Ithaca College Digital Commons @ IC

Ithaca College Theses

2010

Manipulating the Extent of Delayed Onset Muscle Soreness

Ankita Dubey Ithaca College

Follow this and additional works at: http://digitalcommons.ithaca.edu/ic_theses Part of the <u>Exercise Science Commons</u>

Recommended Citation

Dubey, Ankita, "Manipulating the Extent of Delayed Onset Muscle Soreness" (2010). Ithaca College Theses. Paper 315.

This Thesis is brought to you for free and open access by Digital Commons @ IC. It has been accepted for inclusion in Ithaca College Theses by an authorized administrator of Digital Commons @ IC.



Manipulating the Extent of Delayed Onset Muscle Soreness

A Master's Thesis Proposal presented to the Faculty of the Graduate Program in Exercise and Sport Sciences Ithaca College

In partial fulfillment of the requirements for the degree Master of Science

by

Ankita Dubey

Spring 2010

Ithaca College School of Health Sciences and Human Performance Ithaca, New York

.

CERTIFICATE OF APPROVAL

MASTER OF SCIENCE THESIS

This is to certify that Thesis of

Ankita Dubey

submitted in partial fulfillment of the requirements for the degree of Master of Science in the School of Health Sciences and Human Performance at Ithaca College has been approved

Thesis Adviser:

ABSTRACT

Purpose: The purpose of study was to determine if three discrete levels of soreness can be identified using various magnitudes of eccentric triceps exercise in non-resistance trained, college-aged students. Methods: Male (n=12) and female (n=12) subjects were recruited and randomly assigned to a 20-, 40-, or 60-repetition group (n=8). Subjects performed maximal eccentric triceps contraction at 90° s⁻¹ on a Cybex isokinetic dynamometer. Measurement of peak torque (PT), arm circumference (2, 6, and 9 cm), relaxed arm angle (RANG), elbow range of motion (ROM), descriptor differential scale (DDS) (sensation and unpleasantness), and creatine kinase (CK) were done at baseline and 24, 48, 72, and 96 h post-soreness induction. A 3 x 5 repeated-measure ANOVA was then done for each dependent variable to assess interactions as well as time- and group-main effects. To eliminate a dampening effect of baseline values on group data, a univariate ANOVA was also done to determine if groups were similar at baseline. If they were, a univariate ANOVA on group was done collapsing all time periods but excluding baseline. **Results:** A reduction in PT was proportionate to the amount of exercise, as strength decreased by 13.6%, 32.9%, and 47.3% following 20-, 40- and 60-repetitions, respectively, compared to baseline for all time points combined. Arm circumference (2 cm) was significantly different between 20- and 60- repetition groups, whereas RANG was significantly different between the 20- and 40- and 20- and 60-; there were no differences between the 40- and 60- repetition groups for either variable. Also, 60-repetitions reduced ROM more than 20-repetitions, but no difference was found for 40-repetitions. Surprisingly, DDS (sensation) was same for all three groups, but DDS (unpleasantness) was significantly different between the 20- and 60- repetition groups, as was CK. Changes in all variables except CK peaked at 48 h and had

iii

not returned to baseline at 96 h. **Conclusion:** Under the present experimental conditions, DOMS can be manipulated into three discrete levels as measured by strength loss (PT) and into two levels when assessed with changes in arm circumference at 2 cm, ROM, RANG, and unpleasantness of soreness. This information may be helpful to researchers, as well as health care and exercise professionals, when assessing efficient treatment and prevention strategies for DOMS.

iv

ACKNOWLEDGEMENTS

I would sincerely thank and express my gratitude to following individuals for their assistance and insight throughout this thesis. First, I would like to thank Dr. T. Swensen and Dr. G. Sforzo for their patience, dedication, and advice. Also thanks go to Dr. D. King and Dr. M. Kaye for their statistical guidance. I would like to thank students of Ithaca College, Exercise and Sports Science for their continuous support and participation. Also thanks to all the participants who volunteered for this study. To end, thanks to my family and friends for the continual support, understanding, and encouragement.

DEDICATION

This thesis is dedicated to my wonderful parents, who have raised me to be the person I am today. You have been with me every step of the way, through good times and bad. Thank you for all the unconditional love, guidance, and support that you have always given me, helping me to succeed and instilling in me the confidence that I am capable of doing anything I put my mind to. Thank you for everything. I love you!

TABLE OF CONTENTS

	Page
ABSTRACT	iii
ACKNOWLEDGEMENTS	iv
DEDICATION	v
LIST OF FIGURES	xii
LIST OF TABLES	xiv
Chapter	
1. INTRODUCTION	1
Statement of Purpose	4
Hypothesis	.4
Assumptions of the Study	Å
Definition of Terms	4
Delimitations	5
Limitations	6
2. REVIEW OF LITERATURE	7
DOMS Defined	7
Mechanism	9
1 The Lactic Acid Theory	9

vii

TABLE OF CONTENTS (continued)

2. The Muscle Spasm Theory	9
3. The Connective Tissue Damage Theory	10
4. The Muscle Damage Theory	10
5. The Inflammation Theory	.11
6. The Enzyme Efflux Theory	11
Signs and Symptoms	. 12
Markers of DOMS	13
1. Cellular and Sub-Cellular	14
2. Magnetic Resonance Imaging (MRI)	15
3. Torque	16
4. Blood Markers	17
5. Circumference and ROM	18
6. Soreness	19
Treatment and Prevention	21
1. Therapeutic Management	21
2. Pharmacological Management	24
3. Nutritional Supplements	25
Summary	26
METHODOLOGY	27

viii

3.

TABLE OF CONTENTS (continued)	
Subjects	27
Experimental Design	27
Procedure	28
1. Baseline Measurement	28
2. Soreness-Inducing Protocol	28
Measurements	29
1. DDS	29
2. Serum CK	29
3. Arm Circumference	30
4. RANG	30
5. Elbow ROM	30
6. Peak Torque	31
Statistical Analyses	31
RESULTS	33
Subject Characterstics	33
Peak Torque	33
Arm Circumference (2 cm)	38
Arm Circumference (6 cm)	42
Arm Circumference (9 cm)	47

ix

4.

TABLE OF CONTENTS (continued)

Relaxed Arm Angle	50
Elbow Range of Motion	53
DDS (Sensation)	59
DDS (Unpleasantness)	62
Creatine Kinase	65
Summary	69
5. DISCUSSION	72
Varying Repetition Number and Physical Measures	72
Varying Repetition Number and Soreness Rating	75
Varying Repetition Number and Creatine Kinase	76
Time Course of Soreness-Induction	77
Practical Implications	80
Summary	81
6. SUMMARY, CONCLUSIONS and RECOMMENDATIONS	82
Summary	82
Conclusions	83
Recommendations	83
REFERENCES	84

х

APPENDICES	
A. Informed Consent Form	92
B. Medical History and Health Habit Form	94
C. 24-Hour Health and Activity History Form	95
D. Instructions	97
E. Differential Descriptor Scale (DDS)	98
F. Soreness Data Collection Sheet	100
G. Raw Data Tables	102

xi

LIST OF FIGURES

Figure		Page
1. Mean peak torque for e	each group at all times	37
2. Torque after collapsing	all time intervals excluding baseline	
measurement for each	group	39
3. Mean arm circumferend	ce (2 cm) for each group at all times	41
4. Arm circumference (2 c	cm) after collapsing all time intervals	,
excluding baseline mea	asurement for each group	43
5. Mean arm circumferend	ce (6 cm) for each group at all times	46
6. Mean arm circumferend	ce (9 cm) for each group at all times	49
7. Mean RANG for each g	group at all times	52
8. RANG (degrees) after o	collapsing all time intervals excluding	
baseline measurement	for each group	54
9. Mean ROM for each g	roup at all times	57
10. ROM after collapsing a	all time intervals excluding baseline	نا هد
measurement for each	n group	58
11. DDS (sensation) for ea	ach group at all times	61
12. DDS (unpleasantness)) for each group at all times	64

LIST OF FIGURES (continued)

	13. DDS (unpleasantness) after collapsing all time intervals excluding	
-	baseline measurement for each group	.66
	14. CK (I L^{-1}) for three groups at each time interval	68
	15. CK (I·L ⁻¹) after collapsing all time intervals excluding baseline	
	measurement for each group	7Ó

LIST OF TABLES

Table	
1. Subject Characterstics	34
2. Peak Torque: ANOVA Summary Table	35
3. Arm Circumference (2 cm): ANOVA Summary Table	40
4. Arm Circumference (6 cm): ANOVA Summary Table	45
5. Arm Circumference (9 cm): ANOVA Summary Table	48
6. RANG: ANOVA Summary Table	51
7. ROM: ANOVA Summary Table	55
8. DDS (Sensation): ANOVA Summary Table	60
9. DDS (Unpleasantness): ANOVA Summary Table	63
10. CK: ANOVA Summary Table	67

Chapter 1

INTRODUCTION

Delayed onset muscle soreness (DOMS) is a well-known experience for both novice and elite athletes (Armstrong, 1984). DOMS is a sensation of pain and stiffness in muscles that occurs for one to five days following unaccustomed eccentric exercise (Armstrong, 1984). DOMS results in reduced physical performance due to loss of voluntary force production and occurs typically at beginning of the sporting season when athletes are returning for training following a period of reduced activity (Cheung, Hume, & Maxwell, 2003). As a result of the pain and strength loss, DOMS potentially reduces athletic performance in many ways, while the perception of functional impairment, reduced joint excursion, and strength loss increase the risk of further injury (Cheung et al., 2003).

In common experiences, DOMS can be mild producing only a little discomfort for involved muscles. However, DOMS can also be so severe that it leads to hospitalization (Sayers, Clarkson, Rouzier & Kamen, 1999). Severe, eccentrically induced-muscle damage can induce exertional rhabdomyolysis, which describes degeneration of muscle cells and is characterized by elevated serum enzyme level, swelling, pain, stiffness of muscles, fever, nausea, vomiting, abnormal histology, hemoglobinuria, and myoglobinuria (Knochel, 1982). High levels of myoglobin can lead to renal failure, which is an extreme consequence of severe DOMS (Knochel, 1982).

Eccentric exercise produces greater severity of DOMS related symptoms than isometric or concentric exercise (Brown, Child, Day & Donnelly, 1997; Smith, 1992; Walsh, Tonkonogi, Malm, Ekblom & Sahlin, 2001). Brown, Day and Donnelly (1999) assessed muscle damage following 50-maximal concentric knee

extensor contraction followed by same number of eccentric contractions and concluded that concentric exercise induced no change in maximum isometric voluntary contraction (MVC) nor in serum creatine kinase (CK) or lactate dehydrogenase (LDH). Eccentric exercise reduced MVC by 23 ± 19% (mean ± SD) and increased CK and LDH on day three postexercise. The study concluded that eccentric but not concentric contraction induced muscle damage. Similarly, Malm et al. (2004) studied the effects of three grades of running on markers of DOMS. They reported that 45 min of running downhill (-8°) produced greater soreness and pain than running downhill (-4°) or uphill (4°). This study illustrated that downhill running induced greater soreness dependent on the degree of eccentric exercise, with greater eccentric work yielding greater soreness are based on the degree of eccentric exercise and the markers of DOMS should follow suit.

Direct measures of exercise-induced muscle damage include sub-cellular disturbances, particularly Z-line streaming, resulting in distortion of these weak links in the myofibrillar chain (Clarkson & Hubal, 2002). Some indirect markers of muscle damage include an increase in T2 signal intensity via MRI, decreased force production, increased level of inflammatory markers in the injured muscle and blood, increased appearance of muscle proteins in the blood, and elevated ratings of muscle soreness (Clarkson & Hubal, 2002). Common means to measure the consequences of DOMS include: torque, swelling, and range of motion (Warren, Lowe, & Armstrong, 1999). Brown et al. (1997) looked at exercise-induced skeletal muscle damage, creatine kinase (CK), and soreness at three exercise intensities. Three groups performed 10, 30, or 50 maximal eccentric contractions of the quadriceps. CK levels and perception of soreness were highest after the 50 repetition condition. According to this study, indirect markers of DOMS appear to be related to the magnitude of the exercise stimulus.

Researchers have suggested many treatments to allay DOMS, but most are not well substantiated and vary widely in application (Smith, 1991; Ernst, 1998). Interventions for DOMS can be divided into three broad categories: pharmacological therapy, physical modalities, and nutritional supplements (Connolly, Sayers, & McHugh, 2003). A common intervention to attenuate DOMS is massage therapy. Various studies assess the efficacy of massage to reduce soreness and improve muscle function but results are inconsistent. In one study, soreness and inflammatory responses diminished following 30 min of postexercise massage (Smith et al., 1994). In another study, no change in soreness or inflammatory response occurred after 10 min of massage therapy immediate and 24 h postexercise (Lightfoot, Char, McDermott, & Goya, 1997). Inconsistency in study results may be attributed to different types and intensity of the soreness-induction protocols used. Thus, treatment strategies do not consider different levels of muscle damage, or DOMS, due to varying levels of exertion. Treatment effectiveness may be entirely dependent on whether DOMS is in a mild, moderate, or severe form. In other words, when the extent of DOMS varies than treatment protocols may need to vary accordingly.

While it is commonly understood that DOMS emerges in different levels, no prior study specifically attempted to manipulate intensity of DOMS. In this study we intentionally attempt to manipulate soreness and study varying levels of DOMS. If DOMS can be observed at different levels, than future studies can assess if specific treatments are effective at various levels of soreness. By identifying varying levels of soreness, researchers may also be able to better understand the underlying mechanisms for DOMS.

Statement of Purpose

The purpose of this study was to determine if three discrete levels of soreness (i.e., mild, moderate, and severe) could be identified using various magnitudes of eccentric triceps exercise in non-resistance trained, college-aged students.

Hypothesis

The null hypothesis was that eccentrically produced muscle damage, as measured by peak torque (PT), relaxed elbow angle (RANG), elbow range of motion (ROM), arm circumference, soreness ratings, and serum CK can not be differentiated into three discrete levels (i.e., mild, moderate and severe) by varying the eccentric exercise load.

Assumptions of the Study

For the purpose of this study, the following assumptions were made:

1. Isokinetic eccentric triceps exercise induces muscle damage.

2. Arm circumference, PT, soreness level, RANG, elbow ROM and CK are indirect but sensitive markers of the degree of eccentrically-induced muscle damage.

3. Perception of pain caused by eccentrically-induced muscle damage is rated similarly among subjects.

Definition of Terms

- Untrained Subjects: Subjects who have not participated in a regular resistance training program (more than twice a week) for upper limb muscles over the past three months.
- 2. Active elbow ROM: The measurement of the achievable distance between the flexed position and the extended position of elbow joint. The normal range of elbow flexion is 0-150 degree and extension is 150-0 degrees when measured by goniometery.

- 3. Relaxed Elbow angle (RANG): It is an indirect measure of muscle or soft tissue stiffness, measured standing with the arm loosely hanging by side. It is the angle between the longitudinal axis of arm and longitudinal axis of forearm when the palm of hand faces towards the body.
- 4. Arm circumference measurement: Measured to assess the edema caused by eccentrically-induced muscle damage. Measurement of arm circumference is typically done at distance of 2, 6, and 9 cm from the medial humeral epicondyle so that information is obtained across the whole muscle belly.
- Eccentric contraction: A type of muscle contraction where the external load exceeds the muscles ability to actively resist the load and the muscle is forced to lengthen active tension is generated.
- 6. Torque: It is a tendency of a force to rotate a joint about an axis.
- Peak torque: For this study, it is the average torque recorded using three maximal eccentric triceps contractions. It is a reliable measure of decreased muscle function following DOMS.
- Eccentric triceps exercise: During an eccentric triceps contraction, the elbow starts movement at straight angle and then bends as the hand moves towards shoulder while subject is trying to straighten elbow producing lengthening contraction of triceps.

Delimitations

- 1. The subjects in this study were all untrained college-aged students.
- The effects of eccentric exercise were only observed on triceps muscles of the non-dominant arm.
- The study was conducted using a Cybex isokinetic dynamometer with three specified protocols (one, two and three sets of 20 repetitions each at 90°·s⁻¹) of maximum isokinetic eccentric triceps contractions intended to induce mild, moderate and severe effects.

4. Muscle damage was estimated with indirect variables including: Elbow ROM, RANG, PT, arm circumference, descriptor differential scale (DDS), and CK.

Limitations

- 1. The results may only be applicable to untrained college-aged students.
- The results may only be applicable to the eccentric workloads (20, 40 and 60 maximal eccentric contraction at 90° s⁻¹) applied.
- 3. The results may only be applicable to the non-dominant triceps muscle.
- 4. The results may only be applicable to DOMS markers studied (i.e., ROM, PT, arm circumference, DDS, RANG, and CK).

Chapter 2

REVIEW OF LITERATURE

Nearly everyone has experienced DOMS at some time. Originally, it was thought that DOMS simply resulted from micro-tears in muscle fibers, but research shows that the mechanism for DOMS is more complicated (Clarkson et al., 2002). Many studies have examined the indicators, treatments, and prevention schemes for DOMS (Clarkson et al., 2002). However, little work has been done to examine the potential to elicit various levels of DOMS intensity. This chapter reviews literature relevant to the study of DOMS. The first section of this review outlines the definition, mechanisms, symptoms, and proposed treatments and preventions for DOMS. The second section outlines the different markers of DOMS and focuses on markers that might reflect the level of DOMS , related to muscle damage.

DOMS Defined

Following unfamiliar physical activity, sensations of pain and stiffness are felt in the exercised muscles. The intensity of discomfort increases within the first 24 h following exercise, peaks between 24 and 72 h, subsiding and eventually disappearing in five to seven days postexercise (Cheung et al., 2003). This exercise-induced phenomenon is referred to as DOMS and is perhaps the most common and recurrent sports injury. DOMS usually follows unaccustomed exercises but can also occur with increased intensity and duration of regular exercise. Any muscle that is overly exerted may suffer DOMS. For example, prolonged downhill running might cause pain in major extensors and flexors of the hip, thigh, and leg (Malm et al., 2004). Moreover, there is evidence suggesting that fast twitch muscle fibers are more susceptible to DOMS than

slow twitch fibers. The reason for this difference might be the inherent structural weakness of fast twitch fibers or enhanced recruitment of fast twitch motor units for eccentric exercises (Macpherson, Schork & Faulkner, 1996). Exercise-induced muscle damage is more frequent and limiting in stiff muscles rather than flexible muscles. Individuals with greater muscle stiffness appear to experience greater DOMS after eccentric exercise (McHugh et al., 1999).

DOMS is commonly associated with unaccustomed high force muscular work especially if it involves an eccentric component (Cheung et al., 2003). Eccentric contractions are the lengthening contractions of muscles. Thus, if external load exceeds the muscle's ability to actively resist the load, the muscle is forced to lengthen while active tension is generated (Stauber, 1989). Eccentric exercises result in greater disruption of muscle tissue than concentric exercises, as evidenced from histological and electron microscopy (Armstrong, Ogilvie, & Schwane, 1983). To produce a given muscular force, fewer motor units are recruited during eccentric contraction than concentric contraction. Thus, with eccentric contractions, force is distributed over a smaller cross sectional area of muscle (i.e., greater tension is generated per active cross sectional area) (Armstrong, 1984). It is probable that this increase in tension generated per unit area is the cause of greater mechanical disruption both in muscles and in connective tissue surrounding it. Thus, eccentric exercise can produce DOMS at greater rate and intensity than any other form of exercise. In fact, many eccentric exercises (e.g., downhill running, ballistic stretching, and isokinetic dynamometer) produce DOMS (Brown et al., 1997; Malm et al., 2004; Smith, 1992; & Walsh et al., 2001).

Eccentric exercise results in micro-injury to muscles at greater frequency than any other types of muscle action (Cheung et al., 2003). Warren et al. (1999) suggested that 75% or more of the decline in tension after eccentric exercise was

attributable to a failure of the excitation-contraction coupling process, whereas the reminder of the decline is attributed to the physical disruption of the tensionbearing element within the muscle. Eccentrically-induced muscle damage is associated with connective and contractile tissue micro-trauma (Armstrong, 1986). At the cellular level, eccentric exercise disrupts the cell membrane, setting off an inflammatory response that leads to prostaglandin (PGE₂) and leukotrienes synthesis that causes signs and symptoms of DOMS (Connolly, Sayers, & McHugh, 2003). However, the exact mechanism that leads to DOMS is still controversial.

Mechanism

The purported mechanisms for DOMS include: lactic acid accumulation, muscle spasm, connective tissue damage, muscle cell damage, inflammation, and enzyme efflux (Cheung et al., 2003). However, no one existing mechanism fully accounts for the DOMS phenomenon. Consensus among researchers is that DOMS may be caused by an interaction of some of the following mechanisms (Cheung et al. 2003).

1. The Lactic Acid Theory

It is based on assumption that lactic acid continues to accumulate in the muscle fiber even after exercise is stopped. However, this theory has been rejected as lactic acid levels return to pre-exercise state within one hour following exercise. Moreover, Schwane, Johnson, Watrous, and Armstrong (1983) measured blood lactate level pre-test, during, and sporadically for 72 h after downhill and level run and could not find a relationship between lactic acid and soreness level.

2. The Muscle Spasm Theory

Eccentric exercise increases resting muscle electromyographic (EMG) activity after a training session. It is proposed that this increased activity indicates

9.

a tonic localized spasm of motor units. This spasm leads to compression of blood vessels, ischemia and accumulation of pain substances, which results in a vicious cycle, further increasing muscle spasm (de Vries, 1992). However, one study showed that sore muscles do not have increased EMG activity (Abraham, 1977) whereas others showed that DOMS is associated with a change in EMG activity. However, there is no relationship between magnitude of change in EMG activity and DOMS (Bobbert, Hollander, & Huijung, 1986).

3. The Connective Tissue Damage Theory

There is a difference in composition of connective tissue surrounding type I and type II muscle fibers, as Type I have more connective tissue than type II fibers. As a consequence, type II fibers are more susceptible to stretch-induced injury (Stauber, 1989). Measurement of hydroxyproline and hydroxylysine (components of mature collagen) in urine samples following exercise support this theory, but these collagen components are also found in the urine when collagen synthesis increases. Therefore, it is difficult to fully interpret the significance of the increase in urine hydroxyproline and hydroxylysine following eccentric exercise.

4. The Muscle Damage Theory

Hough (1902) described disruption of contractile tissue, particularly Zlines, following eccentric exercise. A widespread disruption of sarcomere architecture and myofibrillar Z-line is observed following eccentric contraction (Friden & Lieber, 1992). Following tissue disruption, there is stimulation of nociceptors located in muscle connective tissue, arterioles, capillaries, and musculotendinous junction that leads to subsequent pain. CK is a reliable measure for assessing muscle membrane permeability and an increase in blood CK levels is found following disruption of Z-line and damage to sarcolemma (Clarkson & Ebbeling, 1988). Since there is a discrepancy in the rise in serum CK

and peak soreness, the muscle damage theory is not likely the sole explanation for DOMS (Cheung et al., 2003; Clarkson et al., 1988).

5. The Inflammation Theory

The basis for inflammatory theory is that repeated eccentric muscle action activates white blood cell, which precipitate edema (Smith, 1991). Eccentric exercise damages the muscle fiber, which releases proteolytic enzymes and certain factors e.g., bradykinins, histamines, and prostaglandins, all of which attract neutrophils and monocytes to the injured site (Fielding, Manfredi, & Ding, 1993; MacIntyre, Reid, Lyster, Szasz, & McKenzie, 1996). After acute neutrophil accumulation, there is gradual increase in monocytes level in muscle. Subsequently, inflammatory cytokines produced by monocytes are released, such as interleukin (IL)-1, IL-6, tumor necrosis factor and IL- α ; anti-inflammatory cytokines like IL-10 are also released. These factors collectively mediate an inflammatory response that increases osmotic pressure in the injured tissue, thereby causing edema or swelling, which evokes pain. Although peak edema (volume measure and girth measurement) coincides with peak soreness (Gulick, Kimura, Sitler, Paolone, & Kelly, 1996), the time course for inflammatory cell infiltration is not as well correlated (Schwane, Johnson, & Vandenakker, 1983). Consequently, it remains controversial as to whether inflammation and subsequent edema are largely responsible for DOMS.

6. The Enzyme Efflux Theory

Gulick and Kimura (1996) described how calcium accumulates in muscles after sarcolemmal damage. They suggested that cell damage leads to inhibition of cellular respiration at mitochondrial level. Subsequent decline in adenosine triphosphate (ATP) production slows calcium influx into sarcoplasmic retinaculae, which increases intracellular calcium accumulation. This accumulated calcium thereby activates proteases and phospholipases that increases muscle damage.

Consequently, chemical stimulation of pain nerve endings occurs, inducing the discomfort associated with DOMS (Armstrong, 1984).

In summary, none of the aforementioned theories fully account for DOMS. Instead, DOMS likely involves aspects of most of these purported mechanisms. Consequently, an interpreted model for the DOMS has been developed (Cheung et al., 2003). This model suggests that high tensile forces associated with eccentric exercise leads to muscle and connective tissue damage that causes elevated levels of calcium which activates proteases and lipases that cause further sarcomere disruption. Collectively, muscle damage is roughly correlated with increased serum CK levels. Within a few hours inflammation increases, a product of neutrophil infiltration and degranulation. Neutrophil degranulation attracts monocytes, which leads to greater histamine production. In the presence of an inflammatory environment, monocytes convert to macrophages, producing prostaglandins that sensitize type III and type IV nerve endings to mechanical, thermal and chemical stimulation and that also activate nociceptors within the muscle fiber. Subsequent edema elevates pressure within the damaged tissue further increasing sensory neuron activation. At present this model is not fully supported; further research needs to validate the precise mechanistic events describing the occurrence of DOMS (Cheung et al. 2003).

Signs and Symptoms

Muscle and connective tissue damage that occurs with eccentric exercise can alter muscle function and joint mechanics. DOMS is usually subclinical with the sensation varying from slight muscle stiffness, which rapidly disappears during the daily routine, to severe debilitating pain, which restricts movements. The symptoms associated with DOMS last for about one to five days following the exercise. The signs and symptoms associated with DOMS include dull, diffuse pain, tenderness, stiffness, swelling, and decreased muscular strength

(Fitzgerald, Rothstein, Mayhew, & Lamb, 1991). Pain and tenderness typically peak one to three days after exercise and subside within seven days. Pain initially presents at the musculotendinous junction and may gradually spread throughout the muscle belly (Noonan & Garrett, 1992). Stiffness and swelling peak in three to four days after exercise and typically resolve within 10 days.

Significant reduction in joint excursion is observed following eccentrically induced muscle damage. Reduction in joint excursion results from reduced joint range of motion as well as reduced muscle flexibility. One study found 30% reduction in elbow range of motion and increased stiffness following 60 maximal contractions of eccentric elbow flexors (Zainuddin, Newton, Sacco, & Nosaka, 2005). Another study found a 17.8% decrease in elbow range of motion at 72 h postexercise following 30 sets of 10 repetitions of eccentric dumbbell curls at 90% of 10 repetition maximum (Isabell, Durrant, Myrer, & Anderson, 1992).

DOMS is also associated with a decrease in strength, which may peak immediately after exercise or within the first 48 h, and usually lasts for more than five days (Connolly et al., 2003). Peak torque reduction is more pronounced 24-48 h following DOMS-inducing exercises and more persistent with eccentric exercise than any other form of exercise (Smith, 1992). Duration of strength reduction is also greater following eccentric exercises and reconciled in 8-10 days (Ebbeling & Clarkson, 1989). Prolonged torque reduction following eccentric exercise is an indicator of damage to contractile elements, impairments in excitation-contraction coupling, and inflammation (Clarkson et al., 2002).

Markers of DOMS

Numerous data highlight the mechanisms underlying DOMS, and identify preventive and therapeutic treatments for the discomfort. But for any clinical or scientific problem it is important to develop 'markers' or measures that permit specific quantification of the problem (Warren et al., 1999). There are a wide

variety of criteria for quantifying muscle injury, but no general agreement on the best method to do so (Warren et al., 1999). Clarkson et al. (2002), for example, describe direct and indirect measures of muscle damage. Direct measures include assessment of cellular and sub-cellular disturbances through the use of muscle biopsy. Because of inherent errors with this technique, most scientists use indirect markers to assess degree of damage. Indirect measurements include muscle soreness, changes in MRI, decrease in force production, swelling, reduced range of motion, and elevated proteins, and inflammatory markers in blood.

1. Cellular and Sub-Cellular

Unaccustomed exercise damages cellular and sub-cellular muscle structures. The first evidence of muscle damage following eccentric exercise was provided by Friden, Sjostrom, and Ekblom (1981), who used soleus biopsies at two and seven days postexercise to show myofibrillar damage and Z-line streaming after stair descent. In another study Friden, Sjostrom, and Ekblom (1983) analyzed muscle samples collected at one hour, three days, and six days after backward cycling and they found changes in ultra-structural integrity of myofilament, mitochondrial loss, disarrangement of A-band, and Zline streaming. Data from these studies led some to postulate that the Z-line was the weak link in the myofibrillar chain. Further examination concluded that cytoskeletal protein desmin, which links Z-lines together, may be susceptible to exercise-induced damage. In addition, mast cell degranulation occurs in the perimysial area near blood vessels, thereby attracting mononuclear cells, suggesting that there is damage to the extracellular matrix and possible damage to capillaries as well (Stauber, Clarkson, Fritz, & Evans, 1990).

Collectively, these data showed that the initial exercise insult damages the ultra-structure of the muscle fiber, extracellular matrix, and possibly associated

capillaries. But results with biopsy are inconsistent and possibly associated with error. Malm et al. (2000) studied multiple biopsies taken over seven days from placebo and experimental groups (eccentric cycling exercise) and found that there were similar changes in both groups. The biopsy itself might produce damage in muscle that confounds interpretation of changes associated with DOMS. Moreover, biopsies by nature are a small sample of the whole muscle, which makes quantification of damage challenging. It is possible that the amount of damage in the muscle might be underestimated or overestimated with the biopsy technique (Clarkson et al., 2002).

2. Magnetic Resonance Imaging (MRI)

In comparison to biopsy, which gives focal results, MRI is a better, albeit much more expensive, technique because it examines the whole muscle rather than a small sample. Shellock, Fukunaga, Mink, and Edgerton (1991) found increased T2 signal intensity after eccentric exercises, which may be attributed to edema. Another study by Takahashi et al. (1994) found increased signal intensity after eccentric exercise and attributed it to increased water content in the damaged muscle, a consequence of injured connective tissue, increased capillary permeability, or damaged muscle cells. In addition, MRI is useful in assessing which muscles are damaged after exercise. For example, after eccentric forearm flexion exercise, subjects differed in the extent of damage in synergistic muscles with some subjects showing damage in biceps, some in brachialis, and some in both (Nosaka & Clarkson, 1996). Nevertheless, studies assessing muscle damage by MRI are still unclear; thus further studies are 'warranted to assess the correlation between changes in MRI signals and force, torgue, and CK activity. Because of the issues with the aforementioned direct markers of injury, many researchers rely on indirect markers to better understand DOMS.

<u>3. Torque</u>

Change in muscle function may be the best means to evaluate magnitude and time-course of muscle injuries resulting from eccentric exercises. Muscle function is defined as the ability to exert force over a certain range of motion, at a fixed muscle length, or at a given velocity, external load, and level of activation (Warren et al., 1999). Maximum voluntary contraction (MVC) torque, which is directly proportional to the force produced by a muscle, is the most common method used to assess muscle damage. Reliability of the MVC torque measurement is generally high (interclass correlation coefficient \ge 0.85) (Kellis & Baltzopoulos, 1995). Warren et al. (1999) concluded in their review that MVC torque is the best measure of muscle damage resulting from eccentric contractions.

Concentric protocols are associated with a strength loss of 10-30% after exercise, with strength returning to baseline within hours (Jones, Newham, & Torgan, 1989). Low intensity eccentric exercises reduce muscle strength by 10-30%, with a recovery period longer than concentric protocols (Mizrahi, Verbitsky, & Isakov, 2001). High intensity eccentric exercises decreases force by 50-65% and recovery takes 10-12 days (Newham, Jones, & Clarkson, 1987). For example, 60 maximal eccentric contractions of elbow flexors reduced isometric torque by 60% and strength did not return to preexercise levels for 10 days (Zainuddin, Newton, Sacco, & Nosaka, 2005). Similarly, 40 eccentric elbow flexors contractions reduced isometric PT by 22% (Connolly, McHugh, & Padilla-Zakour, 2006). These data show that the drop in PT may be related to the number of contractions or intensity of contractions. It appears that higher the intensity of eccentric exercise, the greater the extent of muscle damage, and hence, the more strength is reduced.

4. Blood Markers

CK is an enzyme that buffers cellular ATP and ADP concentrations by catalyzing the reversible exchange of high energy phosphate bonds between phosphocreatine and ADP produced during contractions (Brancaccio, Maffulli, & Limongelli, 2007). The serum level of this skeletal muscle enzyme is a marker of the functional status of the muscle tissue and varies widely in both pathological and physiological conditions (Brancaccio et al., 2007). Increases in serum CK occur after muscle damaging eccentric exercises, because of changes in the sarcolemma which increases interstitial CK and subsequently circulating CK levels (Brancaccio et al., 2007). After damage, total serum CK begins to increase at around 24 h, peaks around 48 h, and then gradually returns to normal values (Armstrong, 1984). Smith et al. (1994) found a marked increase in CK activity after 26 maximal eccentric chest presses; the baseline CK value was 95.16 IU·L⁻¹, whereas at 48 h it rose to 1410.01 IU·L⁻¹, peaking at 72 h at 2361.01 IU·L⁻¹. Similarly, CK peaked at 72 h to 2704 IU·L⁻¹ after 60 maximal eccentric biceps contraction (Zainuddin et al., 2005). From these studies it can be concluded that peak CK occurs at 72 hours or later postexercise.

Using a compilation of studies, the serum CK can be directly correlated with intensity of exercise. The normal serum CK level is 50 -220 IU·L⁻¹ (Brancaccio et al., 2007). Paulsen et al. (2005) found total serum CK of 5,500 IU·L⁻¹ after 96 h following 300 sub-maximal eccentric contraction of the non-dominant quadriceps. Paschalis, Koutedakis, Jamurtas, Mougios, and Baltzopoulos (2005) found total serum CK level of 1,600 IU·L⁻¹ after 96 h following 120 maximal eccentric contractions of the same muscle group. Collectively, these data showed that the greater the intensity of exercise, the higher the CK level in serum.

Other circulating enzymatic markers of muscle damage include lactate dehydrogenase, aspartate aminotransferase, and carbonic anhydrase isoenzyme II (Sorichter, Puschendorf, & Mair, 1999). In addition to circulating enzymes, circulating muscle proteins are also used to indirectly assess damage. These proteins include myoglobin, fatty acid binding protein, troponin, and myosin heavy chain (Sorichter et al., 1999). Although all the aforementioned enzymes and muscle proteins increase with DOMS, CK receives most of the attention. Since there is no systematic study of all these markers together, we know very little about how they will correlate to each other and to the extent of muscle damage.

5. Circumference and ROM

The effects of DOMS are also assessed by examining the various parameters associated with the inflammation response to eccentric exercise, such as neutrophil accumulation and change in the concentration of various cytokines, e.g., IL-13. Cannon et al. (1989) found that downhill running increased IL-13 for up to 5 days. Similarly, Malm et al. (2000) found increased T-cell expression (inflammatory mediator) at 6 h, macrophages at 48 h, and natural killer cells from 6 h to 7 days after exercise. In a subsequent study, Malm et al. (2004) found that downhill and uphill running at different degrees of incline increased leukocytes levels, with a peak at 6 h in all subjects. Granulocytes, lymphocytes and monocytes were also elevated in all subjects. Since these inflammatory responses induce edema, various researchers have indirectly measured soreness by circumference and ROM.

The inflammatory signals induce an influx of protein rich fluid (exudates) into the damaged muscles by increasing capillary permeability (Smith, 1991). Peak edema level (as measured by limb girth) coincides with peak soreness (Gulick et al., 1996). Swelling peaks around 3-4 days following exercise, as

measured by changes in limb girth, and subsides within 10 days. Cleak and Eston (1992) found that arm circumference increased by 1 cm four days after eccentric biceps exercises. Similarly, 225 eccentric wrist extensor contractions increased forearm circumference by 60 mm from baseline (Gulick et al., 1996).

ROM is defined as the arc through which a joint moves. ROM is determined not only by muscles but all structures that surround the joint like the skin, subcutaneous tissue, tendon, cartilage, and ligaments. Passive ROM is usually used to assess degree of restriction following muscle damage. Full muscle excursion is important for maintaining adequate ROM. But DOMS results in damage to muscle fiber, stiffness, and swelling, which reduces passive ROM. Zainuddin et al. (2005) found that 60 maximal eccentric contractions of elbow flexors reduced passive elbow flexion by 30% with full recovery taking approximately four days.

Some researchers also measure RANG to assess muscle stiffness postexercise. RANG is the angle formed between the forearm and arm when the limb hangs loosely by side of the body and is measured with the elbow as a fulcrum (Cleak & Eston, 1992). Eccentric exercises markedly reduces RANG. Mean RANG decreased to 26° after 70 maximal eccentric contractions of the elbow flexors; the decline peaked at 96 h, with full recovery taking 10 days (Cleak & Eston, 1992). Similarly, Prasartwuth, Taylor, and Gandevia (2005) found a 12.6 \pm 8° decrease in RANG immediately after various quantities of submaximal eccentric elbow flexions. ROM and RANG might be reliable markers of soreness, but further research is warranted to prove reliability across different study protocols.

6. Soreness

Evaluation of pain is one or the most important criteria to assess DOMS. Pain is highly variable and subjective. To evaluate pain, multiple scales have

been developed such as the visual analog scale (VAS), verbal rating scale, and a numerical rating scale. Of these, VAS is the most widely used to measure pain associated with DOMS because it is reliable (Zusman, 1986). A drawback to the VAS is that it gives a less stable estimate of clinical pain than a scale composed of multiple items (Gracely & Kwiloz, 1988). Another problem with the VAS is it assesses pain in only one dimension, while pain is multi-dimensional in nature. The DDS may be better used to assess pain (Gracely et al., 1988) as it measures both the affective and sensory aspects of pain. Consequently, the DDS may be superior to the VAS (Doctor, Slater, & Atkinson, 1995). With a DDS, subjects are asked to rate the magnitude of their clinical pain relative to 12 graded descriptors of pain intensity (sensory dimension) and 12 graded descriptors of pain unpleasantness (affective dimension). Ratings relative to each of the 12 descriptors are averaged within each dimension of pain (intensity and unpleasantness) to get the total score for pain intensity and unpleasantness (Doctor et al., 1995).

Soreness appears many hours after damage inducing exercise, peaking at 24-48 h. Soreness results from the stimulation of group IV nociceptors by noxious chemicals like histamine, bradykinin, and prostaglandin (Friden, Sfakianos, & Hargens, 1986). Level of soreness should be directly related to the degree of muscle damage. Hence, low level exercises like downhill running and isokinetic quadriceps exercise produce soreness values of four or five on a scale of 10 (one equals no soreness and 10 equals maximum soreness), whereas high intensity eccentric elbow exercise produce soreness values of about seven to eight. This timing is also consistent with force loss and increase CK levels. In contrast, peak swelling does not coincide with the soreness level. After eccentric elbow flexor exercise, swelling begins at about 72 h postexercise, peaking by 96 h, subside by day 10 whereas soreness begins to

increase 24 h after exercise and peaked after 72 h and subsides by day eight (Cleak & Eston, 1992).

Treatment and Prevention

Researchers have investigated many treatment and prevention strategies to manage and prevent DOMS, which can be broadly divided into three categories: pharmacological, therapeutic, and nutritional interventions (Connolly et al., 2003).

1. Therapeutic Management

Standard physical therapy treatments for DOMS are cryotherapy, ultrasound, electrical stimulation, stretching, massage, compression, and exercise. Other alternative treatment techniques include hyperbaric oxygen therapy, homeopathy and electromagnetic shielding (Cheung et al., 2003). Despite a large volume of work in this area, there is little agreement among practitioners as to the most efficient treatment to manage DOMS.

Commonly used methods to alleviate DOMS are passive stretching and massage. Initially, it was thought that static stretching pre and postexercise would decrease DOMS, as it relieves muscle spasm and helps disperse edema (Wessel & Wan, 1999). A meta-analysis of five studies (72 subjects) showed that stretching minimally reduced soreness 72 h after exercising (Herbert & Gabriel, 2002). One study concluded that stretching pre and postexercise does not provide protection from muscle soreness and preexercise stretching does reduce the risk of muscle injury (Herbert & Gabriel, 2002).

A number of researchers have assessed the effects of massage on DOMS and indirect markers of muscle damage. As with stretching, the effect of massage on DOMS is also inconclusive (Tidus, 1997). Theoretically, massage reduces symptoms associated with DOMS by altering inflammation, circulation,
endorphin release, and mood state (Bale & James 1991; Farr et al., 2002; Hilbert, Sforzo, & Swensen, 2003; Smith et al., 1994; Tidus & Shoemaker, 1995).

Hilbert et al. (2003) found that 30 min of massage two hours after eccentric hamstring exercise reduced soreness as assessed by DDS and improved mood state. There was no effect, however, on muscle function and neutrophil count. They concluded that massage has psychological rather than physiological effects, a conclusion supported by the work of Tidus et al. (1995). Lightfoot et al. (1997) also found that massage did not alter physiological variables after DOMS, such as leg volume and plasma CK levels; they also found massage did not alter analog soreness ratings. Similarly, Weber et al. (1994) studied the effect of massage, upper body ergometery and micro current electrical stimulation on DOMS and found that there was no change in maximal voluntary isometric contraction and muscle analog soreness ratings among the three modalities.

In contrast to the aforementioned studies that show massage has no physiological effects on DOMS, Rodenburg et al. (1994) found that pre-treatment stretching and ergometery and post-treatment massage improved isotonic force and elbow flexion and lowered CK activity after DOMS. They also found massage did not alter analog soreness ratings, elbow extension, and circulating myoglobin levels. Since the researchers used three different modalities as a treatment, it is difficult to differentiate the relative contribution of each to the reported significant findings. Other studies looking at the effect of massage on DOMS also show it reduces CK levels (Smith et al., 1994). This study, however, found that massage reduced analog soreness ratings, a finding supported by the work of Farr et al. (2002).

As the aforementioned discussion illustrates, the effects of massage on DOMS is equivocal. Perhaps, as suggested by Hilbert et al. (2004), massage may have more psychological rather than physiological effects.

Cryotherapy is also used to alleviate symptoms associated with DOMS. A decrease in tissue temperature following cryotherapy causes vasoconstriction, which reduces swelling and inflammation, thereby decreasing symptoms associated with DOMS. Data from studies that examine the effect of cryotherapy on DOMS are also inconsistent. For example, cryotherapy did not reduce muscle soreness, or improve isometric and isokinetic torque, after 60 maximal eccentric contractions of elbow flexors (Paddon-Jones, & Quigely, 1997). In contrast, 15 min of cold water immersion reduced stiffness and plasma CK level following eccentric elbow flexors exercise (Eston & Peters, 1999).

Another purported palliative for DOMS is continuous compression, which is a low cost intervention for patients suffering from DOMS. Pneumatic compression sleeves prevent joint motion, decrease perception of soreness, and enhance recovery of muscle function (Kraemer et al., 2001). Intermittent pneumatic compression for 20 min immediately after eccentric elbow exercise and daily for five consecutive days reduces swelling and soreness (Kraemer et al., 2001). Further research is required to confirm effectiveness of compression in reducing symptoms associated with DOMS.

Electrotherapeutic modalities used to alleviate symptoms associated with DOMS are ultrasound and electrical stimulation. Ultrasound promotes tissue healing by increasing blood flow and temperature but its effectiveness to manage DOMS is uncertain. Hasson, Mundrof, Barnes, William, and Fuji, (1989) found ultrasound reduced the symptoms of DOMS when given 24 h postexercise. While Ciccone, Leggin, and Callamaro (1991) found ultrasound exacerbated symptoms following eccentric exercise of elbow flexors. The effect of micro-current, high volt

pulsed galvanic (HVPG) electrical stimulation and trans-cutaneous electrical nerve stimulation (TENS) on DOMS is not well studied and no study to date has shown these techniques alleviate DOMS (Schmitz, Martin, Perrin, Iranmanesh, & Roqol, 1997; Weber et al., 1994)

Exercise may be one of most effective means to alleviate DOMS and associated symptoms (Armstrong, 1984). Exercise increases blood flow and endorphin levels, while breaking adhesions in sore muscles and accelerating removal of toxic waste products from the active musculature. However, studies that have examined the effects of exercise on DOMS are inconclusive. One study, showed that upper arm ergometery performed for 8 min immediately following eccentric elbow extensors exercises did not significantly reduce DOMS (Weber et al., 1994). In contrast, another study showed that high velocity concentric isokinetic exercise performed after stepping exercise decreases DOMS (Hasson et al., 1989). The difference in findings between these studies was attributed to differences in exercise protocols. Therefore, studies using similar exercise protocol are warranted to determine the effect of exercise in managing DOMS.

Although there is some evidence that DOMS can be alleviated with ice compression, stretching, massage and other modalities, an efficient treatment strategy has not been established. Treatments suggested so far are inconclusive and the reliability of the data is questionable, which may lead to reliance on unreliable evidence or ineffective treatment (Cheung et al., 2003).

2. Pharmacological Management

One of the main treatments advocated to alleviate signs and symptoms of DOMS is non-steroidal anti-inflammatory drugs (NSAIDs). Despite a strong theoretical basis for efficacy, the majority of studies showed that NSAIDS do not reduce DOMS (Connolly et al., 2003). Ibuprofen and flurbiprofen, for example do not reduce signs and symptoms of DOMS (Donnelly, McCormick, Maughan,

Whiting, & Clarkson, 1988). In contrast, naproxen and diclofenac may act prophylactically as well as therapeutically. O'Gardy et al. (2000) showed that a prophylactic dose of diclofenac (150mg) reduces soreness, swelling, and stiffness after eccentric box stepping exercise in 27 subjects. Similarly, daily administration of 1,000 mg of naproxen for seven days resulted in reduced soreness three days after exercise and enhanced recovery of quadriceps strength relative to placebo group (Lecomte, Lacroix & Montgomery, 1998). The inconsistency in results among the studies might be a consequence of the different eccentric exercise protocols, types of NSAIDs, and the subjective nature of pain perception by the subjects (Connelly et al., 2003).

3. Nutritional Supplements

Nutritional supplements are a popular means with which to combat DOMS, as they generally have no side-effects. The most commonly used nutritional supplements are antioxidants. Free radicals proliferate as a result of exercise-induced muscle damage; a consequence of neutrophil activation and phagocytosis (Pyne, 1994). Hence, supplements that reduce formation of free radicals prior to exercise may act as a preventive measure for reducing DOMS. Two such supplements, vitamin C and E are widely used and Cannon et al. (1989) reported that vitamin E supplementation (400 IU d⁻¹) decreased CK level and accelerate DOMS recovery. In contrast, Jakeman and Maxell (1993) reported that 400 mg d⁻¹ of vitamin E for 21 days before an eccentric exercise bout did not affect DOMS. Given the various methodologies used in studies that have examined the effects of nutritional supplements on DOMS, it is difficult to reconcile data from them.

In summary, multiple treatments have been purported to alleviate symptoms associated with eccentrically-induced muscle damage. The efficacy of

the treatments examined is inconsistent. Further research is warranted in each of the aforementioned areas so that potentially reliable treatments can be identified.

Summary

Unaccustomed exercise, especially eccentric exercise, induces muscle damage and soreness. Signs and symptoms associated with DOMS include swelling, pain, and tenderness as well as reduced muscle force and joint ROM.

The mechanism for DOMS is not fully resolved, although a consensus among researchers is forming. An integrated scheme suggests that high intensity eccentric exercise initiates an acute inflammatory response, coupled with various cellular and sub-cellular events that lead to myofilament damage which together give rise to symptoms associated with DOMS. Future studies are necessary to validate the biochemical and cellular events that initiates DOMS.

Even though the exact mechanism for DOMS is not known, it is clear that two reliable markers are reduced muscle force and joint ROM (Warren et al., 1999). Other markers such as serum muscle proteins and soreness are less reliable and moreover, their time course is not well correlated to changes in muscle function.

Many treatments strategies are purported to manage and prevent DOMS, but results are highly inconsistent. To identify a reliable and consistent treatment strategy, it may be essential to understand the mechanism of injury. It is also possible that methodological issues compromise the interpretation of research on the treatment of DOMS. For example, researchers typically use a high number of repetitions to induce DOMS. Perhaps the resulting muscle damage is too severe for any palliative treatment to be consistently effective. Some palliative treatments may effectively prevent or treat a mild or moderate case of DOMS, but might not be effective with severe DOMS.

Chapter 3

METHODOLOGY

The present study aimed to induce muscle damage by giving three different regimes of eccentric triceps contractions. This chapter describes the methods used to achieve that purpose. The sections included are subjects, experimental design, procedures, measurements, and statistical analyses.

<u>Subjects</u>

Twenty-four untrained college-aged students were recruited for this study. Previous research showed that this sample size is sufficient to elicit significant differences in the dependent variables studied (Brown et al., 1997). Subjects were excluded if they participated in any upper body resistance training in the previous three months for more than twice a week regularly, had a previous arm injury or surgery, or any disease that might affect muscle function. They were also excluded from participation if using any pain medications. Subjects were informed of all experimental procedures and the possible risks and benefits associated with the project. Afterwards, they read and signed an informed consent form (Appendix A) that was approved by the Ithaca College Human Subjects Research (HSR) Board.

Experimental Design

The study was a repeated measure design with measures conducted immediately before (baseline), and 24, 48, 72 and 96 h after the sorenessinducing protocol. Subjects were randomly divided into three equal groups: The following variables were measured: muscle soreness via DDS; arm circumference; relaxed arm angle (RANG); elbow ROM (active flexion); peak torque (PT); and serum CK. Subjects were divided into 20-, 40-, and 60-repetition groups. The triceps brachii muscle of non-dominant arm was used so that

consequences of DOMS were less likely to impact the daily activity of participants. On the first day of data collection, each subject underwent baseline measurements including all of the above followed by the soreness-inducing protocol.

Procedures

1. Baseline Measurement

Prior to baseline measurement, all subjects completed informed consent, health history and health habit (Appendix B), and 24 hour history (Appendix C) forms. All subjects were instructed to avoid upper body strength training while participating in study but could continue leg exercises (e.g., cycling, or running). Baseline measurements of DDS, arm circumference, RANG, ROM, PT, and serum CK were made just prior to the soreness-inducing exercise protocol.

2. Soreness-Inducing Protocol

Subjects in the 20-repetition group performed one set of 20 eccentric triceps contractions, while those in the 40-repetition group completed two sets of 20 repetitions, and those in the 60-repetition group completed three sets of 20 repetitions of maximal eccentric triceps contractions. The protocol of 20, 40, and 60 maximal eccentric contractions is derived from a compilation of the DOMS literature (Lenn et al., 2002; Paulsen et al., 2005). During exercise, subjects started movement from full elbow extension and resisted the lever arm in the direction of extension causing an eccentric triceps contraction. After each contraction, the subject actively extended the arm for next contraction. There was a two-minute rest between each set with no pause between contractions. After the subjects completed the pre-determined number of contractions, postexercise measurements were taken at 24, 48, 72 and 96 h. Measurements were DDS, serum CK, arm circumference (2, 6, and 9 cm), RANG, elbow ROM, and PT.

Afterwards, subjects were given an instruction sheet (Appendix D) that explained how they should maintain their daily routine while participating in the study.

<u>Measurements</u>

The dependent variables for the study were measured in following order: <u>1. DDS</u>

Subjects completed the DDS to assess muscle soreness. The DDS is a reliable and valid measure for assessing pain (Doctor et al., 1994). This instrument applies psychophysical scaling to clinical pain assessment and measures both the sensory and affective components of pain. With the DDS, subjects were asked to grade their clinical pain relative to 12 graded descriptors of pain intensity (sensory dimension) and 12 graded descriptors of pain unpleasantness (affective dimension) (Appendix E).

2. CK

Following DDS administration, blood sampling was done. Blood was drawn (3 ml) from the antecubital vein of the non-exercising arm by a trained phlebotomist. Blood was collected in a serum separator tube, allowed to sit for 20-60 min, and then centrifuged (IEC Centra-MP4R, Needham Heights, MA) at 3,500 rpm for 10 min. Serum was then pipetted in a microtainer tube and stored in a refrigerator. At the end of each day, tubes were transferred for storage to a freezer at -80°C. At end of data collection, all tubes were transported to an outside laboratory (Human Metabolic Research Laboratory, Cornell University, Ithaca, NY) for CK analysis using a Dimension Xpand Plus automated chemistry analyzer (Siemens Medical Diagnostics Solutions, Newark, DE). According to the manufacturer, in this method adenosine monophosphate, and [P1, P5-di (adenosine-5') pentophosphate are added to inhibit adenylate kinase (AK), ethylenediaminetetraacetic acid (EDTA) is added to suppress calcium inhibition of CK; dithioerythritol (DTE) is added to activate CK; 2-N-(morpholino) ethane

sulfonic acid (MES) is used as the buffer, and the reagents were simultaneously optimized for maximum activity. Sample and reagent were mixed and values were recorded in U·L⁻¹. Duplicates were run for the test to inspect for error.

3. Arm Circumference

Measurement of the non-dominant arm circumference was completed using a standard anthropometric tape. Subjects were standing with the arm in anatomical zero position, relaxed and hanging loosely. Measurements were performed at 2, 6, and 9 cm with the medial humeral epicondyle as the reference point. Three measurements were done at each point and averaged. Permanent ink marks were made at each point to maintain consistency over trials.

<u>4. RANG</u>

RANG measurement was done with the subject in standing position and arm by side, with the universal goniometer. The reference points for the measurement of the angle include: the lateral humeral epicondyle, acromion process, and midpoint on wrist. Three measurements were done and averaged for recording. A permanent ink marker was used to maintain consistency over trials.

5. Elbow ROM

Elbow ROM was measured with a standardized goniometer for active flexion range. The active flexion ROM was determined as the difference between the actively flexed and extended elbow joint angles. Subjects were tested in the supine position to stabilize the shoulder and upper body. The fulcrum of goniometer was kept at the lateral epicondyle of the humerus, while the stationary arm was parallel to longitudinal axis of humerus and the movement arm was parallel to the longitudinal axis of forearm. Measurement was done from a fully extended position to a fully flexed position (Zainuddin et al., 2005). Placement locations of fulcrum, stationary and movement arm were marked with permanent ink to maintain consistency throughout trials. Three measurements were taken and the mean value was used for analysis.

6. Peak Torque

To measure PT, subjects completed eccentric elbow extension on a Cybex isokinetic dynamometer (Computer Sports Medicine, Inc. Humac®/Norm™, Model 770, Stoughton, MA). Prior to any Cybex test, the machine was adjusted according to settings made during the baseline lab visit. In addition, every PT test was preceded by a standardized warm-up that included five sub-maximal and two maximal eccentric triceps contractions at 90° s⁻¹. After the warm-up, the subjects rested two minutes before completing three maximal eccentric triceps contractions was recorded as PT in Newton-meters. A pilot study performed to test the reliability of the PT protocol yielded a coefficient of variation of 4.3% (n=21).

Statistical Analyses

A 3 x 5 (Condition x Time) repeated measures analysis of variance (RM ANOVA) was done for each dependent variable: DDS (sensation and unpleasantness), CK, arm circumference (2, 6, and 9 cm), RANG, ROM, and PT. The three conditions corresponded to the three groups, who completed 20, 40, and 60 repetitions, respectively, while the five time periods corresponded to the measurement points at 0, 24, 48, 72, and 96 h. Where indicated, significant differences among groups were located with Bonferroni post-hoc analyses. To further examine the research hypotheses, additional analyses included a one-way ANOVA, which was used to compare groups at baseline to look for initial group differences. If no differences were found for a dependent variable at baseline, data for that variable, for each group across all time periods excluding

baseline, were collapsed to assess overall group differences. The alpha level for all tests, except for Bonferroni, was set at 0.05.

Chapter 4

RESULTS

The purpose of this study was to manipulate the extent of DOMS. For this, 24 subjects were randomly divided in three groups. The groups performed 20, 40, or 60 maximal eccentric triceps contractions using a Cybex dynamometer. The dependent variables measured at the baseline and 24, 48, 72, and 96 h post-soreness induction were arm circumference at 2, 6, and 9 cm, RANG, ROM, PT, soreness, and CK. The Appendix G contains raw data for all the variables. This chapter includes results analyzing all dependent variables and includes the following subsections: Subject characteristics; Peak torque; Arm circumference (2 cm); Arm circumference (6 cm); Arm circumference (9 cm); RANG; ROM; DDS (unpleasantness); CK.; and Summary.

Subject Characteristics

Age, height and weight were recorded on the first day of testing and are reported in Table 1. The mean age for subjects across groups was similar as all were recruited from a cohort of college students. No significant difference between groups was found with respect to height or weight according to two-tailed, two-sample, equal variance t-tests (p > 0.05).

Peak Torque

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in PT among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(4.3, 45.2)}$ = 2.2, p > 0.05] was observed indicating that groups behaved similarly across time for PT (Table 2)

Table 1

Subject Characteristics

Subject	Age(years)	Height(cm)	Weight(kg)
Group A (n=8)	18.3 ± 0.7	169.1 ± 10.8	72.2 ± 10.1
Group B (n=8)	19.8 ± 3.4	169.3 ± 6.8	71.1 ± 8.4
Group C (n=8)	19.1 ± 1.3	160.5 ± 10.8	64.5 ± 17.1

Note: Groups differ by repetition number with A=20, B=40, and C=60; Values are mean \pm SD.

Table 2

ì

, **F**

Peak Torc	que: ANOVA	Summar	/ Table

Source	SS	_ df	MS	F	p
Time	5800.1	2.2	2692.2	28.5	0.000*
Error (time)	4278.6	45.2	94.6		
Group	10580.1	2.0	5290.0	3.5	0.049*
Error (group)	31919.9	21.0	5290.1		
Group x Time	899.6	4.3	208.8	2.2	0.079

Note: * denotes significance (p < 0.05)

There was, however, a significant time main effect found for PT [$F_{(2.2, 45.2)}$ = 28.46, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustment to compare means among time points. The comparison revealed a significant decrease in torque after baseline (p < 0.05) at all four subsequent times. Examining Figure 1 it can be seen that reduction in torque is maximum at 48 h (37.5 ± 19.6 Nm) when compared with baseline (56.3 ± 17.9 Nm). The torque was significantly (p < 0.05) lesser than baseline (56.3 ± 17.9 Nm) at 72 and 96 h (39.25 ± 21.4 and 41.7 ± 24.5 Nm, respectively). These data showed that the recovery of PT after muscle damage induced by eccentric exercise took longer than 96 hours.

There was also a significant group main effect found for PT [F (2.0, 21.0) = 3.48, p < 0.05] using a 3 x 5 RMANOVA. This indicates that the reduction in PT was not similar among the groups. A post-hoc t-test with Bonferroni adjustments was done to identify the difference among the groups. The analyses revealed a significant difference only between the 20- and 60-repetition groups (p < 0.05) (Figure 1). The 20-repetition group had more strength (54.2 ± 24.4 Nm) than 60repetition group $(31.2 \pm 14.88 \text{ Nm})$ suggesting that subjects who did greater repetitions had a greater loss in strength. To examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for PT at baseline yielded non-significant results [F (2.0, 21.0) = 0.75, p > 0.05]. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing the four postexercise time points excluding the baseline. ANOVA results were significant between groups [F (2.0, $_{93.01}$ = 17.07, p < 0.05] demonstrating a significant difference in PT for at least one of the between-group analysis. Post-hoc t-tests with Bonferroni adjustment



Figure 1. Mean peak torque for each group at all times: * Denotes that torque was significantly different (p < 0.05) from baseline at each time point: + Denotes that torque is significantly different (p < 0.05) between 20- and 60-repetition groups.

were significant (p < 0.05) and figure 2 shows that the 20-repetition group produced greater PT than both the 40-repetition and 60-repetition groups. Similarly, the 40-repetition group produced greater PT than 60-repetition group. Accordingly, it appears that as the number of maximal eccentric contractions increases it causes greater muscle damage and subsequent strength loss. Examining PT across the four days post-soreness induction, the 20-repetition group produced about 25.9% greater PT than 40-repetition group and 49.8 % greater PT than 60-repetition group. Therefore, eccentrically-induced muscle damage as measured by PT can be differentiated into three discrete levels depending on the number of eccentric contractions performed.

Arm Circumference (2 cm)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in arm circumference (2 cm) among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(4.1, 42.8)} = 1.73$, p > 0.05] was observed indicating that groups behaved similarly across time for arm circumference (2 cm) (Table 3).

There was, however, a significant time main effect found for arm circumference (2 cm) [$F_{(2.0, 42.8)}$ =22.89, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustments to compare means among time points. The comparisons revealed a significant (p < 0.05) increase in arm circumference (2 cm) compared to baseline at all four subsequent times. Examining Figure 3 it can be seen that the increase in arm circumference (2 cm) is greatest (26.5 ± 2.4 cm) at 48 h (p < 0.05) when compared with baseline (25.8 ± 2.3 cm). The arm circumference (2 cm) was also significantly (p < 0.05) greater than baseline (25.8 ± 2.3 cm) at 72 and 96 h (26.4 ± 2.4 and 26.3 ± 2.3 cm, respectively). These data



Figure 2. Peak torque after collapsing all time intervals excluding baseline measurement for each group: + Denotes significant difference (p < 0.05) for torque between 20 and 40 repetitions group: * Denotes significant difference (p < 0.05) for torque between 20 and 60 repetitions group: and Δ Denotes significant difference (p < 0.05) for torque between 40 and 60 repetitions group.

Source	SS	df	MS	F	p
Time	6.5	2.1	3.2	22.9	0.000*
Error (time)	5.9	42.8	0.1		
Group	49.4	2.0	24.7	0.9	0.430
Error (group)	590.2	21.0	28.1		
Group x Time	0.9	4.1	0.2	1.7	0.160

Arm Circumference (2 cm): ANOVA Summary Table

Note: * denotes significance (p < 0.05)



Figure 3. Mean arm circumference (2 cm) for each group at all times: * Denotes that arm circumference was significantly different (p < 0.05) from baseline at each time point.

show that edema persists for more than 96 h, irrespective of the how many maximal eccentric contractions were performed.

There was no significant group main effect found for arm circumference (2 cm) [F value (2.0, 21.0) = 0.88, p > 0.05]. This leads to a conclusion that arm circumference (2 cm) was similar for the groups throughout the study. However, to examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for arm circumference (2 cm) at baseline yielded non-significant results [$F_{(2.0, 21.0)} = 1.42$, p > 0.05]. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing the four postexercise time points excluding baseline. ANOVA results were significant [$F_{(2,0,93,0)} = 3.34$, p < 0.05] demonstrating a significant difference in arm circumference (2 cm) for at least one between-group analysis. Post-hoc t-tests with Bonferroni adjustment were significant (p < 0.05) and figure 4 shows that the 60-repetiton group had greater swelling than 20 repetition group. Accordingly, it appears that 60 maximal eccentric contraction causes greater increase in arm circumference and therefore swelling. Examining arm circumference across the four days post-soreness induction period 20repetiton group had 5.36% less swelling than 60-repetition group. Therefore, eccentrically produced muscle damage as measured by arm circumference (2 cm) can be differentiated into two discrete levels for 20- and 60-repetition groups; however, 40-repetition did not yield significantly discrete results compared to 20or 60-repetitions.

Arm Circumference (6 cm)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in arm circumference (6 cm) among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-



Figure 4. Arm circumference (2 cm) after collapsing all time intervals excluding baseline measurement for each group: * Denotes significant difference (p < 0.05) for arm circumference (2 cm) between 20- and 60-repetition group.

Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(3.4, 35.3)} = 1.09$, p > 0.05] was observed indicating that groups behaved similarly across time for arm circumference (6 cm) (Table 4).

There was, however, a significant time main effect found for arm circumference (6 cm) [$F_{(1.7, 35.3)} = 23.19$, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustment to compare means among time points. The comparisons revealed a significant increase in arm circumference (6 cm) compared to baseline (p < 0.05) at all four subsequent times. Examining Figure 5 it can be seen that the increase in arm circumference (6 cm) is greatest (28.3 ± 2.7 cm) at 48 h (p < 0.05) when compared with baseline (27.7 ± 2.8 cm). The arm circumference (6 cm) was also significantly (p < 0.05) greater than baseline (27.7 ± 2.8 cm) at 72 and 96 h (28.2 ± 2.8 and 28.2 ± 2.7 cm, respectively). These data show that edema persists for more than 96 h, irrespective of the how many maximal eccentric contractions were performed.

There was no significant group main effect found for arm circumference (6 cm) [*F* value (2.0, 21.0) = 0.53, $\rho > 0.05$]. This leads to a conclusion that arm circumference (6 cm) was similar for the groups throughout the study. To examine research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for arm circumference (6 cm) at baseline yielded a non-significant results [*F* (2.0, 21.0) = 0.72, $\rho > 0.05$]. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing the four postexercise time points excluding baseline. ANOVA results were not significant between group [*F* (2.0, 93.0) = 2.13, $\rho > 0.05$]. Accordingly, it appears that groups had similar swelling and number of contractions did not impact the amount of arm swelling when measured at 6 cm.

Table 4

					· ·	
Source	SS	df	MS	F	р	
Time	5.5	1.7	3.3	23.2	0.000*	
Error (time)	5.1	35.3	0.1			
Group	43.0	2.0	21.5	0.5	0.597	
Error (group)	852.6	21.0	28.1			
Group x Time	0.5	3.4	0.2	1.1	0.368	

Arm Circumference (6 cm): ANOVA Summary Table

Note: * denotes significance (p < 0.05)



ï

Figure 5. Mean arm circumference (6 cm) for each group at all times: * Denotes that arm circumference (6 cm) was significantly different (p < 0.05) from baseline at each time point.

Arm Circumference (9 cm)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in arm circumference (9 cm) among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus for this variable Greenhouse-Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(4.6, 48.4)} = 1.47$, p > 0.05] was observed indicating that groups behaved similarly across time for arm circumference (9 cm) (Table 5).

There was, however, a significant time-main effect found for arm circumference (9 cm) [$F_{(2.3, 48.4)} = 11.15$, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustment to compare means among time points. The comparison revealed a significant increase in arm circumference (9 cm) after baseline value (p < 0.05) for all four subsequent time measures. Examining Figure 6 it can be seen that the arm circumference (9 cm) is greatest (29.6 ± 2.9 cm) at 48 h (p < 0.05) when compared with baseline (29.1 ± 3.0 cm). The arm circumference was also significantly (p < 0.05) greater than baseline (29.1 ± 3.0 cm) at 72 and 96 h (29.5 ± 3.0 cm and 29.4 ± 2.9 cm, respectively). These data showed that edema persists for more than 96 h, irrespective of the how many maximal eccentric contractions were performed.

ੁ

There was no significant group-main effect found for arm circumference (9 cm) [*F* value _(2.0, 21.0) = 0.5, p > 0.05]. This leads to a conclusion that arm circumference (9 cm) was similar for the groups throughout the study. To further examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for arm circumference (9 cm) at baseline yielded a non-significant results [*F* _(2.0, 21.0) = 0.6, p > 0.05]. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing the four post-exercise time points excluding baseline.

Table 5

Ş

7

- y i						
Source	SS	df	MS	F	р	
Time	3.8	2.3	1.7	11.2	0.004*	
Error (time)	.7.2	48.4	0.2			
Group	41.9	2.0	20.9	0.5	0.646	
Error (group)	985.7	21.0	46.9			
Group x Time	1.0	4.6	0.2	1.5	0.200	

Arm Circumference (9 cm): ANOVA Summary Table

Note: * denotes significance (p < 0.05)



Figure 6. Mean arm circumference (9 cm) for each group at all times: * Denotes that arm circumference is significantly different (p < 0.05) from baseline at each time point.

ANOVA results were not significant for group [$F_{(2.0, 93.0)} = 1.77$, p > 0.05]. Accordingly, it appears that groups had similar swelling and number of contractions does not impact the amount of arm swelling when measured at 9 cm.

Relaxed Arm Angle (RANG)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences RANG among three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-Geisser was used to calculate the Group x Time interaction effect. A significant interaction [$F_{(5.2, 54.7)}$ = 4.16, p < 0.05] was observed indicating that groups behaved differently across time for RANG (Table 6). A univariate ANOVA was done to identify group differences at each time point. No significant differences were found between groups for any of the time points (p > 0.05). Perhaps the statistical power was not large enough, or variability in data was too great to locate any significant differences.

There was also a significant time main effect found for RANG $[F_{(2.6, 54.7)} = 20.8, p < 0.05]$. Pairwise comparisons were done with Bonferroni adjustment to compare group means among time points. The comparisons revealed a significant increase in RANG compared to baseline (p < 0.05) at all four subsequent times. Examining Figure 7 it can be seen that the RANG was greatest (24.9 ± 4.6 degrees) at 48 h (p < 0.05) when compared with baseline (22.7 ± 4.6 degrees). The RANG was also significantly (p < 0.05) greater than baseline (22.7 ± 4.6 degrees) at 72 and 96 h (24.2 ± 4.9 and 23.9 ± 5.1 degrees, respectively). These data show showed that stiffness and swelling associated with eccentric exercise persists and complete recovery takes longer than 96 h.

Table 6

1

RANG: ANOVA Summary Table

Source	SS	df	MS	F	p
Time	70.5	2.5	27.5	22.0	0.000*
Error (time)	70.2	54.7	1.3		
Group	217.6	2.0	108.8	0.9	0.405
Error (group)	2421.1	21.0	115.3		
Group x Time	27.9	5.2	5.3	4.2	0.002*

Note: * denotes significance (p < 0.05)



Figure 7. Mean RANG for each group at all times: * Denotes that RANG is significantly different (p < 0.05) from baseline at each time point.

There was no significant group main effect found for RANG [F value (2.0. $_{21.01}$ = 0.94, p > 0.05]. This indicates that increase in RANG was similar between groups. To examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for RANG at baseline yielded non-significant results [$F_{(2.0, 21.0)} = 0.45$, p > 0.05]. Follow-up analysis using univariate ANOVA for group differences were then done after collapsing all the four postexercise time points excluding baseline. ANOVA results were significant for group [$F_{(2.0, 93.0)} = 4.7$, p < 0.05] demonstrating that there was a significant difference in RANG for at least one between-group analysis. Post-hoc t-tests with Bonferroni adjustments were significant (p < 0.05) and figure 8 shows that the 20-repetition group had a smaller increase in RANG than the 40- and 60-repetitions group. Accordingly, it appears that as the number of maximal eccentric contraction increases, so does muscle stiffness and therefore RANG. The change in RANG across four day post-soreness induction for the 20 repetition group was approximately 12 % lower than the changes in the 40- and 60-repetition groups. Therefore, eccentrically-induced muscle damage can be differentiated into two discrete levels based on the manipulations used in this study.

Elbow Range of Motion (ROM)

A 3 x 5 (Group x time) RM ANOVA with repeated measures on time was done to identify differences in ROM between the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(4.0, 42.2)}$ = 1.2, p > 0.05] was observed indicating that groups behaved similar across time for ROM (Table 7).



Figure 8. RANG (degrees) after collapsing all time intervals excluding baseline measurement for each group: Δ Denotes significant difference (p < 0.05) for RANG between 20- and 40-repetition group: * Denotes significant difference (p < 0.05) for RANG between 20- and 60-repetition group.

ROM: ANOVA Summary Table

and the second						
Source	SS	df	MS	F	p	
Time	536.8	2.0	267.1	14.3	0.000*	
Error (time)	789.8	42.2	18.7		•	
Group	527.6	2.0	263.8	0.9	0.430	
Error (group)	6294.5	21.0	299.7			
Group x Time	90.6	4.1	22.5	1.2	0.323	

Ç.

×

Note: * denotes significance (p < 0.05)

There was, however, a significant time main effect found for ROM [$F_{(2.0, 42.2)} = 14.3$, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustment to compare means among time points. The comparison revealed a significant decrease in ROM compared to baseline (p < 0.05) for all four subsequent time measures. Examining Figure 9 it can be seen that the reduction in ROM was greatest (136.3 ± 9.5 degrees) at 48 h (p < 0.05) when compared with baseline (142.5 ± 8.0 degrees). The ROM was also significantly (p < 0.05) lesser than baseline (142.5 ± 8.0 degrees) at 72 and 96 h (138.4 ± 7.2 and 139.4 ± 6.5 degrees, respectively). These results showed that stiffness associate with eccentric exercise persists and complete recovery takes longer than 96 h.

There was no significant group main effect found for ROM [F (2.0, 21.0) = 0.88, p > 0.05]. This indicates that reduction in ROM was similar for the groups throughout the study. To examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group. differences for ROM at baseline yielded non-significant results [$F_{(2.0, 21.0)} = 0.11$, p > 0.051. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing all time points except baseline. The ANOVA results were significant between groups [$F_{(2.0, 93.0)} = 4.5$, p < 0.05] demonstrating that there was a significant difference in ROM for atleast one between-group analysis. A post-hoc t-test with Bonferroni adjustment was significant (p < 0.05) and figure 10 shows that 60-repetiton group had a larger reduction in ROM than 20 repetition group. Accordingly, it appears that as the number of maximal eccentric contraction increases, so does muscle stiffness, which leads to greater reduction in ROM. Examining ROM across the four day post-soreness induction period, ROM was 4.18 % high in the 20-repetition group than 60-repetition group.



Figure 9. Mean ROM for each group at all times: * Denotes that ROM is significantly different (p < 0.05) from baseline at each time point


Figure 10. ROM after collapsing all time intervals excluding baseline measurement for each group: * Denotes significant difference (p < 0.05) for ROM between 20- and 60- repetition group.

Therefore, eccentrically produced muscle damage as measured by ROM can be divided into only two discrete levels as based on the manipulations in this study.

DDS (Sensation)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in DDS sensation among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus Greenhouse-Geisser was used to calculate the Group x Time interaction effects. No significant interaction [$F_{(5.7, 59.5)} = 0.59$, p > 0.05] was observed indicating that groups behaved similar across time for DDS (sensation) (Table 8).

There was, however, a significant time main effect found for sensation [*F* $_{(2.8, 59.5)} = 33.3$, p < 0.05]. Pairwise comparisons were done with Bonferroni adjustment to compare means between time points. The comparisons revealed a significant increase in sensation compared to baseline value (p < 0.05) for all four subsequent time measures. Examining Figure 11 it can be seen that the difference \pm SE (5.6 ± 0.66) in sensation was greatest (5.6 ± 3.2) at 48 h (p < 0.05) when compared with baseline (0.0 ± 0.0). The sensation was significantly (p < 0.05) greater then baseline at 72 and 96 h (4.1 ± 3.0 and 2.9 ± 2.7 , respectively). These data showed that pain persists longer than 96 h after eccentric exercise.

There was no significant group main effect found for sensation [$F_{(2.0, 21.0)} = 0.47$, p > 0.05]. This indicates that sensation was similar for the groups throughout the study. To further examine this research hypothesis more thoroughly, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. All the subjects had no pain sensation prior to exercise session; hence groups were same at the baseline. A univariate ANOVA for group differences were then done after

DDS (Sensation): ANOVA Summary Table

C					•	
Source	55	ar	WIS	F	p	
Time	449.1	2.8	158.5	33.4	0.000*	
Error (time)	282.5	59.5	4.7			
Group	20.6	2.0	10.3	0.5	0.632	
Error (group)	462.4	21:0	22.1			
Group x Time	15.9	5.6	2.8	0.6	0.726	

Note: * denotes significance (p < 0.05)



Figure 11. Mean DDS (sensation) for each group at all times: * Denotes that sensation is significantly different (p < 0.05) from baseline at each time point.

collapsing the four postexercise time points excluding baseline. The ANOVA results were not significant between groups [$F_{(2.0, 93.0)} = 1.2$, p > 0.05]. Thus, the number of muscle contractions did not affect the sensation of muscle soreness.

DDS (Unpleasantness)

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in DDS (unpleasantness) among the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus for this variable Greenhouse-Geisser was used to calculate Group x Time interaction effect. No significant interaction [$F_{(4.6, 48.6)} = 1.3$, p > 0.05] was observed indicating that groups behaved similar across time for DDS (unpleasantness) (Table 9).

There was, however, a significant time main effect found for unpleasantness [$F_{(2.3, 48.6)} = 9.6$, p < 0.05]. Pairwise comparisons were done to compare means between time points. The comparison revealed a significant increase in unpleasantness after baseline (p < 0.05) at all four subsequent time measures. Examining Figure 12 it can be seen that the unpleasantness was greatest (3.5 ± 4.3) at 48 h (p < 0.05) when compared with baseline (0.0 ± 0.0). The unpleasantness was significantly (p < 0.05) greater (2.5 ± 3.3) at 72 h, but not at 96 h (1.7 ± 3.2) when compared with baseline. This data showed that the unpleasantness associated with eccentric exercise resolved in 72 hours.

There was no significant group main effect found for unpleasantness [$F_{(2.0, 21.0)} = 2.4, p > 0.05$]. This indicates that sensation was similar for the groups throughout the study. To further examine the research hypothesis, it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. All the subjects had no unpleasantness prior to the soreness-induction exercise session; hence groups were the same at the baseline. A univariate ANOVA for group differences were done after collapsing the four postexercise time points excluding baseline. ANOVA results were

Table 9

Source	SS	df	MS	F	p
Time	175.4	2.3	75.8	9.6	0.000*
Error (time)	282.5	59.5	4.7		· ·
Group	148.5	2.0	74.3	2.4	0.117
Error (group)	462.4	21.0	22.1		
Group x Time	48.5	4.6	10.5	1.3	0.270
		•	, .		

DDS (Unpleasantness): ANOVA Summary Table

Note: * denotes significance (p < 0.05)



Figure 12. Mean DDS (unpleasantness) for each group at all times: * Denotes that unpleasantness is significantly different (p < 0.05) from baseline at each time point but 96 h.

significant between groups [$F_{(2.0, 93.0)} = 7.9$, p < 0.05] demonstrating that there was a significant difference in ROM for atleast one between-group analysis. Post-hoc t-tests with Bonferroni adjustment were significant (p < 0.05) and Figure 13 shows that 60-repetitons produced greater unpleasantness than seen in the 20-repetition group. Accordingly, it appears that as the number of maximal eccentric contraction increases it causes greater unpleasantness and mood change. Examining unpleasantness across the four days post-soreness induction, the unpleasantness was 75 % lower in the 20 repetition group than the 60- repetition group. Therefore, eccentrically produced muscle damage as measured by DDS (unpleasantness) can be divided into only two discrete levels based on the manipulations used in this study.

Creatine Kinase

A 3 x 5 (Group x Time) RM ANOVA with repeated measures on time was done to identify differences in CK between the three groups. Mauchly's test of sphericity was significant (p < 0.05) and thus for this variable Greenhouse-Geisser was used to calculate the Group x Time interaction effect. No significant interaction [$F_{(2.5, 25.6)} = 1.5$, p > 0.05] was observed indicating that groups behaved similar across time for CK (Table 10).

There was no significant time main effect found for CK [$F_{(1.3, 25.6)} = 2.97$, p > 0.05]. Examining Figure 14 it can be seen that no significant difference in CK was found across time. This lack of significance despite of upward trend might be explained by large standard deviation seen in CK at each point.

There was no significant group main effect found for CK [$F_{(2.0, 21.0)} = 1.38$, p > 0.05]. This indicates that CK is similar between groups throughout the study.



Figure 13. DDS (unpleasantness) after collapsing all time intervals excluding baseline measurement for each group: * Denotes significant difference (p < 0.05) for DDS (unpleasantness) between 20- and 60-repetition groups.

Table 10

CK: ANOVA Summary Table

		-			
Source	SS	df	MS	F	р
Time	53691586.6	1.3	41938288.2	2.9	0.088
Error (time)	3.6	25.6	14076612.9		
Group	1.0	2.0	50038462.7	1.4	0.274
Error (group)	7.2	20.0	36159417.2		
Group x Time	55044383.9	2.5	21497476.4	1.5	0.234

Note: no significant difference (p > 0.05)



Figure 14. CK (I·L⁻¹) for three groups at each time interval.

To examine this research hypothesis more thoroughly: it was deemed necessary to further inspect group differences by eliminating the averaging effect of baseline on group data. A one-way ANOVA examining group differences for CK at baseline yielded a non-significant results [$F_{(2,0,21,0)} = 0.12$, p > 0.05]. Follow-up analysis using univariate ANOVA for group differences was then done after collapsing the four postexercise time points excluding baseline. ANOVA results were significant between groups [$F_{(2.0, 93.0)} = 5.06$, (p < 0.05) demonstrating that there was a significant difference in CK for atleast one between-group analyses. Post-hoc t-tests with Bonferroni adjustments were significant (p < 0.05) and Figure 15 shows that 60-repetiton group had a greater increase in CK than 20repetition and 40-repetition groups. However, there were no significant (p > 0.05) differences between 20- and 40-repetitions group. Accordingly, it appears that a great number of maximal eccentric contractions causes muscle damage and therefore significantly greater CK levels. Examining CK across the four day postsoreness induction period, it is seen that the CK levels were 91% and 77% higher in the 60-repetition group compared to 20- and 40-repetition groups, respectively. Therefore, eccentrically produced muscle damage as measured by CK can be divided into two discrete levels as based on the manipulations used in this study.

Summary

We studied the effect of three different volumes of eccentric exercises on physical measures as well as soreness-rating and CK levels. The variables showed varying sensitive to three exercise protocols. PT is most sensitive as all three groups were significantly different following the soreness-inducing protocol. All other physical measures were not sensitive enough to allow for three discrete level of soreness to be detected. We did not find any group difference in



Figure 15. CK (I·L⁻¹) after collapsing all time intervals excluding baseline measurement for each group: * Denotes significant difference (p < 0.05) for CK between 20 and 60 repetitions group: \triangle Denotes significant difference (p < 0.05) for CK between 40 and 60 repetitions group.

sensation ratings, while there was difference in unpleasantness between 20- and 60-repetiton groups. A significant time-main effect was found for all the variables, but CK. The change in most variables peaks at 48h and begin to reduce thereafter. Variability in CK can be attributed to large standard deviations in each group. There was not any significant time-main effect observed, however CK was most pronounced in 60-repetiton group and kept on increasing until 96 h. Hence, it can be concluded that some variables are more sensitive to eccentric exercise volume than others and DOMS can be differentiated into discrete levels using certain variables with PT apparently being the most useful.

Chapter 5

DISCUSSION

Damage to skeletal muscle after novel eccentric exercise is well documented (Armstrong, 1984). Muscle fatigue, chronic force loss, pain, swelling, and leakage of muscle-specific enzyme (e.g. CK) are common following unaccustomed eccentric exercise. The purpose of the present study was to attempt to manipulate the extent of DOMS by varying the exercise volume (i.e. number of eccentric contractions). The dependent variables measured were each significantly changed after the soreness-inducing protocol. Moreover, varying the number of eccentric contractions did not affect all variables equally. This chapter discusses these results in following subsections; 1) Varying repetition number and physical measures; 2) Varying repetition number and soreness rating; 3) Varying repetition number and CK; 4) Time course of soreness-induction; 5) Practical implications; and 6) Summary.

Varying Repetition Number and Physical Measures

The primary aim of this study was to see if one could separate physical measures that describe DOMS into three discrete levels depending on the number of eccentric repetitions performed by the subjects. PT was a primary variable of interest as soreness-induced muscle damage is directly correlated with the loss in muscle strength (Warren et al., 1999). I observed a significant decrement (13.6%, 32.9%, and 47.3% with 20-, 40- and 60-repetitions, respectively) when compared with baseline in the eccentric strength of the triceps following the eccentric exercise protocol. This PT decrement following soreness-induction corresponded well with previous literature on DOMS (Brown et al., 1997; Cleak & Eston, 1992; Lenn et al., 2005; & Paddon-Jones et al., 1997).

Most previous studies used one exercise intensity to cause and/or attempt to manage DOMS (Lenn et al., 2001; Paddon-Jones et al., 1997; Zainuddin et al., 2005). Brown et al. (1997), however, examined the effect of three intensities of eccentric exercise and found that maximum voluntary contraction was most affected after greater exercise effort. The present study agreed with Brown et al. (1997) in that, as the amount of eccentric exercise increased, the more PT was reduced. In the four days after soreness induction, there was 47.3% loss after 60-repetitions, a 32.9% loss after 40-repetitions and only a 13.6% loss in PT after 20-repetitons. Hence, the decrement in PT was directly related to the amount of novel eccentric work performed in a near linear fashion.

The 47.3% decrement in strength following 60-repititions in this study is similar to the 46% reduction in maximum voluntary contraction following 50 maximal eccentric contractions of elbow flexors measured by Prasartwuth et al. (2005). Zainuddin et al. (2005) found a 60% reduction in isometric strength immediately after 60 maximal eccentric biceps contractions, which remained low for the following 48 h. So the present average loss of 47.3% over four days in the 60-repetition group is consistent with previous results. Slight differences between present result and the work of Zainuddin et al. may be attributed to using different muscle group (i.e., triceps vs. biceps) and the type of contraction (i.e., eccentric vs. isometric) used to cause muscle injury. In summary, the extent of muscle damage and subsequent loss in PT is directly influenced by the magnitude of eccentric exercise used to cause DOMS.

DOMS is also associated with significant increases in swelling, stiffness, which increase RANG and decreased ROM (Armstrong, 1984). Following eccentric exercise an inflammatory reaction in muscle causes the accumulation of inflammatory substances and subsequent edema (Smith, 1990). A significant increase in arm circumference was found across all eccentric-exercise groups.

Swelling at 2 cm from the lateral epicondyle of the humerus was significantly different between 20-and 60-repetition groups. This suggests that greater eccentric effort (i.e., 60-repetition) leads to greater muscle damage and leakage of inflammatory substance, and therefore, more edema than fewer repetitions. The arm circumference or swelling measure was, however, less sensitive to variations in exercise intensity than PT, could not be differentiated as arm swelling into three discrete levels. Swelling may be a less sensitive measure than PT because it involves an inflammatory reaction in muscle as well as in the perimuscular connective tissues (Cheung et al., 2003). Hence, swelling occurred secondary to muscle damage.

In contrast to the arm circumference measure at 2 cm, there were no differences in arm circumference among the groups when measured at 6 and 9 cm from the lateral epicondyle of humerus. The arm circumference data from this study suggest that only the 2 cm site can be used to distinguish levels of muscle damage (i.e., severe and mild) associated with high and low amounts of novel eccentric exercise.

A between groups analysis for RANG revealed results similar to swelling. There was a significant difference in RANG between the 20- and 40-, and 20-and 60-repitition groups, but no difference between the 40- and 60- repetition groups. This finding suggests that the higher repetition groups adopted a more flexed posture and likely suffered greater discomfort while relaxing the arm, which indicates that they suffered more swelling. The peak change in RANG was 3.6 degree decrease at 48 h after 60 maximal eccentric triceps contractions. In contrast, Cleak and Eston (1992) found a 26 degree decrease in RANG after 70 maximal eccentric elbow flexor contractions at 96 hours. The difference in results between the studies may be due to using a slightly less intense protocol (i.e., 60 maximal eccentric contractions) for a different muscle (i.e., triceps). In addition,

in present study subjects did their eccentric exercise on a Cybex isokinetic dynamometer with constant speed of 90° s⁻¹, whereas Cleak and Eston (1992) had their subjects manually lower their forearm against resistance.

The present study also found that 60-repetitions caused a greater reduction in ROM than 20-repetitions, which also supports the contention that more repetitions caused greater inflammation and sensory stimulation than fewer repetitions. Similar to arm swelling, no difference was observed between the 40and 20- and 40- and 60- repetition groups, suggesting that ROM was also comparatively less sensitive than PT in discerning difference among groups that completed various amounts of exercise. In all, arm swelling, RANG, and ROM are capable of distinguishing between mild and severe DOMS-induced muscle damage. These variables are not as sensitive as PT in differentiating moderate levels of muscle damage from varying levels of insult.

Varying Repetition Number and Soreness Rating

Muscle soreness following a novel eccentric exercise protocol is well documented (Armstrong, 1984). Previous research consistently shows increased pain ratings following various soreness-induction protocols (Cleak and Eston, 1992; Friden et al., 1986; Lenn et al., 2002; & Zainuddin et al., 2005). In this study the DDS scale was used to assess arm sensation and unpleasantness associated with eccentric-exercise induced soreness. Surprisingly, there were no group differences for sensation of soreness, suggesting that all subjects, regardless of exercise protocol, had similar pain sensations. The degree of discomfort was similar to that reported by Hilbert et al. (2003), who measured muscle soreness in subjects who completed eccentric hamstring curls. In contrast to the present study, Brown et al. (1997) found that subjects who performed greater repetitions had greater soreness. The reason for inconsistency between the studies may be the different method of soreness rating used. Brown

et al. used an analog measure of muscle soreness, whereas we used the DDS, which is a more complicated rating scale. Perhaps subjects had difficulty using the DDS leading to poor sensation rating.

Unlike the sensation of soreness, we found that there was a significant difference in soreness unpleasantness between the 20- and 60-repetition groups, as the subjects who did more repetitions had greater unpleasantness than subjects who did fewer repetitions. Similar to arm swelling, RANG, and ROM, the unpleasantness measure was not fully sensitive to exercise intensity, as no difference was observed between the 40-repetition group and the 20- and 60-repetition groups. The greater psychological disturbances associated with higher repetitions may be attributed to limitations in activities of daily living associated with triceps swelling and reduced ROM.

Varying Repetition Number and Creatine Kinase

Many studies have assessed the presence of muscle-specific proteins in the blood following an unaccustomed maximal eccentric exercise protocol. CK is considered the most accepted indirect enzymatic, marker of muscle damage and is widely analyzed in DOMS related research (Brancaccio et al., 2007; Brown et al., 1997; Lenn et al., 2002; Malm et al., 2004; Paulsen et al., 2005; Zainuddin et al., 2005). A significant group difference was found across the four days after soreness induction, with CK activity greatest after 60-repetitions compared to 20and 40- repetitions. These data suggest that only the greatest effort (i.e., 60repetitions) resulted in more muscle damage, thereby leading to leakage of CK into the blood. A study by Brown et al. (1997) showed that there was significant increase in CK levels after 30 and 50 maximal eccentric quadriceps contractions but not after 10 repetitions. Despite some inconsistency in results, these studies indicate that only higher volume of eccentric exercise leads to significant muscle damage and subsequent CK leakage.

CK level did not resolve by 96 hours after our soreness-induction protocol in this study, which agrees with other research (Brancaccio et al., 2007; Paulsen et al.,2005). Collectively data from these and the current study show that peak CK occurs 96 hours after heavy soreness-induction session. Other studies have found a biphasic pattern for changes in CK levels after eccentric exercise; initially CK levels increase and then decline slightly in the next 47 hours. They then rise again, peaking at 96 hours (Armstrong et al., 1983; Brancaccio et al., 2007; & Paulsen et al., 2005). These researchers suggested that the first increase in CK is due to the initial injury to muscle and the second elevation results from the phagocytic infiltration that occurs in response to the initial injury. There was no biphasic increase in CK in the present study; instead CK levels rose steadily rise for 96 hours. In summary, within the limits of this study (only 96 hours of measurement), CK was comparatively less sensitive than PT in differentiating among various levels of muscle soreness. It is possible that greater efforts are required to cause membrane damage, as lower repetitions did not appear to cause sufficient muscle damage to induce subsequent elevations in CK levels.

Time Course of Soreness-Induction

The consequences of maximal and novel eccentric exercise were studied over four consecutive days. Most DOMS-related studies complete multiple measurements following soreness-induction to assess the time course of muscle damage. All the dependent variables in the present study were impacted as anticipated over the four days, indicating that the soreness-inducing protocol led to muscle damage and associated inflammatory response. The result was pain, swelling, stiffness, loss in muscle function, and leakage of muscle-specific enzymes (i.e., CK) into the blood. These findings are consistent with previous research (Armstrong, 1984). The intensity of discomfort associated with DOMS is said to start in the first 24 h post-exercise, peak between 24 and 72 h

postexercise and resolving in five to seven days (Armstrong, 1984; Cheung et al., 2003; Cleak & Eston, 1992). As expected it was found that all physical measures, as well as reported soreness levels, peaked at 48 h after baseline and most did not return to baseline levels by the fourth day.

It is documented that PT deficit is maximum between 24-48 h following DOMS-inducing exercises (Smith, 1992). It is uniformly believed that the immediate postexercise reductions in PT are attributed to fatigue but later reductions are due to myofibrillar damage (Friden et al., 1983). In the present study, PT did not return to the preexercise value by four days postexercise. Hence, PT recovery following DOMS took longer than four days, which agrees with most DOMS research (Armstrong, 1984; Cheung et al., 2003; Connolly et al., 2003).

There was a significant increase in arm circumference at all measured sites over the four days following soreness induction. This finding is consistent with other studies that found a uniform increase in circumference following eccentric exercise (Cleak & Eston, 1992; Lenn et al., 2003). Lenn et al. (2003) found that at 48 h post-injury, arm circumference increased from 28.4 ± 3.3 and 29.7 ± 3.4 cm to 29.2 ± 3.3 and 30.2 ± 3.5 cm, respectively, at 70 and 100 mm from the lateral epicondyle compared to baseline. These values are consistent with the present findings at 6 and 9 cm at 48 h; at these sites, arm circumference increased from 27.7 ± 2.8 and 29.1 ± 3.0 cm to 28.3 ± 2.7 and 29.6 ± 2.9 cm, respectively, compared to baseline.

A significant increase in RANG was found for all four days postexercise; the change in RANG peaked at 48 h values did not return to baseline by 96 h. Lenn et al. (2003) also found a significant change in RANG at 48, 96, and 168 h after 50 maximal eccentric triceps contractions. The peak change in RANG in their study was also at 48 h after soreness-induction. This decrease in RANG, the time of the peak change, and the time course for resolution was similar between the two studies.

There was also a significant reduction in the range of active elbow flexion for the four days post-soreness induction. The finding coincides with most other studies (Armstrong, 1984; Zainuddin et al., 2005). Zainuddin et al. (2005) found an immediate 30% reduction in elbow flexion after 60 maximal eccentric biceps contractions. A maximum reduction of only 6.5 % was found presently following 60-maximal eccentric triceps contraction at 48 h; ROM began to increase thereafter but was still significantly lower than baseline at 96 h. Hilbert et al. (2000) found a reduction of 16.3% at 48 h after 60-maximal eccentric quadriceps contractions. The reason for inconsistencies might be related to the different muscle groups studied (i.e., triceps vs. hamstring).

The pain associated with DOMS peaked 48 h after soreness induction. After 48 h, soreness started to resolve but did not return to baseline until 96 h. It is believed that the course of soreness development is usually different from the change in muscle strength, ROM, arm circumference, and CK (Nosaka et al., 2002). We found that the time course for all physical measures coincided with soreness except CK, which did not peak until 96 hours post-soreness induction.

Zainuddin et al. (2005) found a significant increase in CK two days following 60 maximal-eccentric biceps contractions; CK remained elevated for 10 days in their study. There was no significant difference in CK across time or among groups in the present study, which is consistent with data from Lenn et al. (2002). Much of the inconsistency in CK data among studies is due to the great variability in CK both within and between subjects (Lenn et al., 2002).

Based on these data and other studies, most of the damage associated with DOMS seems to occur in first two days postexercise and takes longer than four days to resolve. There is probably a relationship between the soreness-

induction effects on range of motion, strength loss, arm circumference, and pain and unpleasantness, as the changes in these variables all peak at 48 h. There is, however, no similar increase in CK and its time course does not clearly coincide with any other physical measures. Thus, there is no clear relationship between the changes seen in CK and the changes in other variables. This finding may be due to the differences in time course for the associated changes. The other variables peak earlier than the changes in CK. In addition, the variability in the CK data makes such comparisons difficult.

Practical Implications

According to the present study, the extent of muscle damage and subsequent loss in PT is directly influenced by the magnitude of eccentric exercise that causes DOMS. Considering future research, the measurement of PT can be used to identify levels of soreness allowing various treatments to be examined on three levels of DOMS. Other variables (swelling, RANG, ROM, DDS, and CK) can be used to more grossly distinguish the effect of treatment on two levels of DOMS. It is conceivable that we will learn that treatment for DOMS can be modulated depending on degree of muscle damage as measured by strength loss. The findings of this study are applicable to real world conditions, as DOMS is the most common consequence following novel eccentric exercise or the initial phase of sports training. Ultimately, the findings from the study could be beneficial for rehabilitation specialist. This study shows how to distinguish levels of DOMS, which may help in selecting the most effective of various interventions to improve patient comfort. Hence the amount of strength loss occurring with DOMS may indicate a particular mode of treatment and thereby improve recovery time.

Some treatment modalities may be effective with lower degrees of DOMS but not acceptable for a higher degree of damage. Massage, for example, may

effectively treat mild to moderate soreness, whereas it may not be useful in treating severe soreness. Research needs to be done to see which treatments can impact different levels of DOMS, especially lower levels of muscle soreness, as almost all previous research has focused on treating a very high level of DOMS induced by extreme soreness-induction protocols (Lighfoot et al., 1997; Paddon-Jones et al., 1997; Zainuddin et al., 2005).

The findings of this study may also be beneficial to sport trainers. For example, since the amount of DOMS is directly related to eccentric effort, sport trainers may change the initial phase of a sports-training program to minimize muscle damage.

The findings of this study may also be applicable to research on the mechanisms that cause DOMS. Lower degree of DOMS may be associated with less muscle damage and subsequent inflammation compared to higher degree of DOMS. Future research on the mechanisms behind DOMS should consider the level of soreness induced by the exercise protocol before drawing conclusions.

Summary

Manipulating the amount of exercise can vary the degree of muscle damage as determined by measuring a host of variables. PT, however, is more sensitive to novel eccentric exercise volume than the others. As the most sensitive variable, PT can be used to differentiate among mild, moderate, and severe muscle damage. This knowledge may lead to determining treatments that are effective for specific levels of DOMS.

Chapter 6

SUMMARY, CONCLUSION AND RECOMMENDATIONS

<u>Summary</u>

The primary purpose of the present study was to determine if the extent of delayed onset muscle soreness (DOMS) following eccentric exercise could be manipulated by varying eccentric exercise volume. If so, this might be valuable information as researchers attempt to determine if specific treatments more effectively resolve various level of soreness. The capability to manipulate the degree of DOMS may also enhance our ability to understand the underlying mechanism for DOMS. To that end, 24 college-aged subjects (males n=12, females n=12) were assigned to three groups, each of which completed an eccentric exercise protocol that included either 20-, 40-, or 60-repetitions with the triceps muscle of the non-dominant arm. Arm circumference (at 2, 6, and 9 cm), ROM, RANG, PT, DDS, and CK were each measured at baseline and 24, 48, 72, and 96 h after the eccentric exercise bout.

A 3 x 5 (Group x Time) RM ANOVA was used to assess group and time differences for each variable. Subsequent univariate ANOVA were completed to exclude the effect of baseline values on group data. Analyses showed that it was possible to manipulate DOMS by varying the amount of exercise as determined by the aforementioned measures. Additionally, PT proved to be the most sensitive variable enabling differentiation among three levels of muscle soreness. The other variables were less sensitive and could only differentiate soreness into two discrete levels. The values for all the measures other than CK peaked at 48 h post soreness- induction, which suggests these variables are well correlated.

CK peaked at or after 96 h and was quite variable across time and between subjects.

Conclusions

The results of this study yield following conclusions:

- DOMS can be manipulated by varying the volume of novel eccentric exercise.
- PT is the key variable to assess the magnitude of muscle damage following DOMS because it is most sensitive to varying exercise volume.
- In the future, this information may prove valuable to rehabilitation and sports medicine professionals who may be able to assess discrete levels of DOMS using PT and then administrating the appropriate and most effective treatment.

Recommendations

The following are recommended for further study:

- Examine the effect of manipulating the exercise volume on DOMS in different muscle groups.
- Determine how manipulation in exercise volume affects DOMS in different age groups.
- Determine the effectiveness of different treatments on the various levels of DOMS using PT as the key to distinguish between mild, moderate, and severe DOMS.
- Examine the changes and pathologies associated with the different levels of soreness to identify better mechanisms leading to DOMS of mild, moderate, and severe degrees.

REFERENCES

- Abraham, W. M. (1977). Factors in delayed onset muscle soreness. *Medicine* and Science in Sport and Exercise, 9(1), 11-20.
- Armstrong, R. B. (1984). Mechanisms of exercise-induced delayed onset muscle soreness: a brief review. *Medicine and Science in Sports and Exercise*, *16*(6), 529-538.

Armstrong, R. B. (1986). Muscle damage and endurance events. *Sports Medicine*, *3*, 370-381.

- Armstrong, R. B., Ogilvie, R. W., & Schwane, J. A. (1983). Eccentric exercise induced injury to rat skeletal muscle. *Journal of Applied Physiology*, 54, 80-93.
- Bale, P., & James, H. (1991). Massage, warm-down, and rest as recuperative measures after short-term intense exercise. *Sports Physical Therapy*, *13* (2), 4-7.
- 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.
- Brancaccio, P., Maffuli, N., & Limongelli, F. M. (2007). Creatine kinase monitoring in sports medicine. *British Medical Bulletin*, 1-22.
- Brown, S., Day, S., & Donnelly, A. (1999). Indirect evidence of human skeletal muscle damage and collagen breakdown after eccentric muscle actions. *Journal of Sports Sciences, 17*, 397-402.
- Brown, S. J., Child, R. B., Day, S. H., & Donnelly, A. E. (1997). Exercise-induced skeletal muscle damage and adaptation following repeated bouts of eccentric muscle contractions. *Journal of Sports Sciences*, *15*, 215-222.
- Cannon, J.G., Fielding, R. A., Fiatarone, S.F., Orencole, S.F., Dinarello, C. A., & Evans, W.J. (1989). Increased interleukin 1β in human skeletal muscle after exercise. *American Journal of Applied Physiology*, 257, 451-455.
- Cheung, K., Hume, P.A., & Maxwell, L. (2003). Delayed onset muscle soreness treatment strategies and performance factor. *Sports Medicine*, 33(2), 145-164.
- Ciccone, C. D., Leggin, B.G., & Callamaro, J. J. (1991). Effects of ultrasound and trolamine salicylate phonophoresis on delayed onset muscle soreness. *Physical Therapy*, *71*(9), 666-678.

Clarkson, P. M., & Ebbeling, C. (1988). Investigation of serum creatine kinase variability after muscle damaging exercise. *Clinical Science*, *75*, 257-261.

- Clarkson, P.M., & Hubal, M.J. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation*, *81*(11), 52-69.
- Cleak, M. J., & Eston, R. G. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Sports Medicine*, *26*(4), 267-272.
- Connolly, D. A., 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.
- Connolly, D. A., McHugh, M. P., & Padilla-Zakour (2006). Efficacy of a tart cherry juice blend in preventing the symptoms of muscle damage. *British Journal of Sports Medicine*, *40*, 679-83.
- DeVries, H. A. (1992). Quantitative EMG investigations the spam theory of muscle pain. *American Journal of Physical Medicine*, 26(4), 267-272.
- Doctor, J. N., Slater, M. A., & Atkinson, J. H. (1995). The descriptor differential scale of pain intensity: an evaluation of item and scale properties. *Pain*, *61*, 251-260.
- Donnelly, A. E., McCormick, K., Maughan, R. J., Whiting, P. H., & Clarkson, P.M. (1988). Effects of non-steroidal anti-inflammatory drug on delayed onset muscle soreness and indices of damage. *British Journal of Sports Medicine*, 22(1), 35-38.
- Ebbeling, C.B., & Clarkson, P.M. (1989). Exercise-induced muscle damage and adaptation. *Sports Medicine*, *7*, 207-230.
- Ernst, E. (1998). Does post exercise massage treatment reduce delayed onset muscle soreness? A systematic review. *British Journal of Sports Medicine*, *32*(3), 212-214.
- Eston, R., & Peters, D. (1999). Effects of cold water immersion on the symptoms of exercise induced muscle damage. *Journal of Sports Sciences*, *17*, 231-238.
- Farr, T., Nottle, C., Nosaka, K., et al. (2002). The effects of therapeutic massage on delayed onset muscle soreness and muscle function following downhill walking. *Journal of Science and Medicine in Sports*, 5 (4), 297-306.
- Fielding, R. A., Manfredi, T.J., Ding, W., Fiatarone, M. A., Evans, W. J., & Cannon, J. G. (1993). Acute phase response in exercise. III. Neutrophil and IL-1 beta accumulation in skeletal muscle. *American Journal of Physiology*, 265, 166-172

- Fitzgerald, G. K., Rothstein, J. M., Mayhew, T. P., & Lamb, R. L. (1991). Exercise-induced muscle soreness after concentric and eccentric isokinetic contractions. *Physical Therapy*, *71*(7), 505-512.
- Friden, J., & Leiber, R. L. (1992). Structural and mechanical basis of exercise induced muscle injury. *Medicine and Science in Sports and Exercise*, 24(5), 521-530.
- Friden, J., Sjostrom, M., & Ekblom, B. (1981). A morphological study of delayed onset muscle soreness. *Experientia*, *37*, 506-507.
- Friden, J., Sjostrom, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. *International Journal of Sports Medicine, 4*, 170-176.
- 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.
- Gracely, R. H., & Kwilosz, D. M. (1988). The Descriptor Differential Scale: Applying psychological principles to clinical assessment. *Pain, 35*, 279-288.
- Gulick, D. T., & Kimura, I. F. (1996). Delayed onset muscle soreness: what is it and how do we treat it? *Journal of Sports 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.
- Hasson, S. M., Mundorf, R., Barnes, W. S., William, J., & Fuji, M. (1989). Effects of ultrasound on muscle soreness and performance. *Medicine and Science in Sports and Exercise*, *22*(4), 199-205.
- Hilbert, J. E., Sforzo, G. A., & Swensen, T. (2003). The effects of massage on delayed onset muscle soreness. *British Journal of Sports Medicine*, *37*, 72-75.
- Hilbert, J. E., Sforzo, G. A., & Swensen, T. (2004). The role of massage in treatment of delayed onset muscle soreness: Brief review. *International Sports Medicine Journal*, *5*(2), 119-128.
- Herbert, R. D., & Gabriel, M. (2002). Effects of stretching before and after exercising on muscle soreness and risk of injury: Systemic review. *British Medical Journal*, *325* (7362), 451-452.
- Hough, T. (1902). Ergo graphics studies in muscular soreness. *American Journal* of *Physiology*, 7, 76-92.

- Isabell, W. K., Durrant, E., Myrer, W., & Anderson, S. (1992). The effect 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.
- Jakeman, P., & Maxwell, S. (1993). Effects of antioxidant vitamin supplementation on muscle function after eccentric exercise. *European Journal of Applied Physiology*, 67, 426-430.
- Jones, D. A., Newham, D. J., & Torgan, C. (1989).Mechanical influences on longlasting human muscle fatigue and delayed-onset pain. *Journal of Physiology, 412*, 415-427.
- Kellis, E., & Baltzopoulos, V. (1995). Isokinetic eccentric exercise. *Sports Medicine, 19*, 202-222.
- Knochel, J. P., (1982). Rhabdomyolysis and myoglobinuria. *Annual Reviews of Medicine*, 33, 435-443.
- Kraemer, W. J., Bush, J. A., Wickham, R. B., Denegar, C. R., Gomez, A. L. Gotshalk, L. A., et al. (2001). Continuous compression as an effective therapeutic intervention in treating eccentric- exercise-induced muscle soreness. *Sport Rehabilitation*, *110*, 11-23.
- 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.
- Lecomte, J. M., Lacroix, V. J., & Montgomerry, D. L. (1998). A randomized controlled trial of the effect of naproxen on delayed onset muscle soreness and muscle strength. *Clinical Journal of Sport Medicine*, *8*, 82-87.
- Lightfoot, J. T., Char, D., McDermott, J., & Goya, C. (1997). Immediate post exercise massage does not attenuate delayed onset muscle soreness. *Journal of Strength and Conditioning Research, 11* (2), 119-124.
- MacPherson, C.D., Schork, A.M., & Faulkner, J.A. (1996). Contraction-induced injury to single permeabilized muscle fibers from fast and slow muscles of the rat following single stretches. *American Journal of Physiology*, 271, C1438-C1446.
- McHugh, M. P., Connolly, A. J., Eston, R. G., Kremenic, I. J., Nicholas, S.J., & Glelm, G. W. (1999). The role of passive muscle stiffness in symptoms of exercise-induced muscle damage. *The American Journal of Sports Medicine*, 27 (5), 594-599.

- 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 Applied of Physiology*, 80, 1006-1013.
- Malm, C., Nyberg, P., Engstrom, M., Sjodin, B., Lenkei, R., Ekblom, B., & Lundberg, I. (2000). Immunological changes in human skeletal muscle and blood after eccentric exercise and multiple biopsies. *Journal of Physiology*, 529, 243-262.
- Malm, C., Sjodin, B., Sjoberg, B., Lenkei, R., Renstrom, P., Lundberg, I., & Ekblom, B., (2004). Leukocytes, cytokines, growth factors and hormones in human skeletal muscle and blood after uphill or downhill running. *Journal of Physiology*, 556(3), 983-1000.
- Mizrahi, J., Verbitsky, O., & Isakov, E., (2001). Fatigue- induced changes in decline running. *Clinical Biomechanics, 16,* 207-212.
- Newham, D.J., Jones, D.A., & Clarkson, P.M. (1987). Repeated high-force eccentric exercise: Effects on muscle pain and damage. *Journal of Applied Physiology*, 63, 1381-1386.
- Noonan, T.J., & Garrett, W.E. (1992). Injuries at the myotendinous junction. *Clinical Sport Medicine*, *11*(4), 783-806.
- Nosaka, K., & Clarkson, P. M. (1996). Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Medicine and Science in Sports and Exercise*, 28, 953-961.
- O'Gardy, M., Hackney, A. C., Schneider, K., Bossen, E., Steinberg, J. M., Douglas, W. J., et al., (2000). Diclofenac sodium (Voltaren) reduced exercise-induced injury in human skeletal muscle. *Medicine and Science in Sports and Exercise*, *32*, 1191-1196.
- Paddon-Jones, D. J., & Quigley, B. M. (1997). Effect of cryotherapy on muscle soreness and strength following eccentric exercise. *International Journal* of Sport Medicine, 18, 588-593.
- Paschalis, V., Koutedakis, Y., Jamurtas, A.Z., Mougios, V., & Baltzopoulos, V. (2005). Equal volumes of high and low intensity of eccentric exercise in relation to muscle damage and performance. *Journal of Strength and Conditioning Research*, 19(1), 184-188.
- ² Paulsen, G., Benestad, H. B., Gundersen, I. S., Morkrid, L., Lappegard, K. T., & Raastad, T. (2005). Delayed leukocytosis and cytokine response to high-

force eccentric exercise. *Medicine and Science in Sports and Exercise*, 37(11), 1877-1883.

- Prasartwuth, O., Taylor, J. L., & Gandevia, S. C. (2005). Maximal force, voluntary activation and musce soreness after eccentric damage to human elbow flexor muscles. *Journal of Physiology*, *567*(1), 337-348.
- Pyne, D. B. (1994). Regulation of neutrophil function during exercise. *Sports Medicine*, *17*, 245-258.
- Rodenberg, J.B., Steenbeck, P., Shiereck, P., et al. (1994). Warm-up, stretching, and massage diminish the harmful effect of eccentric exercise. *International Journal of Sports Medicine*, *15* (7), 414-419.
- Sayers, S.P., Clarkson, P. M., Rouzier, P.A. & Kamen, G. (1999). Adverse events associated with eccentric exercise protocols: Six case studies. *Medicine and Science in Sports and Exercise*, *31*(12), 1697-1702.
- Schmitz, R. J., Martin, D. E., Perrin, D. H., Iranmanesh, A., & Roqol, A. D. (1997). Effect of interferential current on perceived pain and serum cortisol associated with delayed onset muscle soreness. *Journal of Sports Rehabilitation, 6*(1), 30-37.
- Schwane, J. A., Jhonson, S. R., Vandenakker, C. B., Carol, B., & Armstrong, R.
 B. (1983). Delayed onset muscle soreness and plasma CPK and LDH activities after downhill running. *Medicine and Science in Sports and Exercise*, *15*(1), 51-56.
- Schwane, J. A., Johnson, S. R., Watrous, B. G., & Armstrong, R. B. (1983). Is lactic acid related to delayed-onset muscle soreness? *Physiology and Sports Medicine*, *11*(3), 124-127; 130-131.
- Shellock, F. G., Fukunaga, T., Mink, J.H.. & Edgerton, V. R. (1991). Exertional muscle injury: Evaluation of concentric versus eccentric actions with serial MR imaging. *Radiology*, *179*, 659-664.
- Smith, L. L. (1991). Acute inflammation: The underlying mechanism in delayed onset muscle soreness? *Medicine and Science in Sports and Exercise*, 223, 542-551
- Smith, L. L. (1992). Causes of delayed onset muscle soreness and the impact on athletic performance: A review. *Journal of Applied Sports Sciences, 6*(3), 135-141.
- Smith, L. L., Fulmer, M. G., Holbert, D., McCammon, M. R., Houmard, J. A.Frazer, D. D., Nsien, E., & Israel, R. G. (1994). The impact of repeated

bout of eccentric exercise muscular strength, muscle soreness and creatine kinase. *British Journal of Sports Medicine*, *28*(4), 267-271.

- Smith, L. L., Kneating, M. N., Holbert, D., Spratt, D. J., McCammon, M. R., Smith, S. S., & Israel R. G. (1994). The effects of athletic massage on delayed onset muscle soreness, creatine kinase, and neutrophilic count: A preliminary report. *Journal of Orthopedic and Sports Physical Therapy*, 19(2), 93-99.
- Sorichter, S., Puschendorf, B., & Mair, J. (1999). Skeletal muscle injury induced by eccentric muscle action: Muscle protein as markers of muscle fiber injury. *Exercise Immunological Review*, 5, 5-21.
- Stauber, W.T. (1989). Eccentric action of muscles: Physiology, injury and adaptation. *Exercise and Sports Science Reviews*, *17*, 157-185.
- Stauber, W. T., Clarkson, P. M., Fritz, V. K., & Evans, W. J. (1990). Extracellular matrix disruption and pain after eccentric muscle action. *Journal of Applied Physiology*, 69, 868-874.
- Takahashi, H., Kuno, S., Miyamoto, T., Yoshioka, H., Inaki, M., & Akima, H. (1994). Changes in magnetic resonance imaging in human skeletal muscle after eccentric exercise. *European Journal of Applied Physiology and Occupational Physiology*, 69, 408-413.
- Tildus, P.M. (1997). Manual massage and recovery of muscle function following exercise: Literature review. *Journal of Orthopedic Sports and Physical Therapy*, 25, 107-112.
- Tiidus, P.M., & Shoemaker, J. K. (1995). Effleurage massage, muscle blood flow and long-term post exercise strength recovery. *International Journal of Sports Medicine*, *16*, 478-483.
- Walsh, B., Tonkonogi, M., Malm, C., Ekblom, B., & Sahlin, K. (2001). Effect of eccentric exercise on muscle oxidative metabolism in humans. *Medicine and Science in Exercise and Sports*, 33(3), 436-441.
- Warren, G. L., Lowe, D. A., & Armstrong, R. B. (1999). Measurement tool used in the study of eccentric contraction induced injury. *Sports Medicine*, 27(1), 43-59.
- Weber, M. D., Servedio, F. J., & Woodall, W. R. (1994). The effects of three modalities on delayed onset muscle soreness. *Journal of Sports Physical Therapy*, 20(5), 236-242.

Wessel, J., & Wan, A. (1999). Effects of stretching on the intensity of delayed onset muscle soreness. *Clinical Journal of Sports Medicine*, *4*(2), 83-87.

Zainuddin, Z., Newton, M., Sacco, P., & 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.

Zusman, M. (1986). The absolute visual analog scale. *Australian journal of Physiotherapy*, 32, 244-246.

APPENDICES

APPENDIX A

Informed Consent Form

Manipulating the Extent of Delayed Onset Muscle Soreness

1. Purpose of the Study: The purpose of this study is to determine if three discrete levels of soreness (e.g., mild, moderate, and heavy) can be induced in the triceps muscle in the non-dominant arm.

2. Benefits: You may benefit from participating in this study because you will learn about the Cybex dynamometer, a device used in many rehabilitation settings. You will also get first hand experience on how scientific data are collected and receive extra credit. Last, it is expected that your efforts will benefit the scientific community.

3. Your Participation requires you must be at least 18 years of age, able to perform maximal eccentric triceps contraction on the Cybex dynamometer, and have not lifted weights with your arms regularly (twice a week for at least a month) in the previous three months. All tests will be conducted in CHS 401. If you participate, you will have to attend a familiarization session so that you can learn about the Cybex and the various measures I will make in the study. This session will last one hour. With these measurements I will assess your mood and muscle soreness with questionnaires. I will also assess elbow range of motion with a goniometer shown in class, upper arm circumference with a tape measure, and peak triceps strength with the Cybex, which will require you to complete several efforts on the machine. One week after the first session, you will report back to the lab. I will repeat many of these measures as described or shown with one exception; I will mark your non-dominant arm with permanent ink so that I can maintain consistency in circumference and range of motion measurements across the remainder of the project. The ink will wash off after several days. Just prior to the exercise I will also draw 5 ml of blood from a vein in your arm with normal procedures used in a medical clinic. You will then complete an assigned number of maximal triceps eccentric contractions on the Cybex (24, 48, or 72 efforts). Immediately after the exercise and 24, 48, 72 and 96 hours later (1, 2, 3, and 4 days), I will assess arm strength, range of motion, and circumference as shown. At 24, 48, 72 and 96 hours post exercise I will assess mood and muscle soreness and draw an additional blood sample. Each lab visit will take approximately 20 minutes of your time. Thus, the experimental phase of the project will require about two hours of participation time. Total participation time for the entire project, therefore, will be about three hours. As stated, I will also draw five, 5 ml samples of blood or 25 ml total over the course of the study.

4. Risks of Participation: This experiment will make your arm sore for several days. This soreness is called delayed on-set muscle soreness (DOMS) and must of you have probably experienced it. DOMS is associated with pain, tenderness, swelling, reduced range of motion, and decreased muscle strength in the exercised muscles. These symptoms last for two to five days depending on the severity of the damage and are mildly self limiting. To reduce your inconvenience I will have you exercise only a small muscle group on one limb; in this case the triceps muscle on your non-dominant arm. Thus, you will be able to walk normally and to use both hands, such as to type or text after the exercise intervention. You may, however, have a difficult time reaching for items with your non-dominant arm for several days. The symptoms will resolve without any treatment in less than five days. You will not be able to take any anti-inflammatory or pain medications for the duration of the experimental phase of the project; you also cannot apply heat or cold to the sore region. In extreme cases involving large amount of muscle tissue (whole body) and exercise, the resulting muscle damage may induce exertional

rhabdomyolysis, which is characterized by fever, nausea, vomiting, and blood in urine. Rhabdomyolysis can progress to renal failure in chronic stages. Rhabdomyolysis is extremely rare in real world applications and not documented in the type of research proposed and related projects, because they typically involve a small amount of muscle confined to one limb and a limited amount of exercise. The study also involves venipuncture, which will be done with all universal precautions to avoid risk of infection at the puncture site.

5. Compensation for Injury: If you suffer an injury that requires any treatment or hospitalization as a direct result of this study, the cost of such care is your responsibility. If you have insurance, you may bill your insurance company. Ithaca College and the investigator will not pay for any care, lost wages, or provide other compensation.

6. If you would like more information about this study at anytime prior to, during, or following the data collection, you may contact Ankita Dubey at <u>adubey1@ithaca.edu</u> (513)-417-6139, Professor Swensen at <u>tswensen@ithaca.edu</u> (607)-274-3114, or Professor Sforzo at <u>sforzo@ithaca.edu</u> (607)-274-3359.

7. Withdrawal from the study: Participation in this study is voluntary and you may withdraw at any time if you so choose. You will not be penalized for withdrawing.

8. Confidentiality: Information gathered during this study will be maintained in complete confidence. Only the researcher will have access to this information, which will be stored in a locked cabinet in Professor Swensen's office in 321 Center for Health Sciences at Ithaca College and on password protected computer. You and your name will never be associated with this information in any reports. To further insure confidentiality, all files will be number coded and data collection instruments will be kept separately from Informed Consent Forms and sign-up sheets.

I have read and understood the above document. I agree to participate in this study and realize that I can withdraw at anytime. I also understand that I can and should address questions related to this study at any time to any of the researchers involved. I also verify that I am at least 18 years of age.

Your Name (please print)

Your Signature

Date
APPENDIX B

Medical History and Health Habit Form

Name:		Student ID:
Age:	Weight:	Sex:
Dominant Arm		· · ·
Medical/Health H	listory (please check all that	t apply)
[] Heart/Disease		[] Lung Disease
[] Stroke		[] Diabetes
[] Heart Murmur		[] Epilepsy
[] Skipped, rapid	or irregular heart rhythms	[] Injuries to shoulder, elbow, wrist,
fingers	2	
[] High Blood Pr	essure	[] Soft tissue injury to upper limb
[] High Choleste	rol	
[] Rheumatic Fe	ver	
[] Other conditio	ns/comments: (please expla	in)
Present Symptom	is (please check all that have	e applied within the last six months)
[] Chest pain		[] Shoulder, elbow, wrist swelling
[] Shortness of B	Breath	[] Joint/muscle injury requiring
medical attention		
[] Lightheadedne	ess	[] Allergies (if yes, please list)
[] Heart Palpitati	ons	[] Muscle injury (upper limb)
[] Loss of consci	ousness	• ·
[] Illness, surger	y, or hospitalization	
[] Other conditio	ns/comments: (please expla	in)
Is your medication	includes any pain relieving	drug? [] Yes [] No
Current medicati	ons (please list all medicatio	ons presently being taken)
	······································	······
Exercise Habits		
Do you presently e	engage in physical activity?	[]Yes []No
If so, what type of	exercise?	erobic [] Strength Training []
Both		
How hard do you	exercise?	asy [] Moderate []
Hard		2
How many times a	week do you work out on a	verage?
How many times a	day do you work out on ave	erage?
Have you ever had	any discomfort, shortness o	f breath, or pain while exercising?
Have you participa	ted in strength training prot	ocol in last three month especially upper
[]] []]	Ves []No	
If so please evolution	n vour training	4
ii so, picase expiai	n your training	a .

۶.

APPENDIX C

24-Hour Health and Activity History Form

Name:	Date:		<u>_</u>
Current Health Status (please of	check all that apply)		
[] Nausea Headache	[] Sore Throat		[]
[] Body Ache Lethargy	[] Chills		[]
] Nasal Drip Muscle Aches	[] Cramping		[]
[] Chest Pain Dizziness	[] Shortness of Brea	th	[].
DIET			
Have you consumed alcohol in the last 12 hours?		[]Yes	[] No
Have you used caffeine or nicotine in the last three hours?		[]Yes	[] No
Did you eat any food in the last three hours?		[]Yes	[] No
If so, please list:			
Has your diet changed drastically since the last exercise test?		[]Yes	[] <u>N</u> o
If so, please describe:			
Exercise			
Have you exercised in the last 24 hours?		[]Yes	[] No
If so, please describe:			
Has your exercise routine changed i	n last few weeks?	[]Yes	[] No
If so, please explain:			

Have you taken any over the counter drugs (e.g., cold meds, analgesics) in the last 24 hours?

[] Yes [] No

	Has there be	en any change	n your use of	prescription	drugs?	[]Yes	[] No
--	--------------	---------------	---------------	--------------	--------	-------	--------

If so, please explain:

Injury

Have you experienced any physical pain in the last 24 hours?	[]Yes	[] No
If so, please explain:		

Is there any physical injury we should know about before you perform the test?

	[]Yes	[] No

If so, please explain:

Sleep Pattern:

Has your sleep pattern changed since the last exercise test?	[]Yes	[] No
Do you feel drowsy, tired, or run down at this time?	[]Yes	[] No

If so, please describe:

Has there been any change since the last exercise test that you feel could compromise your performance on today's exercise test? [] Yes

[] No

If so, explain:

Other questions/comments/concerns please state below.

APPENDIX D

Instructions

You are scheduled to attend a familiarization session prior to actual test. Your performance depends upon the adherence of these instructions:

- 1. Do not perform heavy exercise in the 24 hours preceding the test.
- 2. Do not drink alcohol for 12 hours preceding the test.
- 3. Do not use stimulants such as caffeine (e.g. coffee) or nicotine (i.e. cigarettes) for 3 h preceding the test.
- 4. Do not eat for one hour preceding the test.
- 5. Do not eat any food that may cause you discomfort the day of the test.
- 6. Do not use any pain medication or any treatment to reduce your symptoms when you are being tested, or after experiment for 4 days.
- 7. Avoid over-the-counter medications for the 12 hours preceding the test. (However, cancel the appointment if you are ill and treat yourself accordingly; we can always reschedule)
- 8. Wear comfortable clothing during the test. (i.e. t-shirt, with loose sleeves or sleeveless t shirt)
- 9. Please, sustain your same lifestyle habits (eating, exercise, medication, etc.) between tests.

I thank you for your cooperation!

j.

APPENDIX E

Differential Descriptor Scale (DDS)

Differential Descriptor Scale (DDS)

Each word represents an amount of sensation.

Rate your sensation in relation to each word with a check mark.

-	Faint	· · · +
-	Moderate	. +
-	Barely Strong	+
		+
	Weak	
-	Strong	· · ·
•	Very mild	+
-	Extremely Intense	+
-	Very weak	· +
-	Slightly Interise	+
		· · · · · · · · · · · · · · · · · · ·
-	Very Intense	+
-	Mild	+

Each word represents an amount of unpleasantness.

Rate your unpleasantness in relation to each word with a check mark.

-	Slightly Unpleasant	+
-	Slightly Annoying	+
-	Unpleasant	+
		+
-	Slight Distressing	+
• 	Very Unpleasant	+
-	Distressing	+
-	Very Annoying	+
-	Slightly Intolerable	+
-	Very Distressing	+
-		+
-	Very Intolerable	+

ÁPPENDIX F

Soreness Data Collection Sheet

Name: Subject ID.....

1. Arm circumference: (three trials for each measurement)

Time		At 2 cm			At 6 cm		At 9 cm		
	1	2	3	1	2	3	1	2	3
0h			-				•		
24h									
48h									
72h								•	
96h									

2. RANG:

Time	1	2	3
0h			
24h			
48h			
72h	•.		
96h			

3. Elbow ROM: (three trials for each ROM)

Time	Active elbow flexion		on	Active elbow hyperext(below 0)		
-	1	2	3	1	2	3
0h	•••					
24h	<u> </u>					······································
48h	·····		·			·
72h				,		
96h	:• :					

Soreness Data Collection Sheet (PEAK TORQUE)

Name:	Age/Sex	Subject	
group			•

Testing-arm: Right/Left

Dynamometer settings:

Chair setting:

Rotation scale:

Back angle:

Fore aft Position:

Back Translation:

Seat Position: Flat

Dynamometer setting:

Tilt:

Height:

·

Accessories: Mechanical stops: Length adapter: Gray:

Hand grip:

Teal:

Peak Torque (Nm): (averaged peak torque for three maximal trials)

Time	Oh	24h	48h	72h	96h
peak torque (Nm)					· · · · · · · · · · · · · · · · · · ·

APPENDIX G

RAW DATA TABLES

Peak torque (Nm):

		·			
Group	Oh	24h	48h	72h	96h
å	38	27	20	22	22
' a	33	27	28	41	· 35 [.]
а	45	39	46	49	. 46
а	58	45	33	39	35
а	58	45	. 46 .	41	52
а	103	85	76	84	83
а	- 77	58	60	65	64
а	75	76	89	89	114
b	37	22	23	23	24
b	76	43	49	50	49
b	75	50	37	39	60
b	60	46	61	65	72
b	49	27	24	26	22
b	73	39	39	41	47
b	-53	42	33	30	33
b	41	39	34	31	26
, C	58	19	16	22	22
Ċ	39	16	18	20	15
С	50	27	31	23	22
С	43	22	15	12 .	18
с	49	22	19	16	20
с	46	31	23	20	24
с	35	26	22	26	33
С	81	57	57	68 [.]	65
С	81	57	57	68 [.]	65

group	Oh	24h	48h	72h	96h
а	26.7	27.5	27.8	28.4	26.8
а	26.5	26.6	26.7	26.5	26.5
a	25.1	25.1	25.4	25.3	25.2
а	24.3	24.6	24.7	24.6	24.5
а	29.8	29.9	30.3	30.3	30.1
а	30.3	30.2	30.7	30.3	30.3
а	25.5	25.6	25.8	25.6	25.6
а	25.9	25.9	26	26.1	26.1
b ·	26.2 [.]	26.9	27.6	27.6	27.4
b	25.3	25.4	25.6	25.3	25.2
b	29.3	29.6	30	29.6	29.5
b	26.5	26.5	26.6	26.6	26.5
· b	22.9	23.6	23.6	23.4	23.1
b	26.1	27.3	27.8	27.2	27.3
b	26.9	27.2	27.2	27.1	27.1
b	24.8	25.2	25.5	25.6	25.1
Ċ	24.5	25.2	25.5	25	25.1
с	22.6	22.8	23.1	23.1	23
· C	25.2	27.3	27.4	27.4	27.3
С	20.7	21.4	21.4	21.4	21.5
С	25.1	25.6	25.5	25.5	25.2
С	23.6	24.5	24.6	24.6	24.6
C.	26.1	26.2	26.5	26.3	26.3
с	30.6	31.3	31.5	31.5	31.1

Arm Circumference at 2 cm (cm):

·····					·····
Group	Oh	24h	48h	72h	96h
а	28.8	29.1	29.5	29.6	29.4
а	28.9	28.9	28.9	28.9	28.9
а	26.1	26.2	26.6	26.5	26.4
а	25.4	25.7	26.2	25.9	25.9
a	31.2	31.6	31.6	32.2	31.8
а	33	33	33	33	33.0
а	27.5	27.6	27.8	27.8	27.7
а	27.2	27.2	28.2	27.7	27.7
b	28.2	28.8	29	29	28.9
b	26.4	27.5	27.6	27.4	27.5
b	31.7	32.1	32.2	32.1	32.1
b	28.5	28.5	28.4	28.4	28.4
b'	23.4	23.6	23.8	23.6	23.7
b	28.8	29.9	30	29.9	29.9
b	29.6	29.8	29.4	29.6	29.6
b	25.8	26.2	26.5	26.6	26.4
С	26.1	27.3 ⁻	27.4	27.2	27.3
- C	23.9	24.3	24.6	24.2	24.4
С	27.3	29	29.4	29.4	29.3
С	22.4	22.6	22.9	22.9	22.8
C	27.5	27.6	27.7	27.5	27.6
С	25.5	25.6	26	26.1	25.9
Ċ	28	28.2	28.2	28.4	28.4
с	33.7	34.4	34.4	34.1	33.9

Arm Circumference at 6 cm (cm):

Arm Circumference at 9 cm (cm):

Group	Oh	24h	48h	72h	96h
а	30.2	30.2	30.7	30.6	30.2
а	30.6	30.5	30.5	30.5	30.5
а	28	28	28.6	28	28.1
а	26.4	26.7	26.6	26.4	26.4
а	33.7	34	33.9	34.2	34.4
а	34.1	34.1	34.1	34.2	34.1
а	28.5	28.6	28.8	28.7	28.6
а	28.1	28.1	28.8	28.2	28.2
b	30.2	30.5	30.6	30.1	30.1
b	27.9	29	29	29.1	28.9
b	33.5	33.9	34.3	34.3	33.8
b	29.2	29.2	29.1	29.2	29.2
b	23.5	23.8	23.9	23.9.	23.8
b.:	30.2	31.6	31.6	31.4	31.4
b	30.6	30.8	30.8	30.6	30.5
b	27.1	27.4	27.7	27.8	27.6
с	27.5	28.9	29.4	28.7	. 28.5
с	25.9	26.1	26.2	26.1	26
с	29.1	29.2	30	31.3	30.8
С	23.6	23.8	24.4	24.4	24.4
С	28.8	28.9	29	28.9	28.9
с.	25.8	26	26.6	26.7	26.6
с	29.6	29.6	29.6	29.5	29.6
с. С.	35.3	35.4	35.5	35.4	35.3

RANG (degrees):

Group	0h	24h .	48h	72h	96h
a	22	23	22 -	22 ⁻	22
а	. 15	16	15	15	15
а	13	16	17	15	15
а	20	22	22	20	20
а	28	30	28	27	27
а	25	25	25	24	25
а	29	30	29	28	28
а	21	24	23	23	22
b	22	23	22	23	22
b	24	26	28	28	28
b	25	26	26	26	26
b	31	31	32	32	32
b	19	19	20	19	19
b	25	29	30	29	29
b	26	. 27	31	30	30
b	19	19	20	20	19
С	18	21	22	21	21
Č ^{z s}	20	25	25	24	20
С	19	20	20	20	21
С	24	27	27	.25	25
Ċ	31	33	34	33	33
С	22	23 ⁻	24	24	24
С	20	23	24	22	21
С	27	32	33	31	31

ROM (degrees):

Group	0h	24h	48h	72h	96h
а	138	134	134	,135	137
а	129	129	130	131	131
a	146	144	145	146	146
а	151	148	149	150	149
а	155	147	146	147	145
а	131	126	127	129	130
а	151	147	146	149	149
а	148	148	147	145	146
b	150	143	141	143	144
b	147	143	143	143	143
b	138	137	138	138	139
b	147	137	142	141	143
b	131	128	128	131	132
b	142	135	127	130	130
b	131	126	120	127	130
b	151	151	149	149	150
С	137	134	133	135	136
С.	145	137	136	134	138
С	140	114	112	131	136
С	149	146	142	143	143
С	150	144	140	141	142
С	146	138	133	139	138
с	137	135	136	135	138
С	130	126	127	129	131

T.

DDS (sensation):

Group	0h	24h -	48h	72h	96h
а	0	5.5	6.08	7.25	1.66
а	0	3.08	2.08	0.833	0
а	0	4.33	7.33	7.66	5.75
а	0	· 5.16	7.91	5.08	1.9
а	0	6.66	6.3	4.5	4.5
~ a	0	1.91	1.41	0.91	0.66
а	0	4.5	1.66	1	0
а	0	5.16	8.75	5.58	4.16
b	0	0.58	2.41	2.58	2.33
b	0	4.5	4.91	6.75	6.6
• b •	0	4.66	9.33	4.25	4.91
`b	0	0	2.08	0	0
b	0	5.58	6.08	8.41	6.33
b	. 0	4	3.6	3.5	1.8
b	0	3.3	2.66	2.08	1.75
b	0	6.33	10.6	1.58	0.16
с	0	10.6	10.9	8.33	5
с	0	1.5	4.33	2.8	2.08
с	0	9	11.25	9.91	9.33
с	0	7.5	6.83	7	7.66
с	0	9.3	7.41	4.8	1.6
C	0	5.41	4.91	2.25	1.6
С	0	1	0.83	0	0
С	0	3.25	4.5	0.42	0

DDS (unpleasantness):

· Group	Oh	24h	48h	72h	96h
а	* 0	. 3.3	3.41	1.58	0
а	0	0	0	0	0
а	0	0.66	5.08	6.33	3.16
а	0	0	2.5	2.08	1.16
а	0	1.16	0	0	0
а	0	0.58	0.75	0	0
а	0	1.25	0.91	0	0
a '	· 0	0.58	0	1.3	0
b	0	0.58	1.16	1.66	1.41
b	0	3.58	4.5	6.66	6.08
b	0	3.25	4.75	2.58	1.91
b	0	0	0	. 0	0 ·
b	0	4.58	4.5	4.8	3.25
b	0	1.1	1.4	1.5	0
b	0	2	0.83	0.83	0
b	0	4.8	9.9	<u></u> 0	0
. C	0	15	1 5.5	5.08	4.66
с	0	0.33	1.83	1.25	0.16
Ċ	0	12.6	13.75	12.33	10.8
С	· 0	4.75	8.58	9.25	10.33
с	0	4.6	2.5	0.91	0
с	0	3.58	1.3	1.9	0
с	0	0.83	0.16	0	0
С	0	0.91	0.83	0.16	0

Creatine kinase (I·L⁻¹):

	·····	· · · · · · · · · · · · · · · · · · ·		1	· · · · · · · · · · · · · · · · · · ·
Group	Oh	24h	<u>48h</u>	72h	96h
а	48.7	#DIV/0!	146.5	348.8	1169
а	176.7	270.9	280.2	291.5	238.9
а	54.9	45.9	43.6	44	35.8
а	119.2	203.5	167.6	124.2	152.6
·a	65.6	70.6	94	. 80.2	74.4
а	123.4	127.4	100.1	102	84
а	453.9	420.3	582.9	752.9	759.3
а	178.9	241.2	270.6	305.4	266.9
b	35.6	49	66.2	248.6	1133.7
b	140.5	, 134.4	597.9	1397.8	5533.7
b	150.1	164.9	318.2	305.8	285.3
b	171.4	96	64.6	57.7	58.4
b	63.4	65	70.9	75.1	72.8
b	85.5	1100.8	1525.8	2608.1	4139.7
b	306.7	194.9	134.9	129.4	182.9
b	- 70.2	66.9	66.7	46.8	62.4
с	80.7	110.7	138.7	187.5	493.8
С	67.6	89.7	58.9	62.6	66.1
с	166.9	6838.9	16008.5	21745.5	19499
С	69.6	78	90.4	811.7	2348.5
C ,	114.1	168.7	139.1	128.2	99.4
С	312.2	289.4	1132.1	8202.6	11440
С	89.5	85.8	92.6	91.7	161.3
с	278.6	339.9	283.7	355.2	400.7