AM taken during the Final Warm-Up Repetition. 44

Table 11. Repeated Measures ANOVA for Rating of Perceived Exertion Associated with the Active Muscle(s) taken during the Final Warm-Up Repetition. 45

Table 12. Midthigh Circumference Measures for Compression and No Treatment Subjects at Four Time Points. 45

Table 13. Repeated Measures ANOVA Summary for Midthigh Circumference. 46

Table 14. Distal Thigh Circumference Measures for Compression and No Treatment Subjects at Four Time Points. 46

Table 15. Repeated Measures ANOVA Summary for Distal Thigh Circumference. 46

Table 16. Strength Measures for Compression and No Treatment Subjects at Four Time Points. 47

Table 17. Repeated Measures ANOVA Summary for Strength Measurement Comparing Compression and No Treatment Subjects at Four Time Points. 48

Table 18. Prone Knee Flexion Angle Measures. 48

Table 19. Repeated Measures ANOVA Summary for Prone Knee Flexion Angle. 49

Table 20. Supine Knee Flexion Angle Measures 49

Table 21. Repeated Measures ANOVA Summary for Supine Knee Flexion Angle. 49

Table 22. Extension Angle Measures. 50

Table 23. Repeated Measures ANOVA Summary for Extension Angle Measures. 51

LIST OF FIGURES

Figure 1. Experimental Protocol 27

Figure 2. Timeline of Symptom Measurement During Experimental Trials 29

Figure 3. Passive Muscle Soreness over a Four Day Time Period. 39

Figure 4. Active Muscle Pain at the Midpoint of the Warm-Up Repetitions over a Four Day Time Period for All Subjects. 41

Figure 5. Rating of Perceived Exertion Associated with the Active Muscle at the Midpoint of the Warm-Up Repetitions for All Subjects. 44

Figure 6. Distal Thigh Circumference over a Four Day Time Period for All Subjects. 47

Figure 7. Supine Knee Flexion Angle over a Four Day Time Period for All Subjects. 50

Figure 8. Knee Extension Angle over a Four Day Time Period for All Subjects 51

AKNOWLEDGMENTS

The author would like to acknowledge the committee members: Drs. Fredric Goss, Elizabeth Nagle-Stilley, Robert Robertson and Mr. Tony Salesi for their guidance throughout this investigation. I would also like to thank Dr. Barbara Wainwright for her help in the statistical analysis component of this investigation and Jason Zaleski for his help in data collection. I would like to extend a special thank you to Dr. Fredric Goss for his support and mentoring throughout my dissertation process and also Dr. Elizabeth Nagle-Stilley, who has advised and provided much needed support and encouragement all through my educational journey.

I offer a sincere thank you to Donna Farrell, Denise Morrin, Dianne Drish and Chris Miller for their patience and always being available to work with me, help me out, and keep me on track with whatever was needed. Also, throughout my years at the University of Pittsburgh, whenever I needed support or advice on any matter, I could always turn to Mr. Louis Fabian, for his advice and encouragement for which I am truly grateful – Thanks Lou!

I would also like to thank my family and friends (especially the crew from 142) for their support, encouragement, and amazing abilities and talents that helped me and contributed to my success. Finally, I would especially like to thank my parents Bob and Carol, for always being there to provide support and encouragement whenever I need it.

1.0 INTRODUCTION

1.1 RATIONALE

It has been well-established that exercise-induced muscle damage occurs following intense exercise involving eccentric contractions. The damage results in a dull, aching pain, known as delayed onset muscle soreness (DOMS). DOMS, the most common type of muscle pain (Solomonow and D'Ambrosia, 1987) is characterized by pain/soreness that begins to occur 8-24 hours post-exercise (Smith, 1991; Newham, 1988; Hough, 1902), and increases in intensity until it peaks at 24-72 hours and subsides 5-7 days post exercise (Bobbert, Hollander, and Huijing, 1986; Armstrong, 1984; Assmussen, 1956). Other clinical signs associated with DOMS are consistent and include swelling, a reduction in muscle strength and a decreased range of motion, all of which can contribute to an impaired ability to perform routine daily activities (Clarkson and Hubal, 2002; McHugh, Connolly, Eston and Gleim, 1999; Clarkson, Nosaka, and Braun, 1992; Brynes, Clarkson, White, Hsieh, Frykman, and Maughan, 1985; Armstrong, 1984). Individuals who are not accustomed to regular physical are more likely to develop DOMS than highly trained athletes.

Numerous studies have been published describing various strategies and interventions (i.e. nutritional supplementation, pharmaceutical treatments, and therapeutic modalities) to prevent the signs and symptoms of DOMS from developing or to alleviate them once present.

These studies have been met with limited success and a sound and consistent treatment for DOMS has not yet been established.

Even though DOMS is a commonly experienced phenomenon that can occur in any individual independent of fitness level, the exact mechanism(s) responsible for DOMS are not completely understood. A number of theories have been proposed in an attempt to explain the underlying mechanism(s) responsible for DOMS. These theories include the following: lactic acid (Assmussen, 1956), muscle spasm (DeVries, 1961), connective tissue and muscle damage (Stauber, 1989; Friden, Sjostrom, and Ekblom, 1981; Tullson and Armstrong, 1978; Abraham, 1977; Hough, 1902), enzyme efflux (Armstrong, 1984), and inflammation (Smith, 1991). To date, no research has been able to conclusively attribute any one mechanism as the primary cause of DOMS or its related sensations; however, much of the research conducted involving exercise-induced muscle damage has documented events similar to those associated with an acute inflammation response.

Since the body responds to all forms of tissue injury by activating the inflammatory response, it is unlikely that a separate and unique response has evolved to deal with the damage incurred following unaccustomed eccentric exercise (Smith, 1991). The initial treatment goals following tissue damage are to protect the damaged tissues and control pain, swelling, and loss of function. This is accomplished by applying the R.I.C.E. principle immediately following the incident of damage (**Rest** – lets the healing process begin, **Ice** – provides an analgesic effect, **Compression** – minimizes swelling, and **Elevation** – assists in controlling swelling).

The harmful effects of DOMS are well documented. A variety of treatment techniques and interventions designed to eliminate or lessen the effects of DOMS have been attempted. These techniques are generally grouped into 3 categories and include nutritional

supplementation, pharmacological treatments and therapeutic modalities (Connolly, Sayers, and McHugh, 2003). Although some success has been reported with various treatments, no standard universal treatment or prophylaxis exists for this condition. However, promise has been seen recently in the use of continuous compression as a significant mediator for minimizing the magnitude of the effects of exercise-induced muscle damage (Kraemer, Bush, Wickham, Denegar, Gomez, Gotshalk, Duncan, Volek, Newton, Putukian, and Sebastianelli, 2001). Immediate compression has been demonstrated to be an effective technique for limiting swelling by providing mechanical support to the tissues and reducing motion in the injured structure.

According to Kraemer et al. (2001), the purpose of compression is to mechanically reduce the amount of space available for swelling by applying external pressure around the injured area. When proteins from the damaged muscle are released into the intracellular fluid, oncotic pressure is increased. Fluid is then drawn out of the circulatory system in an attempt to equalize the osmotic gradient across the capillary membrane resulting in edema formation. The only way that edema can be removed to restore the oncotic pressure system is through the lymph system (Kolb and Denegar, 1983). When edema accumulates in the muscular compartment, it leads to an increased intracompartmental pressure. This pressure inhibits lymphatic and venous drainage of the area, thus promoting further swelling. However, compression can influence swelling by exerting an external force that tends to hold fluid within the capillary system allowing for a new filtration pressure balance to be reached sooner (Kraemer et al. 2001), thus resulting in a reduction in the severity and duration in the functional signs and pain associated with exercise-induced muscle damage. The efficacy of continuous compression following eccentric activity in the treatment of delayed onset muscle soreness and its related signs has received little attention.

1.2 STATEMENT OF PROBLEM

The purpose of this study was to determine the effect of a continuous compression treatment protocol on the clinical signs and pain associated with exercise-induced muscle damage following eccentric exercise. The study compared the effects of a treatment protocol with a control group on DOMS and its related clinical signs following a bout of eccentric exercise. The following treatment protocol was administered: Group 1 – continuous compression (CC) and Group 2 – no treatment (N).

The effect of the treatment protocol on DOMS was examined by measuring the dependent variables of passive muscle soreness, active muscle pain, rating of perceived exertion associated with the active muscle, swelling, extension and flexion angles, and strength measures.

1.3 HYPOTHESES

The following hypotheses were tested:

It was hypothesized that when compared to the no treatment group (N) at 24, 48 and 72 hours following a bout of eccentric exercise designed to induce DOMS, subjects in the (CC) treatment group would display:

a. an overall lower perception of passive muscle soreness.
b. an overall lower rating of perceived exertion in the active muscle(s) during submaximal resistance exercise.
c. an overall lower rating of active muscle pain during submaximal resistance exercise.

d. a decreased amount of swelling.

e. higher mean extension angles.

f. higher mean flexion angles.

g. greater strength values.

1.4 SIGNIFICANCE

Sporting and non-sporting performances alike (i.e. routine daily activities) are impaired if the individual participating is sore or injured. Thus, any practice that limits the extent of damage and/or speeds up the recovery process would be of great interest and practical value to the athletic population, as well as the general public. As a result, DOMS research serves to benefit a wide audience. Information elicited from this study provides active individuals with valuable information concerning the role of continuous compression in managing and treating the clinical signs and pain associated with DOMS.

2.0 LITERATURE REVIEW

2.1 INTRODUCTION

It has been suggested that DOMS represents inflammatory pain following connective and/or muscle tissue injury (Smith, 1991). Inflammation is the body's non-specific response to damage in which the same events occur under the same mediators independent of the course (Denegar, 2000). With any injury, if the inflammatory process is not completed, tissue is not repaired. Compression of the injured area following structural damage is an important step in managing this process. Continuous compression is used to minimize the swelling associated with the inflammation process. However, the effects of continuous compression as a therapeutic intervention on DOMS and its clinical signs have received little attention. Therefore, this investigation examined the effects of continuous compression on DOMS and its clinical signs and symptoms.

2.2 DELAYED ONSET MUSCLE SORENESS (DOMS)

There are two primary types of muscle soreness. The first type of muscle soreness is acute and is frequently associated with muscle fatigue. It is transient and occurs immediately after exercise and may last for up to 6 hours before subsiding (Isabell, Durrant, Myrer, and Anderson, 1992).

The second type of soreness involves delayed muscle pain. Delayed onset muscle soreness (DOMS) is defined as the sensation of discomfort or pain in skeletal muscle(s) that occur(s) following unaccustomed muscular exertion (Armstrong, 1984). DOMS usually begins to appear approximately 8 hours following exercise, peaks from 24-72 hours and gradually subsides so that the muscle becomes symptom-free after 5 or 7 days (Newham, 1988; Bobbert et al., 1986; Friden, 1984). DOMS can occur in anyone regardless of fitness level; although it occurs more often in untrained individuals than in persons accustomed to regular exercise (Lenn, Uhl, Mattacoloa, Boissonneault, Yates, Ibrahim, and Bruckner, 2002; Clarkson et al., 1992; Friden, Sfakianos, and Hargens, 1986).

The earliest known study of exercise-induced-muscle damage was published in 1902, in which Hough described the phenomenon of DOMS in considerable detail and hypothesized the etiology as being the result of microtears in the muscle. In his study, Hough differentiated between the soreness associated with muscle fatigue and the soreness that showed up some hours after exercise (delayed soreness) regardless of muscle fatigue. He attributed the delayed soreness to pain arising from microruptures in the muscle fibers and the associated connective tissue directly related to the forces developed during exercise and their rate of force development. He demonstrated that eccentric movements produced muscle discomfort after exercise and it was further demonstrated that measurable muscle weakness accompanied the muscle discomfort. Even after a brief conditioning period allowing for a resolution of the soreness, the weakness persisted for several days. It was suggested that the diminished performance resulted from a lowered inherent capacity of the muscle to produce force, as well as from a reduced effort due to the sensation of soreness. He interpreted the weakness as being the result of intramuscular damage to the structural elements of the muscle and its surrounding tissues.

DOMS has been the object of much discussion and investigation among researchers; however, the basic observations of Hough continue to be supported.

2.3 ECCENTRIC CONTRACTIONS

Although DOMS may occur following any unaccustomed exertion, the most predictable way of inducing it is to subject muscle to high-force repetitive eccentric contractions in which the activated muscle is forced to elongate while producing tension (Chleboun, Howell, Baker, Ballard, Graham, Hallman, Perkins, Schauss, and Conaster 1995; Newham, 1988; Friden et al., 1986; Komi and Viitasalo, 1977; Assmussen, 1956; Abbott, Bigland and Ritchie, 1952). Eccentric exercise is defined as the force generated muscle activation involving lengthening of a muscle (Garrett and Tidball, 1988). Eccentric muscle activity is an integral component in routine daily activities such as stair descent, squatting, and lowering objects. Athletic participation requires eccentric muscle force for activities such as limb deceleration when throwing a ball, bicycling and downhill running.

During eccentric activity, the force developed in the muscle is approximately twice that developed during isometric contractions; however, the total number of attached cross bridges in a strongly bound state is only ~10% greater than that during an isometric contraction (MacIntyre, Reid, Lyster, Szasz, and McKenzie, 1996; Faulkner, Brooks, and Opiteck, 1993). As a result, the force is distributed over a smaller cross-sectional area of muscle, producing a greater tension per active motor unit, thus increasing the risk for mechanical damage to the myofibrillar materials setting off an inflammatory response that leads to PGE_2 and leukotriene synthesis (MacIntyre

etal. 1996; Armstrong, 1990; Friden et al., 1981). Any form of activity that primarily involves eccentric contractions will produce greater delayed soreness than other types of muscle actions.

2.4 CLINICAL SIGNS AND SYMPTOMS

DOMS presents as a dull, aching pain with swelling, a decreased range of motion (ROM) and strength loss. The pain and impaired clinical signs lead to the short-term disability associated with DOMS; however, there is no evidence that there is any long-term damage or permanent reduced muscle function (Armstrong, 1984). The measurement of these clinical signs (swelling, ROM and strength) coupled with the assessment of pain is the most appropriate way to observe and manage the effects of DOMS (Clark, 1994).

2.4.1 Pain.

A major function of pain is to protect the organism by signaling injury infringement on the tissues. However, the pain associated with DOMS does not fulfill this role. Individuals often begin to feel muscular soreness in the area of the musculotendinous junction between 8 and 24 hours postexercise, with peak levels occurring at around 24-72 hours (Newham, 1988; Bobbert et al., 1986). Because the soreness appears some time after the activity, it presumably does not function to prevent overuse during the activity bout in which the injury occurs (Armstrong, 1984).

The exact mechanism responsible for the delay in pain is not fully understood. However, according to Smith (1991) the biomechanical explanation is related to the delay in macrophage

entry into the injured area. In response to eccentric-based exercise, macrophages are present in large numbers at 24 and 48 hours after tissue injury. She suggested that the sensation of pain was related to the synthesis of PGE_2 by the macrophage. PGE_2 directly causes the sensation of pain by activating pain afferents, most likely myelinated type III and unmyelinated type IV pain afferents (Ebberling and Clarkson, 1989).

2.4.2 Swelling.

Swelling occurs due to the attraction of water to free proteins accumulating in the interstitum following injury. This accumulation results in a disruption of normal capillary filtration pressure, allowing for swelling to occur (Denegar, 2000). Several investigators have suggested that swelling is involved in the generation of pain incurred during DOMS due to the increased local tissue pressure it causes. It has been proposed (Smith, 1991) that increases in intramuscular pressure occur during a contraction or during palpation that then provide a mechanical stimulus for the PGE_2 sensitized receptors that initiate the sensation of DOMS.

2.4.3 Range of Motion.

Range of motion (ROM) is defined as the arc over which a joint may operate, and this constrains the muscle length range (for single joint muscles) (Warren, Lowe, and Armstrong, 1999). A reduction in joint range of motion and a reduction in shock attenuation have been observed during periods of severe muscle soreness. It seems likely that the restriction in motion associated with DOMS arises from a loss of strength (Clarkson et al., 1992), as well as swelling within the

perimuscular connective tissues, especially in the regions of the myotendinal junctions (Howell, Chila, Ford, David, and Gates, 1985).

2.4.4 Strength.

Prolonged strength loss after eccentric exercise is considered to be one of the most valid and reliable indirect measures of muscle damage in humans (Warren et al., 1999). Numerous researchers have documented significant reductions in strength and power parameters during periods of DOMS. The mechanism by which eccentric exercise results in strength loss has not been clearly identified – although it is believed that either the inherent capacity of muscle to produce force is simply lowered due to damage to the contractile units or the strength decrements are secondary to the soreness perceptions (Newham, 1988).

2.4.5 Creatine Kinase.

Creatine Kinase (CK) - the primary enzyme regulating anaerobic metabolism - has been used as an indirect marker of DOMS in healthy individuals. However, since there is a strong correlation (r = 0.67 to 0.96) between CK and the clinical signs of DOMS, its measurement is unwarranted (Rodenburg, Bar, and DeBoer, 1993).

In addition, the use of any muscle protein in the blood as a marker of muscle damage can be difficult to measure and interpret because blood concentration is a function of what is being produced in the muscle and what is being cleared from the blood. CK activity may also be influenced by several factors other than damage, including genetic factors, age, and gender.

There is also great intersubject variation in CK response after exercise (Nosaka and Clarkson, 1996; Clarkson et al., 1992).

2.5 THEORIZED MECHANISMS

A number of theories (i.e. lactic acid, muscle spasm, enzyme efflux, torn connective tissue/muscle damage, inflammation) have been proposed to explain the underlying mechanism of DOMS. Each theory presents a version of chain reactions that manifest in the clinical symptoms/signs of DOMS. It has been established that DOMS results from overuse of muscle, although the specific etiology is not completely understood. It is likely that a combination of theories contribute to the underlying mechanism of DOMS.

2.5.1 Lactic Acid.

The lactic acid theory is based on the assumption that lactic acid continues to be produced following exercise. Initially hypothesized by Assmussen (1956), he stated that the soreness was due to an excessive buildup of lactic acid in the muscle following strenuous eccentric activity. This theory has largely been rejected as the higher degree of anaerobic metabolism and lactic acid production associated with concentric muscle contractions has failed to result in sensations of delayed soreness similar to those experienced by individuals following eccentric muscle contractions. Eccentric exercise, which has been shown to produce the largest amount of muscle soreness in individuals, requires relatively low energy expenditure. The energy used (per unit area of active muscle) is less in eccentric exercise than concentric exercise (Newham, McPhail,

Mills, and Edwards, 1983; Newham, Mills, Quigley, and Edwards, 1983). Thus it was reasoned that if lactic acid were the cause of DOMS, the muscle soreness would be expected to be greater after exercise with a higher anaerobic metabolic cost (concentric exercise) (Szymanski, 2001), which has been shown not to be the case (Newham et al., 1983).

Also adding to discredit this theory, is the fact that peak DOMS is experienced 24-72 hours after eccentric exercise and at this time lactate concentrations are minimal having already returned to pre-exercise levels. (Schwane, Watrous, Johnson, and Armstrong, 1983). For these reasons, it is unlikely that lactic acid accumulation is the underlying mechanism causing DOMS.

2.5.2 Muscle Spasm.

It has also been hypothesized that DOMS is caused by the tonic, localized spasm of motor units. The muscle spasm theory introduced by Devries (1961) followed his observation of increased levels of resting muscle activity after eccentric exercise. Spontaneous resting EMG activity in painful muscles was reported, and DeVries suggested that this led to a vicious circle. He proposed that increased resting muscle activation indicated a tonic localized spasm of motor units, which he believed led to ischemia, and the accumulation of pain substances. In turn this initiated a “vicious cycle”, as further stimulation of pain nerve endings caused further reflex muscle spasms and prolonged ischemic conditions.

However, the muscle spasm theory has been largely discounted by EMG studies (Bobbert etal. 1986; Howell et al., 1985; Abraham, 1977; Talag, 1973) that have failed to demonstrate increased activity in sore muscles or a correlation of activity to soreness following exercise.

2.5.3 Enzyme Efflux Theory.

The enzyme efflux theory was based on a model developed by Armstrong (1984), in which calcium from the interstitium accumulates in injured muscles following sarcolemmal damage. High mechanical forces produced during muscular exercise, particularly in eccentric exercise, cause disruption of structural proteins in muscle fibers and the connective tissue between the active cross bridges. Structural damage to the sarcolemma, or alterations in permeability of the cell membrane, resulting from the high mechanical forces is accompanied by an influx of Ca^{++} from the interstitium. Calcium accumulation inhibits cellular respiration and is thought to activate proteases and phospholipases causing further injury to the sarcolemma as the production of leukotrienes and prostaglandins occurs. The progressive deterioration of the sarcolemma following exercise would be accompanied by diffusion of intracellular components into the interstitium and plasma. These substances would then serve to attract monocytes that convert to macrophages and to activate mast cells and histocytes in the areas of injury. Armstrong hypothesized that the accumulation of histamine, kinins, and potassium in the interstitium stimulated free nerve endings of group IV sensory neurons, which activated the nocioceptors and result in the sensation of DOMS.

2.5.4 Muscle Damage / Torn Connective Tissue.

Both the muscle damage and torn connective tissue theories are consistent with the timing of soreness occurring following eccentric loads. The muscle damage theory, first proposed by Hough (1902), focuses on the disruption of the contractile component of muscle tissue following unaccustomed exercise, particularly at the level of the z-line. The disruption of the contractile

component is the result of increased tension per unit area caused by a reduction in active motor units during the eccentric actions. Nocioceptors situated in the muscle connective tissue and in the region of the arterioles, capillaries, and the musculotendinous junction are then stimulated leading to the sensation of pain.

There are also indications that connective tissue breakdown is part of the underlying mechanism causing DOMS. The connective tissue damage theory examines the role of the connective tissue that forms sheaths around bundles of muscle fibers (Cheung, Hume, and Maxwell, 2003). The torn tissue theory is supported by studies that trace markers of muscle damage such as myoglobin and describe the disruption of subcellular components. These disruptions result in damage to the sarcoplasmic reticulum and T tubule, both of which interfere with normal calcium metabolism.

2.5.5 Inflammation.

The inflammation theory is based on the finding that aspects of the inflammatory response are evident following repetitive eccentric muscle action. Smith (1991) proposed that acute inflammation was the mechanism underlying DOMS. According to this theory, tissue disruption after eccentric exercise leads to an increase in circulating neutrophils that migrate to the site of the injury. Once at the injury site, these neutrophils release chemicals that dissolve the injured cells. A second "shift" of white blood cells, the macrophages, begin infiltrating the damaged area at about 8-12 hours after exercise. These cells synthesize large quantities of prostaglandins (PGE_2s), which provide a mechanical stimulus for pain receptors to be sensitized leading to the sensation of DOMS.

At present, many experts agree that some aspects of the inflammatory response are seen secondary to either muscular or the connective tissue damage following strenuous eccentric activity.

2.5.6 Theory Sequence

The current belief among most researchers is that a single theory cannot explain the onset of DOMS as it may result from a sequence of events involving torn connective tissue, muscle damage, enzyme efflux, and inflammation. As a result, a hypothetical sequence of events has been proposed attempting to describe the mechanism(s) causing DOMS.

Following a review by Armstrong (1984), Smith's sequence (1991) started with the assumption that high tensile forces during eccentric muscle activity cause disruption of structural proteins in muscle fibers (connective tissue and muscle damage theory). This damage to the sarcolemma results in an accumulation of calcium that inhibits cellular respiration (enzyme efflux theory). Within a few hours there is a significant elevation in circulating neutrophils (inflammation theory). Intracellular components and markers of connective tissue and muscle damage diffuse into the plasma and interstitium. Mast cells and histamine production are activated and within a few hours there is a significant elevation in circulating neutrophils at the injury site (inflammation theory). Upon exposure to this inflammatory environment, macrophages produce PGE_2 that sensitize type III and IV nerve endings to mechanical, chemical, and/or thermal inflammation (inflammation theory). The accumulation of histamine, potassium, and kinins from active phagocytosis and cellular necrosis, in addition to elevated pressure from tissue edema and increased local temperature, then activate nocioceptors leading to the sensation of DOMS (inflammation).

The proposed mechanisms of DOMS have allowed researchers to investigate various treatment strategies aimed at alleviating the sensations of DOMS, restoring the maximal function of the muscles as rapidly as possible, and/or reducing the magnitude of the injury (Cheung et al., 2003).

2.6 PREVENTION OF DOMS

Clinicians and therapists are constantly searching for effective treatment modalities and protocols for preventing or reducing the severity of DOMS and its clinical signs. No intervention strategies currently exist for preventing DOMS. The only alternative is to treat the symptoms of DOMS after they occur with the ultimate goal being to restore maximal function of the eccentrically exercised muscles as rapidly as possible (Gulick and Kimura, 1996). Although the results are conflicting, to date, no treatment or intervention has consistently reduced DOMS or its signs. The absence of a preventative measure and the limited effectiveness of current treatment techniques may be the result of the lack of understanding surrounding the exact mechanism of DOMS.

Numerous pre-and post-exercise interventions have been investigated with respect to preventing DOMS or treating its subsequent signs but few treatment strategies have taken steps to address all the clinical signs associated with DOMS. These interventions can generally be grouped into 3 broad categories: (a) pharmacological treatments using non-steroidal anti-inflammatory drugs (NSAIDs), (b) interventions using nutritional supplements and (c) therapeutic treatments using physical modalities (Connolly et al., 2003). Minimal supporting evidence exists for any particular treatment technique.

2.6.1 Pharmacological Treatments.

The value of NSAID therapy in the treatment of DOMS is unclear, with the majority of studies (Lanier, 2003; Connolly et al., 2003) showing no effect despite a possible theoretical basis for its efficacy. The use of anti-inflammatory medication in response to exercise-induced muscle damage has not been successful in decreasing muscle soreness or the degree of inflammation associated with it (Gulick, Kimura, Sitler, Paolone, and Kelly, 1996).

In addition, recently there have been concerns that the use of NSAIDs in attempting to treat DOMS may actually negatively interfere with the inflammation process and delay the healing process (Prentice, 2003).

There is also disagreement between clinicians as to the safety of NSAID therapy use in trying to prevent and/or treat muscle injury because of the vast opportunity for their misuse and abuse (Lanier, 2003). The chronic overuse of NSAIDs has been related to certain adverse effects, i.e. an increased incidence of stomach ulcers, kidney failure, and liver damage. Clinicians and therapists must recognize the potential consequences of promoting NSAID use and must acknowledge their responsibility as educators against abuse (Cheung et al., 2003).

2.6.2 Nutritional Supplements

There is a limited amount of well designed scientific research available describing the use of nutritional supplements in the treatment of DOMS. In general, the treatment of DOMS using conventional antioxidants (vitamins C and E) has not showed much promise. Recent studies (Timmer, 2002; McBride, Kraemer, Triplett-McBride, and Sebastianelli, 1998; Saxton, Donnelly, and Roper, 1994; Kaminski and Boal, 1992), using whole body resistance to induce

DOMS, have found Vitamin E and Vitamin C supplementation did not significantly effect ratings of muscle soreness when compared to a placebo group.

Supplements such as Arnica, Coenzyme Q-10 and L-Carnitine have also been investigated; however this information is limited. More research is needed before a conclusive decision can be made concerning the effectiveness of these supplements in treating DOMS.

2.6.3 Therapeutic Treatments.

2.6.3.1 Cryotherapy.

Various researchers believe that cold application could be beneficial in treating DOMS due to its ability to lower tissue temperature. Lower tissue temperature decreases circulation, metabolism, muscle spasm, the extent of hypoxic injury to cells, and pain (Prentice, 2003). Because of these effects, it was believed that cryotherapy would have a preventative effect on DOMS.

However, studies (Paddon-Jones and Quigley, 1997; Clark, 1994; Isabell et al., 1992) have shown little or no reduction in the magnitude of muscle soreness or the facilitation of its recovery following eccentric activity with the application of cryotherapy. Therefore it appears that cold application, other than its temporary analgesic effect, provides little benefit in the treatment of DOMS.

2.6.3.2 Electrical Current Techniques.

Clinically, electrical current therapy has been shown to be beneficial for creating muscle contraction, stimulating sensory nerves to treat pain, and hastening the healing process (Prentice, 1994).

However, the efficacy of different electrical current stimulation techniques and treatment protocols on the treatment of DOMS and its clinical signs is not well known. Electrical stimulation following muscle-damaging activities may result in temporary analgesic effects, however the signs of DOMS are not significantly improved (Butterfield, Draper, Ricard, Myrer, Durant, and Schulthies, 1997; Denegar and Perrin, 1992; Denegar and Huff, 1988).

2.6.3.3 Hyperbaric Oxygen.

Several authors have investigated the effects of hyperbaric oxygen therapy (HBOT) to enhance recovery from DOMS. HBOT refers to the medical procedure in which individuals inspire 100% oxygen while their entire bodies are subjected to pressure greater than ambient barometric pressure at sea level (Staples, Clement, Taunton, and McKenzie, 1999). There is conflicting evidence (Mekjavic, Exner, Tesch, and Eiken, 2000; Staples et al., 1999) on the impact of HBOT in the treatment of muscle soreness and a sound rationale for its use in the treatment of DOMS has not been firmly established.

2.6.3.4 Massage.

It has been suggested (Ernst, 1998) that if massage is employed during the early stages of inflammation, the mechanical pressure might decrease neutrophil margination, thereby reducing DOMS. However, research (Hilbert, Sforzo, and Swensen, 2003; Lightfoot, Char, and McDermott, 1997; Gulick et al., 1996; Wenos, Brilla, and Morrison, 1990) has reported no significant beneficial effects of massage treatments on DOMS. These studies have shown no difference in soreness levels or force generation following massage treatment. It has been suggested that the inconsistency in research findings may be attributed to the large variety of massage techniques and massage therapists (Ernst, 1998).

2.6.3.5 Stretching.

Studies that have investigated the effect of stretching prior to, after, and before and after eccentric exercise have shown positive limited effects on DOMS (Lund, Vestergaard-Poulsen, Kanstrup, and Serjsen, 1998; Wessel and Wan, 1994; High and Howley, 1989). A sound rationale for why stretching would alleviate DOMS and its signs has not been reported.

2.6.3.6 Ultrasound.

Ultrasound is a deep-heating modality that is primarily used for elevating tissue temperature in an attempt to stimulate the repair of soft-tissue injuries and relieve pain without heating or burning the superficial tissue or structure. No convincing evidence exists supporting the use of ultrasound in the management of DOMS (Symons, Casey, Gater, and Yates, 2004; Plaskett, Tiidus, and Livingston, 1999; Craig, Bradley, Walsh, Baxter, and Allen, 1999).

2.7 COMPRESSION.

A review by Smith (1991) suggests that similarities in pain, swelling and loss of function in acute inflammation following traumatic insult and delayed onset muscle soreness are due to a general response of the body.

In times of damage, inflammation cannot be stopped and is necessary to allow the mechanisms for healing and repair to occur, however the by-products of inflammation such as swelling and pain can cause further debilitation (Prentice, 1994) and therefore need to be cared for. The use of continuous compression is advocated for the treatment of inflammation following acute injury. Compression is likely the single most important technique for controlling

the initial swelling resulting from inflammation (Prentice, 1994). The effectiveness of compression devices resides in their ability to affect pressure gradients of the disrupted environment by mechanically reducing the amount of space available for swelling (Tsang, Hertel, and Denegar, 2003).

Kraemer et al. (2001) investigated the effects of continuous compression as a therapeutic intervention in the treatment of eccentric exercise-induced muscle damage of the upper extremity. They found that a compression sleeve applied immediately following exercise and worn continuously prevented loss of elbow extension, decreased the participants' perception of soreness, reduced swelling and promoted recovery of force production in comparison to the non-sleeve group.

In contrast to other therapeutic interventions/modalities, the compression sleeve used was found to restore force-production ability and reduce soreness more rapidly following the "eccentric-exercise damage model" used. It was hypothesized that the external compression garment provided mechanical support to the damaged region, thus minimizing the loss in force-production. They suggested that by reducing the amount of movement in the exercised arm, the compression sleeve attenuated the inflammatory response thus eliminating further structural damage. This accounted for the differences in circumference measurements and soreness observed between the groups.

It was reasoned that swelling was reduced because of the increased interstitial hydrostatic pressure applied to the tissue, facilitating lymphatic drainage and reducing fluid extravasation from the capillaries. This allowed for muscle damage by-products to be cleared from the damaged area via greater venous return.

2.8 CONCLUSION

Recently, compression was found to decrease the signs and aching pain associated with DOMS in the upper extremity following eccentric based activity. However, research concerning this modality is limited. There are no reported studies that have looked at the effects of continuous compression on the prevention of DOMS and its clinical signs in the lower extremity following eccentric based activity. Therefore, continued research needs to be done to determine the effectiveness of a continuous compression protocol on the clinical signs and perception of soreness following exercise-induced muscle damage in the lower extremity. The results of this investigation provide additional information on whether DOMS and its clinical signs are affected by continuous compression following eccentric exercise. An effective method of treating DOMS and its clinical signs would accelerate the return of individuals to activity in athletic endeavours, as well as normal daily activities.

3.0 METHODS

3.1 EXPERIMENTAL DESIGN

This study examined the effect of a continuous compression treatment protocol versus a no treatment protocol over a three-day period following the induction of DOMS. The design of this study was a 2 x 4 factorial design with repeated measures. The two factors were treatment protocol(s) and time. The repeated measures involved the factor of time.

3.2 VARIABLES.

Independent variables: Treatment protocols and time

Treatment protocols:
1. Continuous Compression (CC)
2. No Treatment (N)

Time:
1. Prior to induction of DOMS
2. 24 hours after induction of DOMS
3. 48 hours after induction of DOMS
4. 72 hours after induction of DOMS

Dependent variables:	Delayed-onset muscle soreness (clinical signs and symptoms)

1. Muscle Soreness (passive)
2. Muscle Pain (active)
3. Rating of Perceived Exertion associated with the Active Muscle (RPE-AM)
4. Swelling (cm.)
5. Strength (kg.)
6. Flexion Angle(s) (degrees)
7. Extension Angle (degrees)

3.3 SUBJECTS.

Twenty healthy college aged males, aged 18-35 yrs, participated in this study. With an $\alpha = .05$, an effect size = .80 and a power of .80, a sample size of 20 was recommended (Cohen, 1988). Participation was voluntary and the subjects were recruited by verbal announcements in the University of Pittsburgh's fitness facilities and physical education classes, as well as through flyers posted throughout campus. The subjects had no prior history of musculoskeletal injury to the lower extremity, full pain-free flexion/extension range of motion of the hip and knee joints and had not been involved in any type of lower body resistance training or extensive physical activity in the past six months.

A physical activity readiness questionnaire (PAR-Q) (Appendix A) was administered to subjects prior to testing. Individuals with orthopedic, cardiovascular, and/or metabolic contraindications to exercise were excluded from participation in this study. The study received University of Pittsburgh Institutional Review Board approval prior to data collection. Each subject signed an informed consent to participate (Appendix B) after the rationale underlying the experiment, as well as, its risks and benefits were explained.

A detailed exercise history questionnaire (Appendix C) was administered before testing in order to obtain physical activity information, including normal exercise frequency, duration, and intensity.

3.4 PROTOCOL.

3.4.1 Day O (Orientation):

Subjects participated in an orientation session 7 days prior to the first experimental trial. Height, weight, and percent body fat were determined during this session. Body height (cm.) and weight (kg.) were measured using a physician scale (Detecto Scales Inc., Brooklyn, NY). Bioelectric impedance analysis (Tanita Body Fat Analyzer, Tanita Inc., Skokie, IL) was used to estimate percent body fat.

Each subject then performed a one repetition maximum (1-RM) test for knee extension in the Trees Hall Health/Fitness Facility, University of Pittsburgh, PA. A 1- RM is the maximum amount of weight that can be lifted for one repetition (Baumgartner and Jackson, 1999). The initial warm-up prior to the assessment of the 1-RM consisted of lower body static stretching followed by 2 minutes of riding a Monark Cycle Ergometer (Monark Exercise Ab, Vansbro, Sweden) with low resistance (0.5 kg) at 50 revolutions ● $minute^{1}$.

After a brief review regarding the proper use of the knee extension exercise machine (Cybex International, Inc.) (Appendix D), the subject performed several practice repetitions with minimal resistance (lowest weight on machine) to familiarize himself with the exercise motion. Next, the subject was asked to estimate his 1-RM for the leg extension exercise. The subject

then performed 8 to 10 repetitions of the weight calculated to be 50% of his estimated 1-RM as a warm-up for the active muscles.

One repetition was considered to be the full extension of the knee from approximately 90° to 180°. As the subject returned the knee to a flexed position, the researcher controlled the weight, so that the quadriceps femoris muscle group was doing no eccentric work. Weight was progressively increased by 5 or 10 pounds ● attempt^{-1} until the subject was unable to perform one lift using proper form and technique. The subject performed 1 repetition with each attempt, and the last successfully completed attempt was recorded as the 1-RM weight (Baumgartner and Jackson, 1999). The subject rested for 1 minute between trials to prevent premature fatigue. A ten repetition maximum (10-RM - the weight that subjects can successfully lift 10 times) was estimated as 73.5% of the 1 RM (Baechle, 1994). This weight was used to induce DOMS.

EXPERIMENTAL TRIALS.

Orientation → 7 Days → Experimental Trials→ 24 Hours→ 48 Hours→ 72 Hours			
Day O	**Day 1**	**Day 2**	**Day 3**	**Day 4**
a. Questionnaires	**a. Baseline Measures**	**a. Muscle Soreness (passive)**
b. Height	**b. Induction of DOMS**	**b. Muscle Pain (active)**
c. Weight		**c. RPE-AM**
d. Percent Body Fat		**d. Swelling**
e. 1-RM		**e. Flexion Angle**
		f. Extension Angle
		g. Strength

*****Subjects in the (CC) group applied the compression garment immediately following Day 1*****

Figure 1. Experimental Protocol

3.4.2 Day 1:

The experimental trials were performed between 8 a.m. and noon. Subjects were matched according to 1-RM results and then randomly assigned to one of the treatment protocol groups (independent variables): continuous compression (CC) or no treatment (N). Thigh circumference and knee flexion and extension angle measurements were conducted prior to DOMS inducement in order to obtain baseline values. Subjects were also given an explanation and instructions on the proper use of the scales for measuring passive muscle soreness (Appendix E), active muscle pain (Appendix F) and RPE-AM (Appendix G).

If assigned to the continuous compression group, the subject were also fitted with a compression garment (McDavid Sports Medicine, IL) and instructed as to its proper application (Appendix H).

3.5 INDUCTION OF DOMS

DOMS was induced to the quadriceps muscle group of the non-dominant leg of each subject. The non-dominant leg was determined by having the subject kick a ball. If the subject kicked with the right foot, the left leg was considered the non-dominant leg and vice versa. The induction of DOMS, as described by Timmer (2002) and Dixon (2002), was accomplished by repetitive eccentric/concentric contractions of the quadriceps muscle group.

The subjects were seated on the exercise machine with their arms stabilized by grasping the handles located adjacent to each thigh. The subject then performed a warm-up of 10

repetitions of the weight calculated to be 50% of their 1-RM.– (muscle soreness, muscle pain and RPE-AM measurements were measured with rating scales).

SYMPTOM MEASUREMENT TIMELINE.

Muscle Soreness – was measured passively before warm-up repetitions (relaxed state).

Muscle Pain – was measured actively at the midpoint and endpoint of the warm-up repetitions.

RPE - AM - was measured at the midpoint and immediately after the last warm-up repetition.

Figure 2. Timeline of Symptom Measurement During Experimental Trials

Immediately following the warm-up repetitions, each subject raised 73.5% of the calculated 1RM from full knee flexion (approximately 90°) to full knee extension (approximately 180°). All calculated resistances were rounded to the nearest pound. Each repetition was completed in 3 seconds. The count was kept by a metronome (Franz Manufacturing Company, Inc) set at a cadence of 60 beats per minute. Subjects performed a concentric contraction for one second, followed by a two second eccentric contraction. The goal of including a longer eccentric contraction phase was to produce a higher level of DOMS (Clarkson and Sayers, 1999). Subjects performed three sets of the knee extension/flexion repetitions. The subjects performed the first set of ten repetitions of knee exercises at their predetermined 10 RM followed by a 90 second rest period. The resistance of the second and third sets stayed the same as the first set with the subject completing as many repetitions as possible as determined by correct form and technique. Subjects had a 2-minute rest period between the second and third sets.

Immediately following the muscle soreness inducing protocol, the subjects in the (CC) group were instructed to put on the compression garment. All subjects were told to limit water temperature and duration when bathing the exercised leg (i.e. no whirlpool or leg soaking, etc.) and to refrain from taking any pain medication/using any pain-relieving modalities (i.e. ice, heat, massage, electrical stimulation, ultrasound, etc.) for the duration of the study. The subjects were also told to refrain from any lower body resistance training or stretching techniques (including elevation of the lower extremities) for the length of the study. Subjects were encouraged to maintain their normal diet throughout the length of the study and avoid consuming large quantities of Vitamin C, Vitamin E and caffeine.

3.6 MEASUREMENT OF DEPENDENT VARIABLES

The dependent variables passive muscle soreness, active muscle pain, RPE-AM, swelling, flexion angle, extension angle and strength were measured prior to and at 24, 48, and 72 hours following the induction of DOMS. The same investigator performed all measurements.

3.6.1 Muscle Soreness/Muscle Pain/RPE-AM.

The intensity and unpleasantness of muscle soreness, muscle pain and rating of perceived exertion in the active muscle(s) was measured during the warm-up repetitions preceding the 1-RM testing sessions using rating scales - explanations and instructions on the proper use of the rating scales were reinforced prior to each testing session.

Passive muscle soreness was obtained using a rating scale modified from Cook (1997). This 10-inch visual analog scale (Appendix E) utilizes verbal anchors "no soreness" on the left to "worst possible soreness" on the right. The subjects drew a line on the scale that corresponded to their perception of muscle soreness. The position of the line was measured starting from 0 to the nearest inch. The measure of quadriceps muscle soreness was obtained while the subject is in a relaxed state seated on the exercise machine with the knee extended.

Active muscle pain was measured by a category rating scale (Cook, O'Connor, Eubanks, Smith, and Lee, 1997) (Appendix F), which utilizes anchors 0 (no pain at all) and 10 (extremely intense pain-almost unbearable). If the subject felt no pain in the quadriceps, he was instructed to respond with the number 0. If the muscle pain was greater than 10, the subject was instructed to respond with the number that represented the pain intensity felt in relation to 10. Measures of active muscle pain occurred at the midpoint and immediately following the warm-up repetitions.

Rating of perceived exertion associated with the active skeletal muscles was measured by a rating scale developed by Robertson et al., (Appendix G), which allows subjects to subjectively rate their feelings during exercise. This scale utilizes anchors (0) Extremely Easy and (10) Extremely Hard that are associated with levels of exertion. Subjects were asked to select the number that corresponded to the RPE-AM at the midpoint and following the last warm-up repetition.

3.6.2 Swelling.

Circumference measurements were obtained with a spring-loaded flexible tape measure (Gulick) at two sites (mid-thigh, distal thigh) while the subject was seated with hip flexed and the knee extended. For each site, three measurements were taken (cm) with the largest used for analysis.

Tension to the tape was applied so that it snugly fit around the portion of thigh being measured but did not indent the skin or compress the subcutaneous tissue. Measurements occurred in a horizontal plane, parallel to the floor.

Mid-Thigh – the tape was applied midway between the inguinal crease and proximal border of the patella.

Distal Thigh – the tape was applied midway between the mid-thigh and femoral condyles.

Ink marks were placed as reference points to insure reproduction of proper circumference alignment for each measurement period.

3.6.3 Flexion Angle.

Flexion angles were measured according to techniques described by Norkin and White (1995) with a standard goniometer with 1° markings. Flexion angle measurement occurred in two testing positions.

For the first measure of knee flexion, the subject was placed in a supine position with a towel placed under the ankle to allow the knee to fully extend. The investigator's right hand moved the subject's non-dominant thigh to approximately 90° degrees of hip flexion and then stabilized the femur to prevent further flexion. The investigator's left hand guided the subject's lower leg through full knee flexion ROM. The fulcrum of the goniometer was centered over the lateral epicondyle of the femur. The proximal arm was aligned with the lateral midline of the femur, using the greater trochanter for reference. The distal arm was aligned with the lateral midline of the fibula, using the lateral malleolus and fibular head as reference points. Ink marks

were placed at these points of reference to insure reproduction of proper goniometer alignment for each measurement period.

For the second measure of knee flexion, the subject was placed in a prone position, with the hip in 0° of abduction, adduction, flexion, extension and rotation. A towel was placed under the thigh to allow the knee to fully extend. At the end of the knee flexion ROM, the examiner's hand aligned the proximal goniometer arm with the lateral midline of the subject's femur, using the greater trochanter as a reference point. The examiner used his other hand to maintain knee flexion and to keep the distal goniometer arm aligned along the fibular head and lateral malleolus of the lower leg. The fulcrum of the goniometer was centered over the lateral epicondyle of the femur. Ink marks were placed at these points of reference to insure reproduction of proper goniometer alignment for each measurement period.

An intraclass correlation of ICC = .99 was previously reported by other researchers for these measurements (Watkins, Riddle, Lamb, and Personius, 1991). Three individual measurements of flexion angle were taken for each position. The three-flexion angle measurements were averaged so there was one recorded measurement of flexion angle for each position per measurement period.

3.6.4 Extension Angle.

Extension angle was measured with a standard goniometer with 1° markings. For the measurement of the knee extension angle, subjects were placed in a supine position with a towel placed under the ankle to allow the knee to fully extend. The investigator stabilized the femur to prevent rotation, abduction, and adduction of the hip. The fulcrum of the goniometer was centered over the lateral epicondyle of the femur. The proximal arm was aligned with the lateral

midline of the femur, using the greater trochanter for reference. The distal arm was aligned with the lateral midline of the fibula, using the lateral malleolus and fibular head as reference points. Ink marks were placed at these points of reference to insure reproduction of proper goniometer alignment for each measurement period.

An intraclass correlation of ICC = .98 was previously reported by other researchers for this measurement (Watkins et al. 1991). Three individual measurements of extension angle were taken. The three extension angle measurements were averaged so there was one recorded measurement of flexion angle per measurement period.

3.6.5 Strength.

Muscular strength values were obtained (Orientation, Days 2, 3 and 4) by measuring the maximal weight subjects could lift for 1 repetition. The subject performed 10 repetitions of knee extension of the weight calculated to be 50% of the subject's estimated 1-RM as determined during the orientation session as a warm-up. As the subject returned the knee to a flexed position, the researcher controlled the weight, so that the quadriceps femoris muscle group was doing no eccentric work. The weight was progressively increased by 5 or 10 pounds ● $attempt^{-1}$ until the subject was unable to perform one knee extension repetition using proper form and technique. The subject performed 1 repetition with each attempt, and the last successfully completed attempt was recorded as the 1-RM weight.

3.7 DAYS 2 (24-HOURS), 3(48-HOURS) AND 4 (72-HOURS) – POST EXERCISE:

Subjects reported at approximately the same time as Day 1. Measurements of the dependent variables were repeated and recorded. The measurements for both experimental groups were conducted with the leg musculature exposed (compression garment removed).

3.8 TREATMENT PROTOCOLS

3.8.1 Continuous compression.

The (CC) group wore a compression garment (McDavid Sports Medicine, IL) continuously during the 3-day recovery period. The thigh support was a 1/8" thick neoprene support sleeve with nylon on both sides. The compression sleeve was removed only for bathing/daily washing and for study measurements. The pressure garment for each subject in the (CC) group was individually fitted according to the manufacturer's specifications for thigh compression. Subjects were required to report at 24, 48, and 72-hour post exercise for measurement of the dependent variables.

3.8.2 Control.

The subjects in the control group did not receive any treatment. They were also required to report at 24, 48, and 72-hour post exercise for measurement of the dependent variables.

3.9 STATISTICAL ANALYSIS

The data for each of the dependent variables of passive muscle soreness, active muscle pain, RPE-AM, swelling, flexion angle, extension angle and strength was analyzed with a two factor (treatment x time) repeated measures analysis of variance (ANOVA) with repeated measures over the second factor (time). A multiple comparison contrast procedure was used for all *post hoc* analysis of significant main effects. Level of statistical significance for all statistical analyses (ANOVA and multiple comparison contrast analyses) was chosen *a priori* as $P < .05$.

4.0 RESULTS

The purpose of this study was to determine the effects of a continuous compression treatment protocol on the clinical signs and symptoms associated with exercise-induced muscle damage following eccentric exercise.

4.1 SUBJECT CHARACTERISTICS

Twenty healthy college-aged males, ten continuous compression (CC) and ten no treatment (N), participated in this investigation. The CC and N subjects did not differ in age, height, weight, and body fat %. (Table 1).

Table 1. Subject Descriptive Data

Characteristic	N	CC	t	p
Age (yr)	22.2 ± 4.6	22.4 ± 4.4	.099	.922
Height (cm)	181.6 ± 8.9	181.3 ± 7.8	- .085	.933
Weight (kg)	80.4 ± 12.6	91.6 ± 21.1	1.436	.168
Body Fat (%)	18.8 ± 8.4	20.1 ± 9.7	.432	.671

Values are means ± SD; N = 10 in each group
N = no treatment, CC = continuous compression

4.2 PASSIVE MUSCLE SORENESS

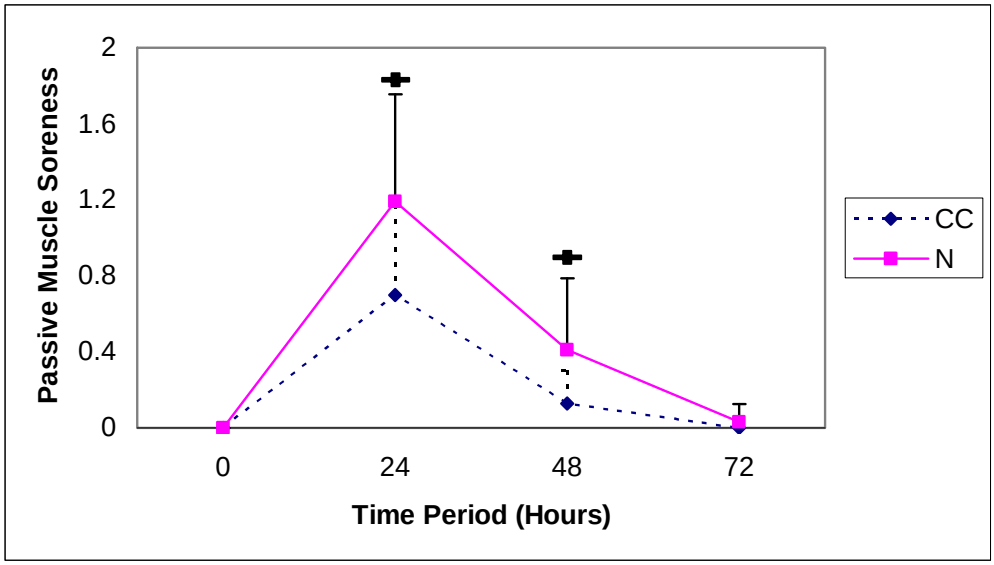
Table 2 contains the means for passive muscle soreness as a function of treatment time and protocol. Table 3 contains a repeated measures ANOVA summary table for passive muscle soreness. There were significant main effects for treatment protocol, $F(1,18) = 5.31$, $p<.05$, time $F(3,54) = 55.27$, $p<.05$, and treatment protocol by time $F(3, 54) = 3.75$ (Figure 3).

Table 2. Passive Muscle Soreness for Compression and No Treatment Subjects at Four

Time		Compression		Control		Marginal
		M	SD	M	SD	M
1	Orientation	.00	.00	.00	.00	.00
2	24-Hours	.70	.48	1.19	.57	.95
3	48-Hours	.13	.17	.41	.38	.27
4	72-Hours	.00	.00	.03	.01	.02
Marginal		.21		.41		.31

Table 3. Repeated Measures ANOVA Summary for Passive Muscle Soreness.

Source	df	MS	F	p
Between Subjects				
Protocol	1	.80	5.31	.03
Error	18	.15		
Within Subjects				
Time	3	3.92	55.27	.00
Time X Protocol	3	.27	3.75	.02
Error	54	.07		



(Mean + SD; N = 10). + indicates P < .05 between CC and N groups.

Figure 3. Passive Muscle Soreness over a Four Day Time Period.

4.3 ACTIVE MUSCLE PAIN

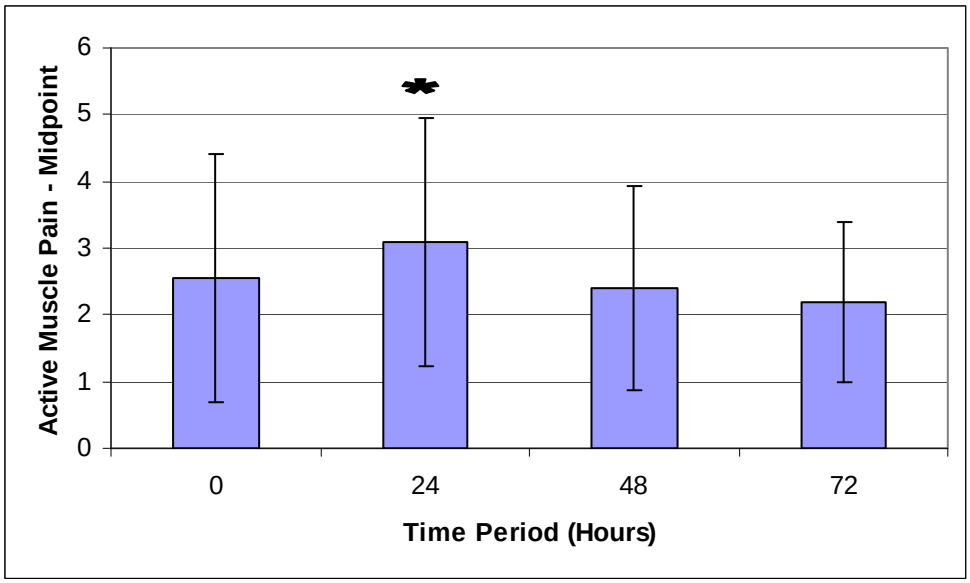
Table 4 contains the means for active muscle pain taken at the midpoint of the one-repetition maximum warm-up repetitions. Table 5 contains a repeated measures ANOVA summary table for active muscle pain taken at the midpoint of the one-repetition maximum warm-up repetitions. There was a significant main effect for time $F(3,54) = 3.187$, $p<.05$ (Figure 4). Table 6 contains the means for active muscle pain taken immediately following the one-repetition maximum warm-up repetitions. Table 7 contains a repeated measures ANOVA summary table for active muscle pain taken immediately following the one-repetition maximum warm-up repetitions. There were no significant differences in active muscle pain between the CC and N groups for any assessment period.

Table 4. Active Muscle Pain Measured at the Midpoint of the Warm-Up Repetitions

Time		Compression		Control		Marginal
		M	SD	M	SD	M
1	Orientation	2.80	1.75	2.30	2.00	2.55
2	24-Hours	3.20	1.62	3.00	2.16	3.10
3	48-Hours	1.90	1.37	2.90	1.60	2.40
4	72-Hours	2.10	1.10	2.30	1.34	2.20
Marginal		2.50		2.63		2.56

Table 5. Repeated Measures ANOVA Summary for Active Muscle Pain Measured at the Midpoint of the Warm-Up Repetitions.

Source	df	MS	F	p
Between Subjects				
Protocol	1	.31	.04	.85
Error	18	8.09		
Within Subjects				
Time	3	2.98	3.19	.03
Time X Protocol	3	2.11	2.26	.09
Error	54	.94		



(Mean + SD; N = 20). * indicates P < .05 from corresponding pre-exercise value.

Figure 4. Active Muscle Pain at the Midpoint of the Warm-Up Repetitions over a Four Day Time Period for All Subjects.

Table 6. Active Muscle Pain Measured Immediately Following the Warm-Up Repetitions.

Time		Compression		Control		Marginal
		M	SD	M	SD	M
1	Orientation	3.70	1.77	3.20	1.93	3.45
2	24-Hours	3.90	2.08	4.10	2.33	4.00
3	48-Hours	3.10	1.52	3.80	1.62	3.45
4	72-Hours	3.00	1.49	3.50	1.90	3.25
Marginal		3.43		3.65		3.54

Table 7. Repeated Measures ANOVA Summary for Active Muscle Pain Measured Immediately Following the Warm-Up Repetitions.

Source	df	MS	F	p
Between Subjects				
Protocol	1	1.01	.10	.75
Error	18	10.01		
Within Subjects				
Time	3	2.08	1.69	.18
Time X Protocol	3	1.38	1.12	.35
Error	54	1.23		

4.4 RATING OF PERCEIVED EXERTION ASSOCIATED WITH THE ACTIVE MUSCLE (RPE-AM)

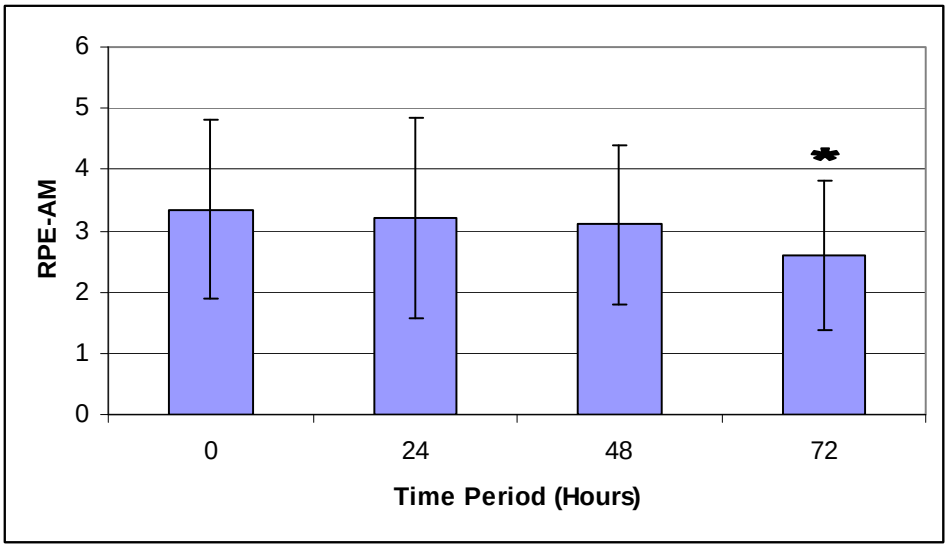
Table 8 contains the means for Rating of Perceived Exertion associated with the Active Muscle taken at the midpoint of one repetition maximum warm-up repetitions. Table 9 contains a repeated measures ANOVA summary table for RPE-AM at the midpoint of the one repetition maximum warm-up repetitions. A significant time main effect was observed $F(3,54) = 3.428$ (Figure 5). Table 10 contains the means for RPE-AM taken during the final one repetition maximum warm-up repetition. Table 11 contains a repeated measures ANOVA summary table for RPE-AM taken during the final one repetition maximum warm-up repetition. No significant main effects or interaction were noted.

Table 8. RPE-AM taken at the Midpoint of the One Repetition Maximum Warm-Up Repetitions.

Time		Compression		Control		Marginal
		M	SD	M	SD	M
1	Orientation	3.70	1.64	3.00	1.25	3.35
2	24-Hours	3.30	1.89	3.10	1.45	3.20
3	48-Hours	3.10	1.37	3.10	1.29	3.10
4	72-Hours	2.60	0.97	2.60	1.51	2.60
Marginal		3.18		2.95		3.06

Table 9. Repeated Measures ANOVA for Rating of Perceived Exertion Associated with the Active Muscle at the Midpoint of the Warm-Up Repetitions

Source	df	MS	F	p
Between Subjects				
Protocol	1	1.01	.16	.70
Error	18	6.47		
Within Subjects				
Time	3	2.11	3.43	.02
Time X Protocol	3	.55	.89	.45
Error	54	.62		



(Mean ± SD; N = 20) * indicates $P < .05$ from previous day.

Figure 5. Rating of Perceived Exertion Associated with the Active Muscle at the Midpoint of the Warm-Up Repetitions for All Subjects.

Table 10. RPE-AM taken during the Final Warm-Up Repetition.

Time		Compression		Control		Marginal
		M	SD	M	SD	M
1	Orientation	4.80	1.87	4.00	1.83	4.40
2	24-Hours	4.30	1.89	4.10	2.08	4.20
3	48-Hours	3.80	1.69	4.30	1.70	4.05
4	72-Hours	3.60	1.35	3.50	1.90	3.55
Marginal		4.13		3.98		4.05

Table 11. Repeated Measures ANOVA for Rating of Perceived Exertion Associated with the Active Muscle(s) taken during the Final Warm-Up Repetition.

Source	df	MS	F	p
Between Subjects				
Protocol	1	.45	.05	.83
Error	18	9.63		
Within Subjects				
Time	3	2.63	2.38	.80
Time X Protocol	3	1.42	1.28	.29

4.5 CIRCUMFERENCE MEASURES

Circumference of the midthigh is presented in Table 12. Table 13 contains a repeated measures ANOVA summary table for midthigh circumference. No significant main effects or interaction were noted. The circumference of the distal thigh is presented in Table 14. Table 15 contains a repeated measures ANOVA summary table for distal thigh circumference measures. A significant time main effect for distal thigh circumference was observed F (3,54) = 4.538 (Figure 6).

Table 12. Midthigh Circumference Measures for Compression and No Treatment Subjects at Four Time Points.

Time		Compression		Control		Marginal
		M (cm)	SD	M (cm)	SD	M
1	Orientation	54.68	5.85	51.82	3.80	53.25
2	24-Hours	54.74	5.90	51.96	3.76	53.35
3	48-Hours	54.60	5.84	51.88	3.85	53.24
4	72-Hours	54.64	5.82	51.84	3.85	53.24
Marginal		54.67		51.88		53.27

Table 13. Repeated Measures ANOVA Summary for Midthigh Circumference.

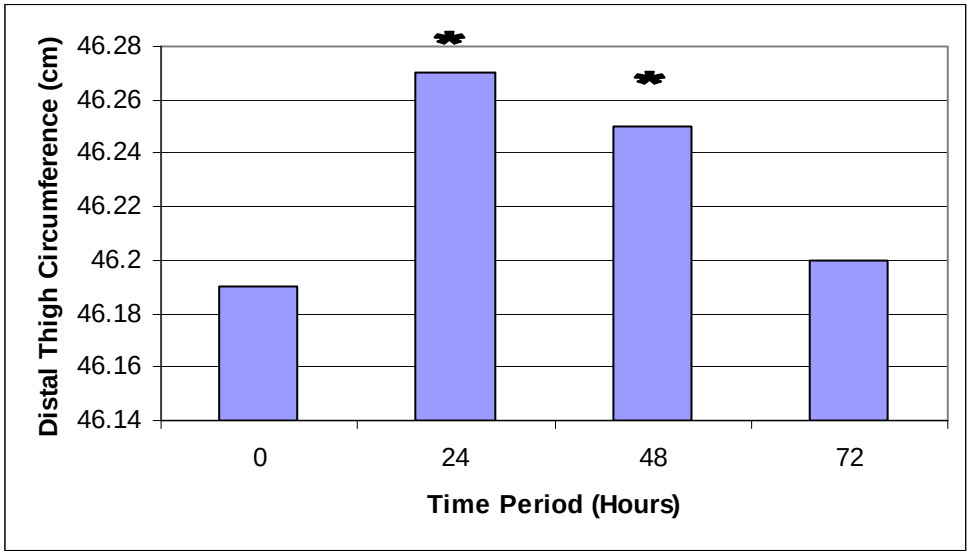
Source	df	MS	F	p
Between Subjects				
Protocol	1	155.68	1.60	.22
Error	18	97.51		
Within Subjects				
Time	3	.06	2.07	.12
Time X Protocol	3	.02	.60	.62
Error	54	.03		

Table 14. Distal Thigh Circumference Measures for Compression and No Treatment Subjects at Four Time Points.

Time		Compression		Control		Marginal
		M (cm)	SD	M (cm)	SD	M
1	Orientation	47.40	4.870	44.98	3.34	46.19
2	24-Hours	47.46	4.813	45.08	3.40	46.27
3	48-Hours	47.44	4.839	45.06	3.29	46.25
4	72-Hours	47.40	4.869	45.00	3.31	46.20
Marginal		47.43		45.03		46.23

Table 15. Repeated Measures ANOVA Summary for Distal Thigh Circumference.

Source	df	MS	F	p
Between Subjects				
Protocol	1	114.72	1.66	.21
Error	18	17.31		
Within Subjects				
Time	3	.03	4.54	.01
Time X Protocol	3	.00	.28	.84
Error	54	.01		



(Mean; N = 20). * indicates $P < .05$ from corresponding pre-exercise value.

Figure 6. Distal Thigh Circumference over a Four Day Time Period for All Subjects.

4.6 STRENGTH (ONE REPETITION MAXIMUM)

The results of muscle strength assessment are presented in Table 16. Table 17 contains a repeated measures ANOVA summary table for muscular strength. No significant main effects were found for one repetition maximum.

Table 16. Strength Measures for Compression and No Treatment Subjects at Four Time Points.

Time	Compression		Control		Marginal
	M (kg)	SD	M (kg)	SD	M
1 Orientation	53.18	9.83	50.91	11.55	52.05
2 24-Hours	54.36	12.50	48.41	8.97	51.39
3 48-Hours	54.55	10.05	50.68	10.17	52.62
4 72-Hours	55.00	10.38	52.05	10.79	53.53
Marginal	54.27		50.51		52.40

Table 17. Repeated Measures ANOVA Summary for Strength Measurement Comparing Compression and No Treatment Subjects at Four Time Points.

Source	df	MS	F	p
Between Subjects				
Protocol	1	282.79	.66	.43
Error	18	3.66		
Within Subjects				
Time	3	16.44	2.26	.09
Time X Protocol	3	12.78	1.76	.17
Error	54	7.27		

4.7 FLEXION ANGLES (PRONE AND SUPINE)

The mean results and a repeated measures ANOVA for knee flexion angle assessment measured in a prone position are presented in Tables 18 and 19, respectively. Table 20 contains the means for knee flexion angle measured in a supine position. Table 21 contains a repeated measures ANOVA summary table for flexion angle taken in a supine position. A significant time main effect was noted $F(3,54) = 6.35$ (Figure 7) for supine knee flexion angle.

Table 18. Prone Knee Flexion Angle Measures.

Time		Compression		Control		Marginal
		M (°)	SD	M (°)	SD	M
1	Orientation	121.10	6.05	125.80	3.88	123.45
2	24-Hours	121.10	6.01	123.80	3.80	122.45
3	48-Hours	121.20	6.11	124.80	4.42	123.00
4	72-Hours	121.70	7.89	124.90	3.93	123.30
Marginal		121.28		124.83		123.05

Table 19. Repeated Measures ANOVA Summary for Prone Knee Flexion Angle.

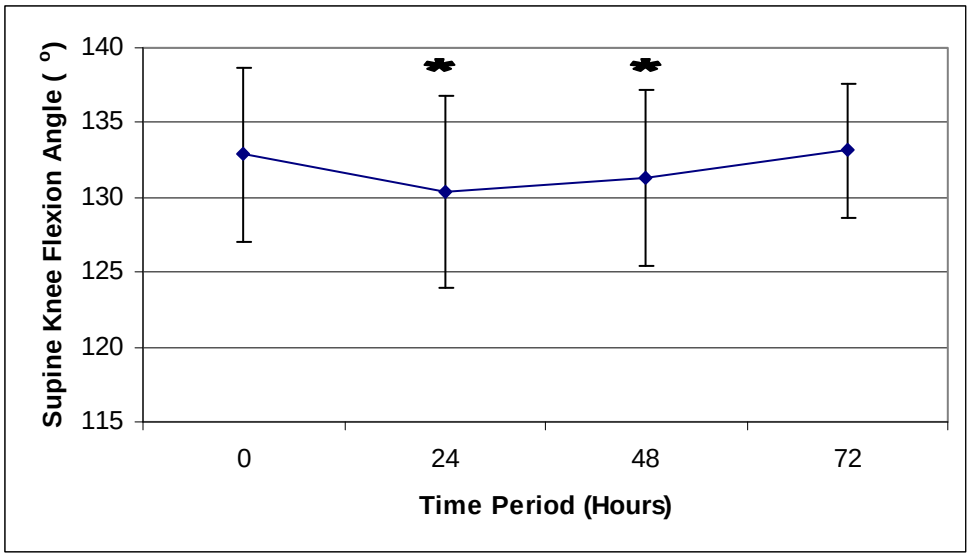
Source	df	MS	F	p
Between Subjects				
Protocol	1	252.05	2.71	.12
Error	18	93.10		
Within Subjects				
Time	3	3.90	.46	.71
Time X Protocol	3	3.62	.43	.73
Error	54	8.40		

Table 20. Supine Knee Flexion Angle Measures.

Time		Compression		Control		Marginal
		M (°)	SD	M (°)	SD	M
1	Orientation	130.80	6.58	135.00	4.30	132.90
2	24-Hours	128.40	7.38	132.40	4.77	130.40
3	48-Hours	129.50	7.25	133.10	3.73	131.30
4	72-Hours	132.00	4.32	134.30	4.57	133.15
Marginal		130.18		133.70		131.94

Table 21. Repeated Measures ANOVA Summary for Supine Knee Flexion Angle.

Source	df	MS	F	p
Between Subjects				
Protocol	1	248.51	2.34	.14
Error	18	106.16		
Within Subjects				
Time	3	34.47	6.35	.00
Time X Protocol	3	3.65	.67	.57
Error	54	5.43		



(Mean ± SD; N = 20). * indicates $P < .05$ from corresponding pre-exercise value.

Figure 7. Supine Knee Flexion Angle over a Four Day Time Period for All Subjects.

4.8 EXTENSION ANGLE

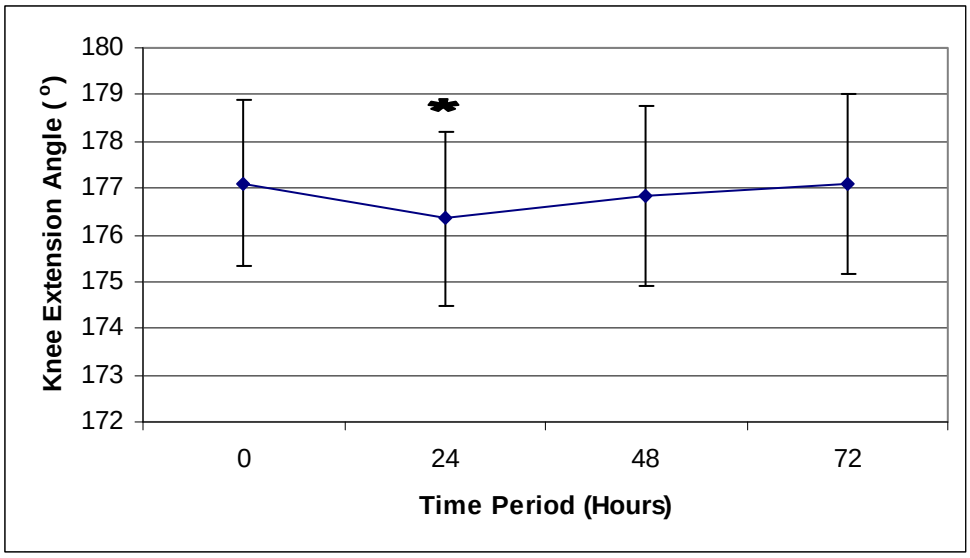
Table 22 contains the results of the assessment of knee extension in a supine position. Table 23 contains a repeated measures ANOVA summary table for knee extension angle. A significant time main effect was noted F(3,54) = 3.065 (Figure 8).

Table 22. Extension Angle Measures.

Time		Compression		Control		Marginal
		M (°)	SD	M (°)	SD	M
1	Orientation	177.40	1.65	176.80	1.93	177.10
2	24-Hours	177.00	1.89	175.70	1.70	176.35
3	48-Hours	177.40	1.71	176.30	2.06	176.85
4	72-Hours	177.50	2.07	176.70	1.77	177.10
Marginal		177.33		176.38		176.85

Table 23. Repeated Measures ANOVA Summary for Extension Angle Measures.

Source	df	MS	F	p
Between Subjects				
Protocol	1	18.05	1.60	.22
Error	18	11.29		
Within Subjects				
Time	3	2.50	3.07	.04
Time X Protocol	3	.48	.59	.62
Error	54	.82		



(Mean ± SD; N = 20). * indicates $P < .05$ from corresponding pre-exercise value.

Figure 8. Knee Extension Angle over a Four Day Time Period for All Subjects.

5.0 DISCUSSION

The primary purpose of this investigation was to determine the effect of continuous compression on the clinical signs and symptoms associated with delayed onset muscle soreness (DOMS) following lower extremity eccentric activity. It was hypothesized that when compared to the no treatment group (N) at 24, 48, and 72 hours following a bout of eccentric exercise designed to induce DOMS, the subjects in the continuous compression (CC) group would display the following:

a. n overall lower perception of passive muscle soreness.

b. an overall lower rating of perceived exertion associated with the active muscle(s).

c. an overall lower rating of active muscle pain.

d. a decreased amount of swelling.

e. greater extension angles.

f. greater flexion angles.

g. greater strength.

The primary findings of this investigation were that continuous compression resulted in a significant difference in the overall perception of passive muscle soreness across time at 24 and 48-hours post exercise. Furthermore, it was noted that none of the other dependent variables

were differentially affected by the treatment protocol when measured at baseline and following induction of DOMS. As such, these findings did not support the experimental hypotheses. In addition, regardless of treatment protocol, there were significant differences across time observed in active muscle pain, rating of perceived exertion associated with the active muscle, distal thigh circumference, supine knee flexion angle, and knee extension angle following the induction of DOMS. The differences observed across time for these independent variables will be discussed. Similar to the findings of Kraemer et al. (2001), the time course of development and the pattern of the clinical signs and symptoms noted presently differed, depending on the measurement. This development and time course of change might be related to the underlying fluid dynamics of the soft tissue injury and recovery (Kraemer et al., 2001). However, the presence of a significant difference in these signs and symptoms verifies the effectiveness of the protocol used to induce DOMS (Hart, Swanik, and Tierney, 2005; Gulick et al., 1996).

5.1 PASSIVE MUSCLE SORENESS

The present finding that continuous compression applied immediately following eccentric exercise decreases passive muscle soreness at 24 and 48-hours following the induction of DOMS is consistent with previous research (Kraemer et al., 2001). It must be noted, however, that this previous investigation involved the application of continuous compression following upper extremity eccentric activity, as opposed to the compression applied to the lower extremity in the current investigation. Consistent with the present findings, Kraemer et al. (2001), reported that subjects in both groups (i.e. continuous compression and control) reported muscle soreness for 48 hours after the exercise session. In the present investigation, the continuous compression

group reported significantly less passive muscle soreness when compared to the non-compression treatment group for 48 hours after the DOMS inducing protocol, with pain peaking in both groups 24-hours post exercise. In a clinical setting, this reduction in soreness from the application of compression may be beneficial in the rehabilitation process and thus worthy of repeated use (Clark, 1994).

The finding that muscle soreness peaked the day following eccentric exercise is consistent with literature that reports that the soreness experienced following unaccustomed eccentric activity is initially felt 8-24 hours post-exercise, with peak levels occurring around 24-72 hours. It is likely that the soreness resulted from the chemical stimulation of the sensory nerve endings of the involved muscular structures from the swelling and pressure in the muscle (Clarkson et al., 2002; Newham et al., 1987; Hough, 1902). During excessive eccentric muscle contractions, muscular structures (i.e. sarcomeres) become overstretched and disrupted, which results in an inflammatory response due to the breakdown of muscle fibers and membrane damage (Trennell, Rooney, Sue, and Thompson, 2006; Proske and Allen, 2005). During the acute stage of the inflammatory response, the synthesis of prostaglandin E2 (PGE2) and the formation of bradykinin stimulate free nerve endings which lead to the uncomfortable sensation. Other chemical mediators including Substance P, which is released from free nerve endings, also add to the discomfort experienced (Denegar, Saliba, and Saliba. 2006).

5.2 ACTIVE MUSCLE PAIN

Voluntary eccentric muscle contraction has been described as one of the most effective strategies for diminishing the pain experienced during DOMS (Symons et al., 2004); possibly due to an

increased removal of noxious waste products through an increased blood flow or an increased endorphin release during the exercise activity

(Armstrong, 1984; Kelly, 1982). Endorphins are part of a complex endogenous opioid system. They are produced and secreted within the central nervous system with the overall effect of decreasing pain (Denegar et al., 2006; Donley and Denegar, 1994; Armstrong, 1984). During exercise, endorphin secretion is increased (Kelly, 1982), which is potentially responsible for the temporary pain relief experienced (Smith, 1992; Armstrong, 1984).

The present finding that active muscle pain peaked at 24-hours following the one-repetition maximum warm-up repetitions regardless of the treatment protocol is evidence that the pain followed the sequence of previously reported studies. However, in the present investigation, the muscular contractions performed by the subjects during subsequent testing sessions may have been responsible for the analgesic effect that was experienced by the subjects at 48 and 72 hours post-exercise. This analgesic effect may have been responsible for the reduction in active muscle pain experienced by the subjects.

5.3 RATING OF PERCEIVED EXERTION ASSOCIATED WITH THE ACTIVE MUSCLE

Perceived exertion can be defined as a measure of the intensity of effort that is felt during exercise (Noble and Robertson, 1996). In the present investigation, a significant main effect was only noted for time when examining the subjects' feeling of strain in the exercising quadriceps muscles over the four-day time period. It is unclear as to why RPE-AM measured at the midpoint of the warm-up repetitions was significantly lower at 72-hours when compared to the

previous day. However, it is possible that the subjects described their overall rating of perceived exertion instead of the RPE specific to the active muscles. RPE-AM measures (exertional ratings arising from the active muscle reflecting peripheral perceptual signals) have been shown (Robertson, Goss, Rutkowski, Lenz, Dixon, Timmer, Dube, and Andreacci, 2003) to be more intense than RPE-O (exertional ratings reflecting the sensations of the cardiopulmonary system) following a resistance exercise protocol. RPEs associated with the active muscle (arm, legs, chest) provide more precise information about anatomically recognized perceived exertion; therefore making them useful in guiding rehabilitation for neuromuscular, articular, and pain disorders (Robertson, 2004). Alternately subjects may have become accustomed to the short time exercise movement and as such, responded with a lower RPE-AM.

5.4 SWELLING

A significant increase in distal thigh circumference was observed following the induction of DOMS across subjects regardless of treatment protocol. During the inflammatory process, chemical mediators cause an opening of the capillary beds (vasodilation), which causes the walls of the capillaries to become more permeable allowing chemical mediators to leak out (i.e. plasma proteins). These proteins exert an osmotic pressure, which causes an increase in proteins in the interstitium. The increase in proteins in the interstitium and the osmotic pressure the proteins exert are responsible for the swelling that is experienced during the inflammatory response (Denegar et al., 2006). External compression promotes rest of the injured tissue and can potentially limit the amount of available space for swelling to accumulate (Brennan and Miller, 1998). In the present investigation, the compression garment was fitted according to the

manufacturer's specifications by measuring around the largest circumference of the thigh (Appendix H). The lengths of the femur and involved surrounding tissues were not considered in this sizing measurement. It is possible that the compression garment did not provide sufficient counterforce at the distal thigh region to effectively limit swelling.

The greatest increase in distal thigh circumference followed the same time course as the soreness experienced by the subjects 24-hours post exercise. It stands to reason that the soreness and swelling of the distal thigh encountered following eccentric activity would follow the same time course. The swelling in the muscle fibers activates free nerve endings in the muscle, contributing to the sensation of discomfort (Clarkson and Hubal, 2002). It is common for the pressure in the muscle exerted by the swelling to lead to a localization of soreness, which can be attributed to a high concentration of muscle pain receptors in the connective tissue at the myotendinous region (Newham, 1982). In the current study, the myotendinous region was located at the distal thigh.

5.5 STRENGTH

The relationship between the development of DOMS and the loss of muscle strength has yet to be clearly established (Lieber and Friden, 1993). The present finding that maximal strength levels did not significantly decrease following unaccustomed eccentric is not consistent with numerous findings that document strength reduction as being most pronounced 24-48 hours following DOMS inducing exercise (Connolly et al., 2003; Isabell et al., 1992; Smith, 1992). It has been reported that muscular weakness associated with DOMS may not be caused entirely from the reluctance to use the muscle because of the pain and soreness; it is believed that the

inherent capacity of the muscle to produce force is lowered due to the loss of force-producing capability in the involved damaged tissue (Newham et al., 1987). The findings that strength levels remained unchanged following the DOMS inducing protocol may have been due to the protocol used not being demanding enough to weaken the muscle fibers and produce extensive tissue damage.

5.6 RANGE OF MOTION

Although ROM values did not differ between groups, there was a significant time main effect. Supine knee flexion angle and knee extension angle decreased at 24-hours when compared to baseline measures. Both groups gradually regained range of motion over the final 48-hours. The significant decrease in supine knee flexion angle and knee extension angle at 24-hours post exercise is consistent with previous investigations in which a decreased ROM and increased swelling was observed following unaccustomed eccentric activity in untrained individuals and the course of swelling observed (Zainuddin, Newton, and Nosaka, 2005; Gulick and Kimura, 2001; Kraemer et al., 2001). It is believed that the reduced range of motion experienced during DOMS may actually place the involved musculature in an optimal position for healing to occur (Smith, 1991).

The reduced joint range of motion associated with DOMS has been attributed to an increase in the swelling of affected tissues within the muscular tissue and region of the myotendinous junction (Howell et al., 1985). During the inflammatory process, capillary membrane permeability increases, which leads to a large number of plasma proteins escaping the area of damaged tissue. The increase in free proteins in the interstitium and the pressure these

proteins exert disrupt normal capillary filtration balance, and swelling occurs, (Denegar et al., 2006; Kraemer et al., 2001) limiting range of motion of the affected area.

5.7 LIMITATIONS

The present study contained certain limitations that may have contributed to the results observed.

1. Even though the DOMS inducing protocol employed in the present study elicited significant clinical signs and symptoms; direct assessment of damage in human muscle is only possible through analysis of muscle biopsies or through magnetic resonance imaging (MRI) (Clarkson & Hubal, 2002). Therefore, the DOMS inducing protocol may not have been demanding enough to cause extensive muscle damage. Following extensive muscle damage, the ratings of unpleasant feelings felt by the individual would be extremely high; however, in the present investigation the subjects rated their muscle soreness and pain with responses lower than those indicative of extensive muscle tissue damage (Tables 2, 4, and 6).
2. The subjects assigned to the continuous compression treatment group were instructed to continuously wear the compression garment over the course of the investigation. It is not possible for the researcher to determine if the subjects followed this instruction.
3. The subjects were instructed to limit lower-extremity activity for the duration of the study. However, due to the nature of everyday activities such as walking, etc., complete rest was not possible.

5.8 CONCLUSIONS

Despite the many studies examining delayed onset muscle soreness, there is limited scientific evidence supporting the efficacy of therapeutic interventions designed to prevent or minimize its effects. The findings from studies evaluating interventions and treatments for DOMS have been inconclusive and conflicting. The present study is one of a limited number of studies to investigate the effect of continuous compression on the clinical signs associated with DOMS following eccentric exercise and is the first to examine the effect of a continuous compression protocol on the lower extremity. The present findings suggest that continuous compression is beneficial in reducing muscle soreness during the 48-hours after unaccustomed eccentric exercise. By providing mechanical support to the damaged tissues and altering the inflammation-induced increase in fluid to the affected area, a compression garment may decrease the detrimental effects associated with DOMS.

5.9 RECOMMENDATIONS FOR FUTURE RESEARCH

Because the exact mechanism of DOMS is not clearly understood, determining the appropriate treatment is difficult. Based on the findings of this investigation, future research concerning DOMS should consider the following:

1. The findings of the present and previous investigations demonstrate the need for further investigation into the underlying etiology and mechanism(s)

responsible for causing DOMS. This will better allow clinicians to prevent DOMS from occurring or effectively restore maximal function as rapidly as possible.

2. The possible interaction between therapeutic modalities and the inflammatory process and its effect on recovery from the detrimental effects of eccentric exercise.

3. The severity and the distribution of the clinical signs and symptoms associated with DOMS are related to intensity, duration, and type of exercise performed. Therefore, research should examine these variables to determine the most effective way of preventing DOMS from occurring.

4. When is the most appropriate and safest time to return to activity or sport following the occurrence of DOMS, without causing more damage to the underlying muscular structure?

5. The application of additional principles of RICE to include cryotherapy and elevation interventions in combination with compression treatments in an attempt to understand the collective effects of these treatments on the clinical signs and symptoms of DOMS.

6. Different levels and types of compression garments used in conjunction with other therapeutic modalities/interventions on other anatomical regions.

7. The present investigation examined the large quadriceps muscle group. Smaller muscles such as the biceps brachii and gastrocnemius should also be examined.

8. Because the perception of muscle pain and soreness is highly subjective, to minimize the confounding effects associated with the difference in individual responses, it is recommended that a limb-to-limb comparison model be used in which a treatment limb is compared with responses from the contralateral limb of the same subject.

9. DOMS is much more easily induced in untrained individuals; few research studies have been conducted investigating the effects of eccentric exercise on trained and elite athletes. Research using subjects accustomed to regular exercise is needed.

10. Few research investigations have been conducted to determine if DOMS is influenced by sex and age. Understanding possible age and sex differences may provide valuable information regarding the etiology, mechanisms, and possible treatment of DOMS.

APPENDIX A

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE

PAR – Q & YOU

Physical Activity Readiness
Questionnaire - PAR-Q(revised 1994)

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day.
Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.
If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below.
If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.
Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
☐	☐	1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
☐	☐	2. Do you feel pain in your chest when you do physical activity?
☐	☐	3. In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
☐	☐	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition? condition?
☐	☐	7. Do you know of any other reason why you should not do physical activity?

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want - as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active - begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal - this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of temporary illness such as a cold or a fever – wait until you feel better; or
- If you are or may be pregnant – talk to your doctor before you start becoming more active.

Please note: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional.
Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction.

NAME: _____

SIGNATURE: _____ **DATE:** _____
SIGNATURE OF PARENT: _____ **WITNESS:** _____
or **GUARDIAN** (for participants under the age of majority)

Note: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

APPENDIX B

INFORMED CONSENT FORM

Approval Date: May 16, 2006
Renewal Date: May 15, 2007
University of Pittsburgh
Institutional Review Board
IRB #0604011

CONSENT TO ACT AS A SUBJECT IN A RESEARCH STUDY

TITLE: The Effects of Continuous Compression as a Therapeutic Intervention on Delayed Onset Muscle Soreness Following Eccentric Exercise.

PRINCIPAL INVESTIGATOR: Brent Fedorko, M.S., ATC/L
Doctoral Candidate
University of Pittsburgh
140 Trees Hall
Telephone: 412-648-9389
Fax: 412-648-7092

CO-INVESTIGATORS: Fredric L. Goss, Ph.D.
Co-Director Center for Exercise and
Health Fitness Research
Associate Professor of Exercise Physiology
112 Trees Hall, University of Pittsburgh
Telephone: 412-648-8259

Elizabeth Nagle-Stilley, Ph.D.
Assistant Professor of Exercise Physiology
149 Trees Hall, University of Pittsburgh
Telephone: 412-648-8268

Robert J. Robertson, Ph.D.
Co-Director Center for Exercise and
Health Fitness Research
Professor of Exercise Physiology
113 Trees Hall, University of Pittsburgh
Telephone: 412-648-8251

SOURCES OF SUPPORT: University of Pittsburgh School of Education

Participant's Initials_____

Description:

You are being asked to participate in the research study because you are a healthy college-aged male - (only males were selected for this study to reduce the variability of hormone levels and substrate utilization between the sexes). The purpose of this study will be to determine the effect of a continuous compression treatment protocol on the clinical signs and symptoms associated with delayed onset muscle soreness – the muscle pain felt the day(s) following strenuous exercise - following eccentric (the exertion of force by a muscle(s) while lengthening) exercise. Twenty male subjects will be evaluated in this study.

In order to participate in this study, you must not have consistently participated in a lower-body resistance training or exercise program in the last six months, have no prior history of musculoskeletal injury to the lower leg (any injury of the lower leg requiring hospitalization, surgery and/or rehabilitation – i.e. anterior cruciate ligament rupture, hip dislocation, etc.), and have full pain-free flexion/extension range of motion of the hip and knee joints.

Study Procedures:

Time Commitment:

You will make a total of 5 visits over an 11 day time period to the Trees Hall Fitness Facility and Center for Exercise and Health Fitness Research. You will spend a total of 3 ½ hours at the facilities across the five visits. Following the exercise protocol, you should limit water temperature and duration when bathing the exercised leg and refrain from using any pain-relieving modalities (i.e. ice, heat, massage, electrical stimulation, ultrasound, etc.) for the duration of the study. Should you require pain medication related to the study procedures or external issues, the medications should not be withheld, however you will be withdrawn from the study. You should also refrain from any lower body resistance training or stretching techniques (including elevation) for the length of the study. You should maintain your normal diet throughout the study.

Orientation Session:

You will attend an orientation session in the Center for Exercise and Health Fitness Research Laboratory (149 Trees Hall) at the University of Pittsburgh. The orientation session will take approximately one hour. You will first sign an informed consent to participate after the rationale underlying the experiment, as well as, its risks and benefits are explained. You will then be asked to complete a physical activity (describing your current and past exercise activity) and a medical (previous medical conditions and medical history) questionnaire. These questionnaires should take ≈10 minutes to complete. You will be excluded if you do not meet the training requirements (which are described later in this consent form) or if you have a medical condition, which would make it unsafe to participate in the exercise study. Following completion of the

Participant's Initials_____

questionnaire, your height, weight, and percent body fat will be measured. Percent body fat will be measured by using a bioelectrical impedance analysis (BIA) device. BIA is a procedure for assessing body composition in which a scale-like device passes an electrical current through the body and measures the body's impedance or opposition to current flow. BIA is a common method of assessing body composition that is extremely safe and is currently available for personal use in many homes.

You will then be familiarized with the leg flexion/extension machine (Cybex International, Inc). Proper form will be demonstrated. You will be required to perform several low-intensity (lightest weight on the machine) repetitions to familiarize yourself with the exercise machine and exercise motion. Following proper demonstration, your one repetition maximum (1-RM) (the most weight you can lift only one time) will be determined (active muscle pain, passive muscle soreness and rating of perceived exertion associated with the active muscle {RPE-AM} measurements will occur during this warm-up using rating scales). The following is a description of the exercise.

Leg Extension – from the seated position, you will lift the weight with your non-dominant leg by extending your non-dominant knee.

This session (Day 0) should take approximately 1 hour to complete. At the end of this session, you will be scheduled to return in 7 days for baseline measurement of the dependent variables (passive muscle soreness, active muscle pain, RPE-AM, swelling, flexion angle and extension angle) by the principal investigator, as well as the delayed onset muscle soreness (DOMS) inducing protocol.

Experimental Trials:

Day 1 – The experimental trials will be performed between 8 a.m. and noon. With the principal investigator blinded to the process, you will be randomly assigned (after being matched with another subject with equal/similar strength values - one subject per protocol group) to one of the protocol groups: continuous compression (C) or no treatment (N). Dependent variable: 1) passive muscle soreness, 2) active muscle pain, 3) rating of perceived exertion associated with the active muscle 4) swelling, 5) strength, 6) flexion angle and 7) extension angle will be measured prior to DOMS inducement in order to obtain baseline values. The following is a description of measurement procedures.

Muscle soreness, muscle pain and RPE-AM - will be measured using rating scales.

muscle soreness – visual analog rating scale
muscle pain – category rating scale
RPE-AM – anchor-based rating scale

Swelling - leg circumference measurements will be obtained with a spring-loaded flexible tape measure.

Participant's Initials_____

Strength- muscular strength values will be obtained by measuring the maximal weight lifted for 1 repetition.

Flexion/Extension Angles – angles will be measured with a standard goniometer (a large protractor with measurement degrees) with 1° markings.

If you are assigned to the continuous compression group you will be fitted with a compression garment (McDavid Sports Medicine, Inc) and instructed as to its proper application. The thigh support is a 1/8" thick neoprene sleeve with reversible nylon on both sides. If you are assigned to the control group you will not receive a compression garment.

Induction of DOMS:

DOMS will be induced to your non-dominant quadriceps muscle – the quadriceps muscle group is located on the front of your thigh. You will perform the leg flexion/extension exercise at a weight equal to 73.5% of your 1-RM. You will perform three sets of repetitions. The first set will consist of 10 repetitions followed by a 90 second rest period. The resistance of the second and third sets will stay the same as the first (73.5%), however you will complete as many repetitions as possible during each set as determined by correct form and technique. You will have a 90-second rest period between the second and third sets.

Continuous Compression Protocol:

Immediately following the muscle soreness inducing protocol, subjects in the (CC) group will be fitted with the compression garment.

This session should take approximately 1 hour to complete.

Days 2 –(24 hrs. post-exercise), 3 – (48 hrs. post-exercise), 4 – (72 hrs. post-exercise). You will be instructed to report at approximately the same time as Day 1 for measurement of the dependent variables. The measurements for all subjects will be collected with the leg musculature exposed (compression garment removed). Each session should take approximately ½ hour to complete.

Risks and Benefits:

Resistance Exercise:

It is possible as with any experiment that harmful effects may occur. To minimize any risk of injury you be instructed on the proper test procedures and only trained laboratory personnel will conduct the testing. In addition, all staff is certified in CPR/AED and First Aid and Safety by the American Red Cross and a Licensed Certified

Participant's Initials_____

Athletic Trainer will be present during all testing sessions. Resistive exercise is a healthy mode of exercise in this population when proper weight lifting technique is used. The principal investigator will be present to assist during all trials.

Any symptoms of muscle soreness that you may develop should subside within 3-5 days (in case of soreness/injury that is greater than the study window – the principal investigator will follow you until the event is resolved. Additional musculoskeletal injury (muscle strain, ligament sprain, tendon strain) are rare – occurs in less than 1% of people (less than 1 out of 100 individuals).

Bioelectrical Impedance Analysis:

According to the U.S. Department of Health and Human Services, there are no reported adverse events induced by BIA, even in the course of thousands of individuals undergoing measurement. The small current magnitudes involved, less than 1mA, are less than the threshold of pain.

To minimize risks associated with exercise testing, you will be asked to complete the PAR-Q medical history questionnaire. If you have any orthopedic, cardiovascular, and/or metabolic problems that may be worsened by resistance exercise you will be excluded from participation. You may also be removed if you show signs of heart, lung, circulation, muscle, joint or kidney abnormalities. In addition, all staff are certified in CPR/AED and First Aid and Safety by the American Red Cross and a Certified Athletic Trainer will be present during all sessions.

You will receive no direct benefit from your participation in this research study. However, your participation in this study may contribute to understanding the role continuous compression may play as a therapeutic intervention on delayed onset muscle soreness. In addition, if you are an untrained subject, you will be introduced to resistance exercise, which if regularly performed, could improve your overall health.

Alternative Treatment: To date, no treatment or intervention has consistently reduced DOMS or all of its signs. The absence of a preventative measure and the limited effectiveness of current treatment techniques may be the result of the lack of understanding surrounding the exact mechanism of DOMS.

Costs and Benefits:

There will be no cost to you for participating in this study. You will receive $50.00 for your participation in this study. If you have unforeseen medical problems, which would require removal from the study by the investigator, payment will be prorated based on the number of sessions/hours (Session 0/1 - $14.28 per session, Session 2, 3, 4 - $7.14 per session) completed.

Participant's Initials_____

New Information:

You will be promptly notified if any new information develops during the conduct of the research study that may cause you to change your mind about continuing to participate.

Compensation for Illness and Injury:

University of Pittsburgh researchers and their associates who provide services at the University of Pittsburgh Medical Center (UPMC) recognize the importance of your voluntary participation in their research studies. These individuals and their staffs will make reasonable efforts to minimize, control, and treat any injuries that may arise as a result of this research. If you believe that you are injured as a result of the research procedures being performed, please contact the Principal Investigator or one of the co-investigators listed on the first page of this form.

Emergency medical treatment for injuries solely and directly related to your participation in this research study will be provided to you by the hospitals of UPMC. It is possible that UPMC may bill your insurance provider for the costs of this emergency treatment, but none of these costs will be charged directly to you. If your research-related injury requires medical care beyond this emergency treatment, you will be responsible for the costs of this follow-up care unless otherwise specifically stated below. There is no plan for monetary compensation. You do not, however, waive any legal rights by signing this form.

Confidentiality:

All records pertaining to your involvement in this research study will be stored in a locked file cabinet. Your identity on these records will be indicated by a case number rather than your name, and the information linking these case numbers to your identity will be kept separate from the research records. This information will only be accessible to the investigators and research study staff listed on the first page of this document. At completion of this study, your records will be kept under lock and key by the primary investigator for a minimum of five years after which time they will be destroyed.

Any information about you obtained from this research will be kept as confidential as possible. You will not be identified by name in any publication of research results unless you sign a separate form giving your permission (release). In unusual cases, your research records may be released in response to an order from a court of law. It is also possible that appropriate government agencies and/or the University Research Conduct and Compliance Office may inspect your research records. If the researchers learn that you or someone with whom you are involved is in serious danger or harm, they will need to inform the appropriate agencies as required by Pennsylvania law.

Participant's Initials_____

Approval Date: May 16, 2006
Renewal Date: May 15, 2007
University of Pittsburgh
Institutional Review Board
IRB #0604011

Right to Withdraw:

You do not have to participate in this investigation and can withdraw at anytime if you change your mind. You can also withdraw if you decide to pursue alternative measures for DOMS treatment. To formally withdraw your consent for participation in this research study you should provide a written and dated notice of your decision to the principal investigator of this research study at the address listed on the first page of this form. You may be removed from the study by the investigators if you do not follow the protocol that has been established or if they feel that it is in your best interest.

VOLUNTARY CONSENT

The above information has been explained to me and all of my current questions have been answered. I understand that I am encouraged to ask questions about any aspect of this research study during the course of this study, and that such future questions will be answered by a qualified individual or by the investigator(s) listed on the first page of this consent document at the telephone number(s) given. I understand that I may always request that my questions, concerns or complaints be addressed by a listed investigator.

I understand that I may contact the Human Subjects Protection Advocate of the IRB Office, University of Pittsburgh (1-866-212-2668) to discuss problems, concerns, and questions; obtain information; offer input; or discuss situations in the event that the research team is unavailable.

By signing this form, I agree to participate in this research study. A copy of this consent form will be given to me.

Participant's Signature	Printed Name of Participant	Date

CERTIFICATION OF INFORMED CONSENT

I certify that I have explained the nature and purpose of this research study to the above-named individual(s), and I have discussed the potential benefits and possible risks of study participation. Any questions the individual(s) have about this study have been answered, and we will always be available to address future questions as they arise.

Printed Name of Person Obtaining Consent	Role in Research Study
Signature of Person Obtaining Consent	Date

Participant's Initials_____

APPENDIX C

EXERCISE HISTORY QUESTIONNAIRE

EXERCISE HISTORY QUESTIONNAIRE

Name:_____

1. How would you classify your weight lifting experience?

    a. No experience
    b. Some experience, but have not consistently lifted weights in the last six months

2. Do you regularly participate in aerobic activities (i.e. jogging, cycling)?

    Yes ____ No____

    If Yes, How often? How long?

    a. <2 times per week . a. < 30 minutes/session
    b. 2-4 times per week b. 30-60 minutes/session
    c. >4 times per week c. >60 minutes/session

3. Have you ever had any chronic problems with your:

    ___ back/spine ___hip(s) ___knee(s) ___ankle(s) ___No Problems

    If Yes, What? _____

4. Do you have any cardiovascular, orthopedic or metabolic (i.e. diabetes, high/low blood lipids, etc.) contraindications to performing resistance exercise?

    a. No
    b. Yes, if so what? _____

This statement acknowledges that I, _____, have answered each question honestly and accurately to the best of my knowledge.

_____
Witness's Signature

APPENDIX D

LEG (KNEE) EXTENSIONS

LEG (KNEE) EXTENSIONS

Starting Position

1. Load the appropriate resistance (lock pull pin into place).
2. Align the axis of the knee with pivot point of the machine and adjust the back pad to support the body while maintaining this position.
3. Grasp the handles to hold the torso immobile.

Upward Motion

4. Extend the knee until it is straight – (flex the knee as far as possible without moving the pelvis/spine).
5. Keep the torso erect and the back firmly pressed against the back pad.
6. Maintain a tight grip on the handles.

Downward Motion

7. Allow the knee to slowly flex back to the starting position with a smooth controlled movement.
8. Keep the torso erect and the back firmly pressed against the back pad.

*Inhale and raise the legs to horizontal.
*Exhale at the end of the exercise.

APPENDIX E

PASSIVE MUSCLE SORENESS

Passive Muscle Soreness - Visual Analog Scale

The scale before you is a visual analog scale that ranges from "no soreness" to "worst possible soreness". Before the warm-up repetitions you will be asked to place a mark along the scale that corresponds with the amount of soreness that you're experiencing in your quadriceps at that moment. Do not over- or underestimate the amount of soreness, just try to estimate it as honestly and objectively as possible.

The scale's range is designed to signify various degrees of soreness ranging from no soreness to the worst possible soreness you can imagine. The left edge of the line represents the complete absence of soreness (no soreness) and the right edge of the line represents the worst possible soreness that you can imagine (worst possible soreness). When rating passive soreness sensation be sure to only attend to the pain in your quadriceps and not other sensations. Remember that the rating you are giving are those of soreness and not those resulting from fatigue you may be experiencing.

In summary, you will be asked to: (i) provide passive soreness ratings in your quadriceps only; (ii) give ratings as accurately as possible; and (iii) not under- or overestimate the soreness, but simply rate your passive pain honestly.

NO SORENESS — **WORST POSSIBLE SORENESS**

APPENDIX F

ACTIVE MUSCLE PAIN

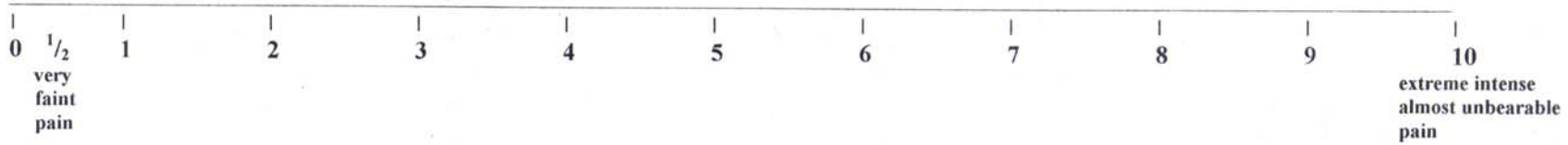
Active Muscle Pain - Category Scale

You are about to undergo an exercise test. The scale before you contains the numbers 0-10. You will use this scale to assess perceptions of active muscle pain in your quadriceps during and after the test. Don't underestimate or over estimate the degree of hurt you feel, just try to estimate it as honestly and objectively as possible.

The numbers on the scale represent a range of pain intensity from "very faint pain (number ½) to extremely intense pain-almost unbearable" (number 10). When you feel no pain in your quadriceps, you should respond with the number 0. If your quadriceps feel extremely strong pain that is almost unbearable, you should respond with the number 10. If the pain is greater than 10, respond with the number that represents the pain intensity you feel in relation to 10. For example, if the pain is twice as intense as 10 give the number 20.

You will be asked to rate the feelings of pain in your quadriceps at the midpoint and immediately following the warm-up repetitions. When rating these pain sensations, be sure to attend only to the specific sensations in your lags and not to report other pains you may be feeling elsewhere. It is very important that your ratings of pain intensity reflect only the degree of hurt you are feeling in your legs either during exercise or following exercise.

In summary you will be asked to: (i) provide active pain intensity ratings in your legs only; (ii) give ratings as accurately as possible; and (iii) not under- or overestimate the pain, but simply rate your rate your pain honestly.



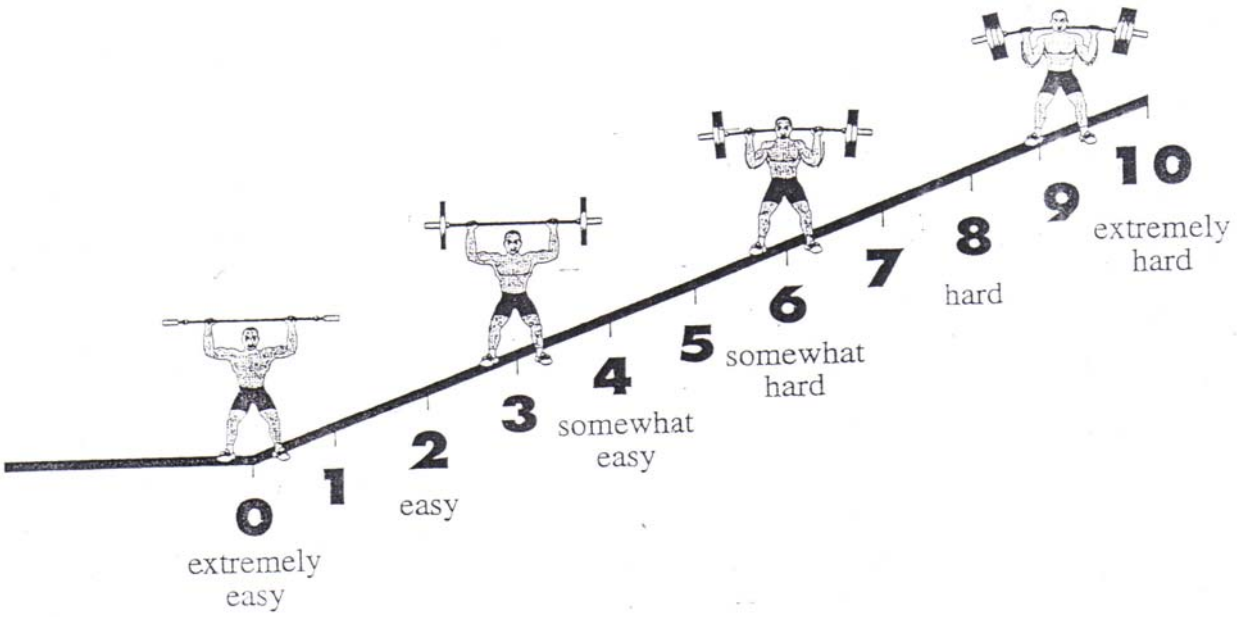
APPENDIX G

RATING OF PERCEIVED EXERTION ASSOCIATED WITH THE ACTIVE MUSCLE (RPE-AM)

Rating of Perceived Exertion associated the Active Muscle (RPE-AM)

You are about to undergo an exercise test. We would like you to use these pictures to describe the effort your quadriceps feel during the warm-up repetitions. You are going to perform weight lifting exercises using one leg. Please look at the person at the bottom of the picture who is lifting a light weight. If you feel like this person when you are lifting the weight the effort will be Extremely Easy. You should give the number zero. Now look at the person at the top of the picture who is barely able to lift a very heavy weight. If you feel like this person when you are lifting the weight the effort will be Extremely Hard. You should give the number 10. If you feel somewhere in between Extremely Easy (0) an Extremely Hard (10), then give a number between 0 and 10. We will ask you to give a number that tells how your quadriceps feel at the midpoint and immediately following the warm-up repetitions.

Remember, there are no right or wrong numbers. Your number can change as you lift the weight. Use both pictures and the words to help select the numbers. Use any of the numbers to describe how you feel when lifting the weight.


0
extremely easy
1
2
easy
3
4
somewhat easy
5
6
somewhat hard
7
8
hard
9
10
extremely hard

APPENDIX H

MCDAVID THIGH SUPPORT

McDavid Thigh Support

471-Thigh Support

Two-sided reversible nylon for durability.

Sizes: S-XL

Reversible thermal neoprene support with nylon facing on both sides for easy application and removal. Heavy-duty nylon on outer side for durability.

Thigh Sizing * Note: Measure around largest circumference of thigh for correct fit.	
Measure around thigh	**Size**
18" - 20"	S
20"- 22"	M
22" - 25"	L
25" - 28"	XL

WARNING: Do not apply over open wounds or if you have a susceptibility to dermatitis, an allergy to rubber or circulatory problem(s).

BIBLIOGRAPHY

Abbott, B.C., Bigland, B. & Ritchie, J.M. (1952). The physiological cost of negative work. Journal of Physiology, 117, 380-390.

Abraham, W.M. (1977). Factors in delayed muscle soreness. Medicine and Science in Sports, 9(1), 11-20.

Armstrong, R.B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Medicine and Science in Sports and Exercise, 16(6), 529-538.

Armstrong, R.B. (1990). Initial events in exercise-induced muscular injury. Medicine and Science in Sports and Exercise, 22, 429-435.

Armstrong, R.B., Warren, G.L. & Warren, J.A. (1991). Mechanisms of exercise-induced muscle fibre injury. Sports Medicine, 12(3), 184-207.

Assmussen, E. (1956). Observations on experimental muscle soreness. Acta Rheumatol Scandinavica, 1, 109-116.

Baechle, T.R. (1994). Essentials of Strength Training and Conditioning. Champaign: IL, Human Kinetcs.

Baumgartner, T., & Jackson, A. (1999). Measurement for Evaluation in Physical Education and Exercise Science. Dubuque, IA: Brown.

Bobbert, M.F., Hollander A.P. & Huijing P.A. (1986). Factors in delayed onset muscular soreness of man. Medicine and Science in Sports and Exercise, 18(1), 75-81.

Brennan, M.J., Miller, L.T. (1998). Overview of treatment options and review of the current role and use of compression garments, intermittent pumps, and exercise in the management of lymphedema. CANCER Supplement 83(12), 2821-2827.

Butterfield, D.L., Draper, D.O., Ricard, M.D., Myrer, J.W., Durrant, E. & Schulthies, S.S. (1997). The effects of high-volt pulsed current electrical stimulation on delayed-onset muscle soreness. Journal of Athletic Training, 32(1), 15-20.

Byrnes, W.C., Clarkson P.M., White, J.S., Hsieh, S.S., Frykman, P.N. & Maughan, R.J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. Journal of Applied Physiology, 59(3), 710-715.

Cheung, K., Hume, P.A. & Maxwell, L. (2003). Delayed onset muscle soreness: treatment strategies and performance factors. Sports Medicine, 33(2), 145-164.

Chleboun, G.S., Howell, J.N., Baker, H.L., Ballard, T.N., Graham, J.L. Hallman, H.L., Perkins, L.E., Schauss, J.H. & Conatser, R.R. (1995). Intermittent pneumatic compression effect on eccentric exercise-induced swelling, stiffness, and strength loss. Archives of Physical Medicine and Rehabilitation, 76, 744-749.

Cohen, J. (1988). Statistical Power Analysis for the Behavioral Sciences, 2nd ed. Hillsdale, NJ; Lawrence Erlbaum Association.

Clark, R. (1994). Effects of cryotherapy and compression treatment protocols on pain and functional signs of delayed-onset muscle soreness. Dissertation. University of Pittsburgh.

Clarkson, P.M., Nosaka, K. & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. Medicine and Science in Sports and Exercise, 24(5), 512-520.

Clarkson, PM & Sayers, S. (1999). Etiology of exercise-induced muscle damage. Canadian J Appl Physiology, 24: 512-520.

Clarkson, P.M. & Hubal M.J. (2002). Exercise-induced muscle damage in humans. American Journal of Physical Medicine and Rehabilitation, 81(11), S52-S69.

Connolly, D.A.J., Sayers, S.P. & McHugh, M.P. (2003). Treatment and prevention of delayed onset muscle soreness. Journal of Strength and Conditioning Research, 17(1), 197-208.

Cook DB, O'Connor PJ, Eubanks SA, Smith JC & Lee M. (1997). Naturally occurring muscle pain during exercise: assessment and experimental evidence. Medicine and Science in Sports and Exercise; 29(8): 999-1012.

Craig, J.A., Bradley, J., Walsh, D.M., Baxter, G.D. & Allen, J.M. (1999). Delayed onset muscle soreness: lack of effect of therapeutic ultrasound in humans. Archives of Physical Medicine and Rehabilitation, 80(3), 318-323.

Denegar, C.R. & Huff, C.B. (1988). High and low frequency TENS in the treatment of induced musculoskeletal pain: a comparison study. Journal of Athletic Training, 23, 235-237.

Denegar, C.R. & Perrin, D.H. (1992). Effect of transcutaneous electrical nerve stimulation, cold, and a combination treatment on pain, decreased range of motion, and strength loss associated with delayed onset muscle soreness. Journal of Athletic Training, 27(3), 200-206.

Denegar, C.R. (2000). Therapeutic Modalities for Athletic Injuries. Champaign, IL. Human Kinetics.

Denegar, C.R., Saliba, E., & Saliba, S. (2006). Therapeutic Modalities for Musculoskeletal Injuries. Champaign, IL. Human Kinetics.

DeVries, H.A. (1961). Prevention of muscular stress after exercise. Research Quarterly, 32, 177.

DeVries, H.A. (1961). Electromyographic observations on the effects of static stretching upon muscular distress. Research Quarterly, 32, 468-479.

Dixon, CB. (2002). The effect of resistance training status on free radical production and muscle damage following an acute resistance exercise bout. Dissertation. University of Pittsburgh.

Donley, P.B. & Denegar, C.R. (1994) Managing Pain with Therapeutic Modalities. In: Therapeutic Modalities in Sports Medicine. 3rd edition, Prentice: Mcgraw-Hill., 38.

Ebbeling, C.B. & Clarkson, P.M. (1989). Exercise-induced muscle damage and adaptation. Sports Medicine, 7, 207-234.

Ernst, E. (1998). Does post-exercise massage treatment reduce delayed onset muscle soreness? A systematic review. British Journal of Sports Medicine, 32, 212-214.

Faulkner, J.A., Brooks, S.V. & Opiteck, J.A. (1993). Injury to skeletal muscle fibers during contractions: conditions of occurrence and prevention. Physical Therapy, 73, 911-921.

Friden, J., Sjostrom, M. & Ekblom, B. (1981). A morphological study of delayed muscle soreness. Experientia, 37, 506-507.

Friden, J. (1984). Muscle soreness after exercise: implications of morphological changes. International Journal of Sports Medicine, 5, 57-66.

Friden, J., Sfakianos, P.N. & Hargens, A.R. (1986). Muscle soreness and intramuscular fluid pressure: comparison between eccentric and concentric load. Journal of Applied Physiology, 61(6), 2175-2179.

Garrett, W. & Tidball J. (1988). Myotendinous junction: structure, function, and failure. In: Woo & Buckwalter (Eds.) Injury and Repair of the Musculoskeletal Soft Tissues, American Academy of Orthopaedic Surgeons., 171-207.

Gulick, D.T. & Kimura, I.F. (1996). Delayed onset muscle soreness: what is it and how do we treat it? Journal of Sport Rehabilitation, 5, 234-243.

Gulick, D.T., Kimura, I.F., Sitler, M., Paolone, A. & Kelly, J. (1996). Various treatment techniques on signs and symptoms of delayed onset muscle soreness. Journal of Athletic Training, 31(2), 145-152.

Hart, J.M., Swanik, C.B. & Tierney, R.T. (2005). Effects of sport massage on limb girth and discomfort associated with eccentric exercise. Journal of Athletic Training, 40(3), 181-185.

High, D.M. Howley, E.T. (1989). The effects of static stretching and warm-up on prevention of delayed onset muscle soreness. Research Quarterly in Exercise and Sport, 60(4), 357-361.

Hilbert, J.E., Sforzo, G.A. & Swenson, T. (2003). The effects of massage on delayed onset muscle soreness. British Journal of Sports Medicine, 37, 72-75.

Hough, T. (1902). Ergographic studies in muscular soreness. American Journal of Physiology, 2, 76-92.

Howell, J.N., Chila, A.G., Ford, G, David, D. & Gates, T. (1985). An electromyographic study of elbow motion during postexercise muscle soreness. Journal of Applied Physiology, 58(5), 1713-1718.

Isabell, W.K., Durrant E., Myrer, W. & Anderson, S. (1992). The effects of ice massage, ice massage with exercise, and exercise on prevention and treatment of delayed onset muscle soreness. Journal of Athletic Training, 27(3), 208-217.

Kaminski, M & Boal, R. (1992). An effect of ascorbic acid on delayed-onset muscle soreness. Pain; 50(3), 327-321.

Kelly, D.D. (1982). The role of endorphins in stress-induced analgesia. Annuals New York Academy of Science,398, 260-270.

Kolb, P. & Denegar, C. (1983). Traumatic edema and the lymphatic system. Athletic Training, 18, 339-341.

Komi, P.V. & Viitasaol, J.T. (1977). Changes in motor activity and metabolism in human skeletal muscle during and after repeated eccentric and concentric contractions. Acta Physiologica Scandinavica, 100, 246-254.

Kraemer, W.J., Bush, J.A., Wickham, R.B., Denegar, C.R., Gomez, A.L, Gotshalk, L.A., Duncan, N.D., Volek, J.S., Newton, R.U., Putukian, M. & Sebastianelli, W.J. (2001). Continuous compression as an effective therapeutic intervention in treating eccentric-exercise-induced muscle soreness. Journal of Sport Rehabilitation, 10, 11-23.

Lanier, A.B. (2003). Use of nonsteroidal anti-inflammatory drugs following exercise-induced muscle injury. Sports Medicine, 33(3), 177-186.

Lenn, J., Uhl, T., Mattacola, C., Boissonneault, G., Yates, J., Ibrahim, W. & Bruckner, G. (2002). The effects of fish oil and isoflavones on delayed onset muscle soreness. Medicine and Science in Sports and Exercise, 34(10), 1605-1613.

Lieber, R.L., & Friden, J. (1993). Muscle damage is not a function of muscle force but active muscle strain. Journal of Applied Physiology74(2), 520-526.

Lightfoot, J.T., Char, D., McDermott, J. & Goya, C. (1997). Immediate postexercise massage does not attenuate delayed onset muscle soreness. Journal of Strength and Conditioning Research, 11(2), 119-124.

Lund, H. Vestergaard-Poulsen, P., Kanstrup, I.L. & Serjsen, P. (1998). The effects of passive stretching on delayed onset muscle soreness, and other detrimental effects following eccentric exercise. Scandinavian Journal of Medicine and Science in Sports, 8, 216-221.

MacIntyre D.L., Reid, W.D., Lyster, D.M., Szasz, I.J. & McKenzie, D.C. (1996). Presence of wbc, decreased strength, and delayed soreness in muscle after eccentric exercise. Journal of Applied Physiology, 80(3), 1006-1013.

McBride, J., Kraemer, W., Triplett-McBride, T. & Sebastianelli, W. (1998). Effect of resistance exercise on free radical production. Medicine and Science in Sports and Exercise, (30), 1, 67-72.

McHugh, M.P., Connolly, D.A.J., Eston, R.G. & Gleim, G.W. (1999). Exercise induced muscle damage and potential mechanisms for repeated bout effect. Sports Medicine, 27, 158-170.

Mekjavic, I.B., Exner, J.A. & Tesch, P.A. (2000). Hyperbaric oxygen therapy does not affect recovery from delayed onset muscle soreness. Medicine and Science in Sports and Exercise, 2000, 32(3), 558-563.

Newham, D.J., McPhail, G., Mills, K.R. & Edwards, R.H.T. (1983). Ultrastructural changes after concentric and eccentric contractions of human muscle. Journal of Neurological Sciences, 61, 109-122.

Newham, D.J., Mills, K.R., Quigley, B.M. & Edwards, R.H.T. (1983). Pain and fatigue after concentric and eccentric muscle contractions. Clinical Science, 64, 55-62.

Newham, D.J. (1988). The consequences of eccentric contractions and their relationship to delayed onset muscle pain. European Journal of Applied Physiology, 57, 352-359.

Noble, B.J., & Robertson, R.J. (1996). Perceived Exertion, 1st ed. Champaign, IL; Human Kinetics.

Norkin, CC. & White DJ. (1985). Measurement of Joint Motion: A Guide to Goniometry. 1st ed. Philadelphia, PA; FA Davis.

Nosaka, K. & Clarkson, P.M.. (1996). Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. International Journal of Sports Medicine, 17, 120-127.

Paddon-Jones, D.J. & Quigley, B.M. (1997). Effect of cryotherapy on muscle soreness and strength following eccentric exercise. International Journal of Sports Medicine, 18(8), 588-593.

Plaskett, C., Tiidus, P.M. & Livingstone, L. (1999). Ultrasound treatment does not affect postexercise muscle strength recovery or soreness. Journal of Sports Rehabilitation, 8, 1-9.

Prentice, W.E. (1994). Therapeutic Modalities in Sports Medicine. 3rd edition. St. Louis, MO; Mosby-Year Book, Inc.

Prentice, W.E. (2003). Arnheim's Principles of Athletic Training, 11th edition. New York, NY; McGraw-Hill.

Proske, U. & Allen, T.J. (2005) Damage to skeletal muscle from eccentric exercise. Exercise and Sports Sciences Reviews, 33, 98-104.

Robertson, R.J. (2004). Perceived Exertion for Practitioners, 1st ed. Champaign, IL; Human Kinetics., 31.

Robertson, R.J., Goss, F.L., Rutkowski, J., Lenz, B., Dixon, C., Timmer, J., Frazee, K., Dube. J., & Andreacci, J. (2003). Concurrent Validation of the OMNI Perceived Exertion Scale for resistance Exercise. Medicine and Science in Sports and Exercise, 35(2), 333-341.

Rodenburg, J.B., Bar, P.R. & DeBoer, R.W. (1993). Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. Journal of Applied Physiology, 74, 2976-2983.

Saxton, J.M., Donnelly, A.E. & Roper, H.P. (1994). Indices of free-radical-mediated damage following maximum voluntary eccentric and concentric muscular work. European Journal of Applied Physiology, 68, 189-193.

Schwane, J.A., Watrous, B.G., Johnson, S.R. & Armstrong, R.B. (1983). Is lactic acid related to delayed-onset muscle soreness? Physician and Sportsmedicine, 11, 124-131.

Smith, L.L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness. Medicine and Science in Sports and Exercise, 23(5), 543-551.

Solomonow, M. & D'Ambrosia, R. (1987). Biomechanics of muscle overuse injuries. a theoretical approach. Clinics in Sports Medicine,62, 241-257.

Staples, J.R., Clement, D.B., Taunton, J.E. & McKenzie, D.C. (1999). Effects of hyperbaric oxygen on a human model of injury. The American Journal of Sports Medicine, 27(5), 600-605.

Stauber, W.T. (1989). Eccentric action of muscles: physiology, injury and adaptation. Exercise and Sport Sciences Reviews, 17, 157-185.

Symons, T.B., Clasey, J.L., Gater, D.R. & Yates, J.W. (2004). Effects of deep heat as a preventative mechanism on delayed onset muscle soreness. Journal of Strength and Conditioning Research, 18(1), 155-161.

Szymanski, D.J. (2001). Recommendations for the avoidance of delayed-onset muscle soreness. Strength and Conditioning Journal, 23(4), 7-13.

Talag, T. (1973). Residual muscular soreness as influenced by concentric, eccentric and static contraction. Research Quarterly, 44, 458-469.

Timmer, J. (2002). The effect of vitamin c supplementation on serum free radical levels and delayed onset muscle soreness following an acute bout of resistive exercise. Dissertation. University of Pittsburgh.

Trenell, M.I., Rooney, K.B, Sue, C.M., & Thompson, C.H. (2006). Compression garments and recovery from eccentric exercise: a p-mrs study. Journal of Sports Science and Medicine, 5, 106-114.

Tsang, K., Hertel, J. & Denegar, C.R. (2003). Volume decreases after elevation and intermittent compression of postacute ankle sprains are negated by gravity-dependent positioning. Journal of Athletic Training, 38(4), 320-324.

Tullson, P.C. & Armstrong, R.B. (1978). Exercise-induced muscle inflammation. Federal Proceedings, 37, 663.

Warren, G.L., Lowe, D.A. & Armstrong, R.B. (1999). Measurement tools used in the study of eccentric contraction-induced injury. Sports Medicine, 27(1), 43-59.

Watkins, M.A., Riddle, D.L., Lamb, R.L., & Personius, W.J. (1991). Reliability of goniometric measurements and visual estimates of knee range of motion obtained in a clinical setting. Physical Therapy, 71(2), 90-96.

Wenos, J.Z., Brilla, L.R. & Morrison, M.J. (1990). Effect of massage on delayed onset muscle soreness. Medicine and Science in Sports and Exercise, 22, 534.

Wessel, J. & Wan, A. (1994). Effect of stretching on the intensity of delayed-onset muscle soreness. Clinical Journals in Sports Medicine, 4(2), 83-87.

Zainuddin, Z., Newton, M., Sacco, P., and Nosaka, K. (2005). Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. Journal of Athletic Training,40(3), 174-180.