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Moderate intensity cycling following eccentric contractions does not attenuate indirect markers of muscle damage.

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Graduate Program in Kinesiology
A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science
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Moderate intensity cycling following eccentric
contractions does not attenuate indirect markers of
muscle damage

By

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Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements
for the degree of Master of Science

The School of Graduate and Postdoctoral Studies
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Brendan P. Major 2013

Abstract

Unaccustomed eccentric muscle contraction causes prolonged changes to indirect markers of muscle damage (Clarkson & Hubal, 2002). It has been proposed that active recovery therapy (ART) consisting of non-eccentric based movements may aid in the recovery process (Sayers et al., 2000). The purpose of this investigation was to compare the effect of ART on changes in one repetition maximum (1RM), dynamic muscle function, swelling, delayed onset muscle soreness (DOMS), and changes in MRI T2 relaxation times post exercise induced muscle damage (EIMD). Ten previously trained participants (at least 1 lower body resistance training session a week) were recruited from within the university community and divided in two groups: a cycling ART group (CG, n = 5) who performed three bouts of moderate intensity cycling in the four days following the EIMD protocol and a non-cycling group (NCG, n= 5) who performed no cycling in the four days following the EIMD protocol. There were no statistical differences between the two treatment groups for height, weight or age ($P < 0.05$). The EIMD protocol reduced 1RM, muscle function, elicited DOMS and increased swelling and T2 relaxation times in both CG and NCG ($P < 0.05$). In the 96 h period post the EIMD protocol no differences were observed between CG and NCG for the magnitude of change or rate of recovery. Three bouts of cycling ART performed at 24 h intervals after an EIMD did not improve indirect markers of muscle damage.

Keywords: magnetic resonance imaging, maximal voluntary contraction, eccentric muscle action, active recovery therapy, delayed onset muscle soreness.

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List of Abbreviations and Symbols

1RM	one repetition maximum
ANOVA	analysis of variance
ART	active recovery therapy
Ca ⁺⁺	calcium
CG	cycling group
CK	creatine kinase
CMJ	counter movement jump
CON	concentric
CSA	cross sectional area
DOMS	delayed onset muscle soreness
DJ	drop jump
ECC	eccentric
EIMD	exercise induced muscle damage
EIA	exercise induced analgesia
ISO	isometric
LDH	lactate dehydrogenase
MR	magnetic resonance
MIC	moderate intensity cycling
NCG	non-cycling group
RFD	rate of force development
ROI	region of interest
ROM	range of motion
RPP	rate of perceived pain
SSC	stretch shortening cycle
SJ	squat Jump
T2	transverse relaxation
VL	vastus lateralis
VM	vastus medialis

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Chapter 1: Introduction

1.1 Exercise Induced Muscle Damage

Exercise induced muscle damage (EIMD) was first described by Hough in 1902. Hough was able to induce EIMD by having human subjects perform eccentric finger extension against a loaded spring. Hough made the distinction between the fatigue (the discomfort experienced during exercise) and EIMD, which occurs after cessation of exercise. Exercise induced fatigue was due to primarily the buildup of metabolites during exercise, such as increased hydrogen ions and their detrimental effect on muscle contractile proteins. These metabolic changes are temporary and generally dissipate within 60 minutes (Allen et al., 2008). Unlike fatigue, Hough attributed the pain and weakness in the days after EIMD to “some sort of rupture within the muscle”. EIMD can cause prolonged changes in muscle function for up to 75 days (Allen, 2006; Byrne et al., 2004).

EIMD is most pronounced in human skeletal muscle following unaccustomed eccentric muscle contraction. Eccentric muscle action features a unique loading profile unlike any other muscle action, combining limited neural recruitment of muscle fibres with a much greater capacity for work (Enoka, 1996). Evidence indicates this loading profile creates an uneven amount of strain on the individual contracting muscle fibres (Enoka, 1996). Eccentric contractions may also involve the preferential recruitment of type II fibres (Fridén & Lieber, 1992), which have a higher capacity to produce force than type I. This creates a loading profile with high force and low fibre recruitment. The uneven distribution of strain is thought to cause cellular disruption within any sarcomeres that are weak and more susceptible to damage (McHugh, 2003).

EIMD frequently occurs in sporting populations where a large number of eccentric type contractions occur and athletes are repeatedly exposed to varied and unaccustomed training loads. Although the effects EIMD on basic muscle function have been well studied; the effect damage has on physical performance needs further examination. There is a lack of effective means other than time of attenuating damage and speeding the recovery from EIMD.

1.2 Recovery From EIMD

Recovery from EIMD refers to both the repair of the physiological damage done to the muscle and the elimination of any performance decrement induced by the exercise. Damaged muscle tissue will naturally recover from EIMD over a period of time, dependent largely on the severity of the initial damage. However, in an athletic setting; restoring optimal function following EIMD caused by training or competition as quickly as possible is vital, as many sports have competition on consecutive days. Minimizing recovery time allows athletes to optimize training schedule and continually perform at optimal levels. A number of interventions are currently used as a means of hastening the recovery or healing process, such as ice, massage and active recovery. Active recovery therapy (ART) involves further muscle actions usually solely concentric and submaximal in nature i.e. cycling or underwater running. The effectiveness of ART in removal of muscle lactate and decreasing the sensation of delayed onset muscle soreness (DOMS) post exercise is well established (Coffey et al., 2004), however its effectiveness in improving recovery of performance measures following damage remains unclear. After competition and training coaches frequently put their players through ART in an attempt to minimize the negative effect of EIMD (Tessitore et al., 2007). However no clear

evidence has been presented supporting the use of ART recovery as an effective means of accelerating recovery of athletic performance post EIMD.

1.3 Stretch – shortening cycle: the effect on performance post EIMD

The negative effect of EIMD on muscle performance may vary depending on the testing protocol used. It has been hypothesized that following EIMD the failure of some of the contractile elements to generate force may be partially overcome through pre-stretching of the muscle (Byrne & Eston, 2002a). After completing 10 sets of 10 squats, participants' squat Jump (SJ) performance, an activity which does not involve a pre-stretch phase, was affected to a greater extent than a counter-movement jump (CMJ) or a drop Jump (DJ). Both of these jump types use a pre-stretch phase where elastic energy is built up within the muscle, prior to the concentric phase (Byrne & Eston, 2002a). The rate of recovery, as well as magnitude of change as a consequence of damage for CMJ, SJ or DJ has also been reported to differ. Following a 90km footrace the time course of recovery was 3, 11 and 18 days for the DJ, CMJ and SJ respectively (Chambers et al., 2010).

1.4 Evaluation tools:

In research on human's difficulties exist in directly studying the damaging effects eccentric exercise has on the muscle. Because of this indirect performance measures such as one repetition maximum (1RM) and dynamic muscle function tests (for example, the jumps mentioned above) are commonly used. Non-invasive magnetic resonance imaging (MRI) techniques can be used to examine markers of cellular change. Several direct and indirect measurements of muscle function have been used to evaluate the effects of

EIMD. Changes in torque, range of motion, histological profile, blood levels concentrations of myofibril proteins, soreness and MRI have been used to measure EIMD (Warren et al., 1999). As yet there is no single gold standard measurement of EIMD (Paulsen, et al., 2012) but the use of MRI to qualify the cellular response to EIMD is becoming increasingly popular (Foley, et al., 1999; Ploutz-snyder, et al., 1996; Jayaraman, et al., 2004).

1.4 Purpose:

- The purpose of this study was to determine whether ART would attenuate the severity of indirect markers of EIMD and increase the rate of recovery of muscle function.
- Examine the effect the SSC has on muscle function following EIMD.

1.5 Hypotheses

- Participants who engage in an ART will not be as adversely affected by EIMD. Post EIMD 1RM, jump height and rate of perceived exertion will return to baseline values in the CG before the NCG.
- The use of the SSC in counter-movement jump will mean: Squat jump performance will decrease more post EIMD when compared with counter-movement jump performance.

2. Literature review

2.1 Introduction:

Muscle damage has been well studied since the early work by Hough in 1902. When skeletal muscle is exposed to unaccustomed, ECC contractions, or the ECC contractions are too strenuous or too frequent then damage can occur. The damage by these contractions is referred to as exercise induced muscle damage (EIMD). Although EIMD does not have a specific definition, in humans it is characterized by an immediate and prolonged reduction in muscle function (Bryne et al., 2001); muscle stiffness, swelling and a sensation of pain (Chapman, et al., 2008) an increase in circulating muscle proteins (including creatine kinase (CK), lactate dehydrogenase (LDH), and myoglobin (Mb) (Newton et al., 1983a; Nosaka & Clarkson, 1996; Sayer & Clarkson, 2003); and a reduction in athletic performance (Sargeant & Dolan, 1987).

2.2 Evaluation tools - MRI

With controversy over the whether or not biopsies increase the level of damage their intrusive nature and unwillingness of athletes to want biopsies performed, the use of MRI may provide a non-invasive means of evaluating the cellular response to EIMD. Direct analysis of EIMD is difficult because histological and biological examinations of muscle biopsy are intrusive, expensive and delicate and may miss the site of damage. The non-uniform nature of muscle damage may partially invalidate the biopsy technique, as the tissue sampled may not be a presentation of total muscle response to damage inducing muscle action. Magnetic resonance imaging (MRI) provides a non-invasive means to assess the direct changes to skeletal muscle as a whole (Marqueste et al., 2008).

In the initial phase after any exercise, acute increase in $^1\text{H-NMR}$ transverse relaxation time (T2) of muscle water in the active tissue can be observed in T2 weighted MRI images (Saab et al., 2000). This acute change in T2 dissipates rapidly, usually within one hour of cessation of exercise. This increase can be instigated by any form of exercise; including those that are non-damaging in nature (Prior et al., 2001).

The second phase is a gradual increase in T2 times occurring anywhere from 12 - 24 h post exercise, peaking 3-5 days after (Fleckenstein et al., 1989; Takahashi et al., 1994). Damaging contractions induces the second increase in T2. The exact mechanisms for this prolonged changes in T2 relaxation times are not yet well known but it is thought to be due at least in part to edema (Clarkson & Hubal, 2001). Changes in a muscles T2 relaxation times correlate well with changes in DOMS, CK and cross sectional area; leading researchers to postulate T2 times may be detecting muscle edema or cell rupture (Takahashi et al., 1994). However the time course of T2 relaxation changes do not correlate well with changes in muscle function (Mair et al., 1992), and the prolonged period of elevation could also reflect adaptation and regenerative changes undergone by muscle cells (Mair et al., 1992). A T2 change in response to ECC exercise may allow researchers to investigate the involvement of individual muscles for particular movements. T2 also could be used to grade the level of damage induced by a particular exercise protocol. And may help with grading the level of damage without the chance of interference from confounding variables such as biopsies or indirect markers of muscle function testing. Testing methods such as MVC or 1RM may cause further damage to the muscle when repeated over several days (Jayaraman et al., 2004). The exact mechanism for increases in T2 times is not well elucidated and needs further research; but the change

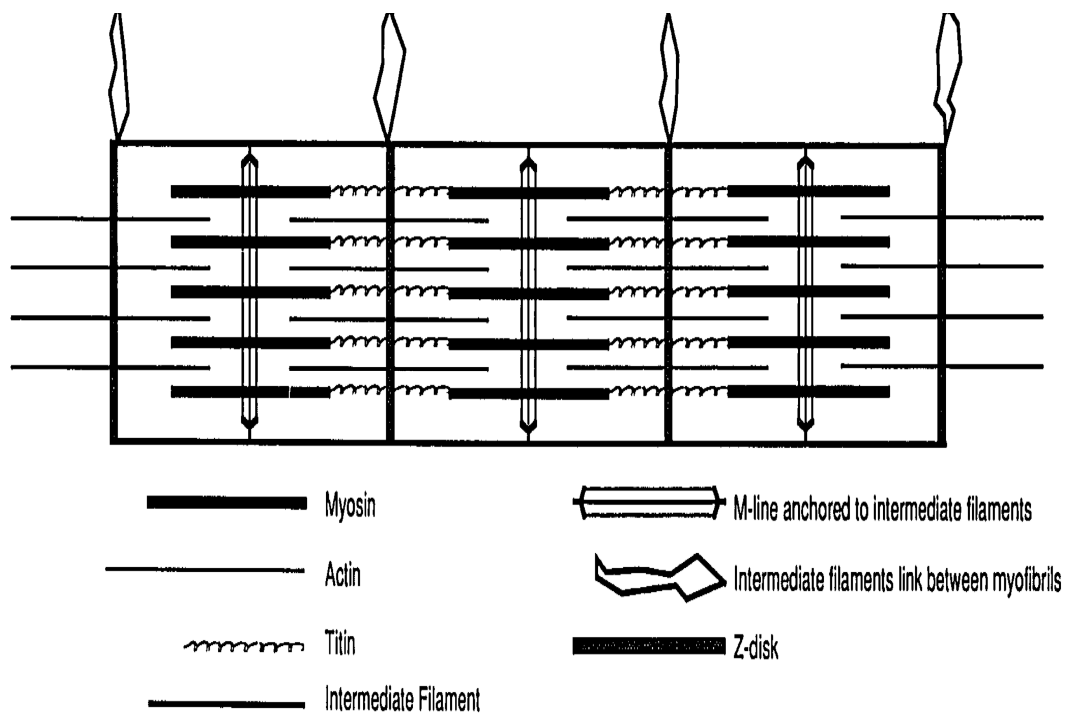
in T2 times may allow researches an alternative method of damage evaluation other than blood protein and muscle function measures.

2.3 Normal muscle function

The human neuromuscular system is comprised of motor neurons and the contractile fibres they innervate. Each muscle will have a number of motor neurons of different sizes, each innervating a different set of muscle fibres. The process of initiating a muscle contraction is achieved via the central nervous system recruiting motor units in a selective order. Motor units are recruited according to the “size principle” (Henneman et al., 1965), which dictates that motor units with the smallest cell diameter will be recruited first, and will be the last to be de-recruited. As the intensity of the contraction increases, motor units are recruited in this fixed order, from smallest to largest cell bodies. Motor units with smaller cell bodies will be excited first as they have less surface area to depolarize and a relatively greater concentration of excitatory current. Once a motor unit has been depolarized it sets in motion a chain of events known as the *excitation-contraction coupling* – this provides the physiologic mechanism whereby an electrical discharge at the muscle initiates chemical events at the cell surface, releasing intracellular Ca^{++} and ultimately causing muscle contraction.

Overall movement is achieved via this longitudinal activation of individual sarcomeres in series, causing them to shorten. Sarcomeres are thus known as the contractile units of skeletal muscle. The summation of all the contracted sarcomeres produces a resultant force; if this force is sufficient to overcome external forces placed on the muscle (for example via its antagonist muscle group and/or gravity) then the muscle will shorten. When the muscle shortens the contraction is termed “concentric” (CON). If

the force produced by the muscle is of equal magnitude to the summation of external forces then no movement occurs, this type of contraction is termed “isometric” (ISO). Finally, if the external forces placed on the muscle exceed its force production then the muscle actively lengthens. This lengthening contraction is referred to as “eccentric” (ECC). Human movement (e.g. locomotion) requires a combination of all three-contraction types.



2.1 Pictorial representation schematic diagram of the sarcomere contractile filaments and cytoskeletal proteins. Adapted from (Fridén & Lieber, 1992).

2.4 Mechanism of muscle damage

Sarcomeres are arranged in series within human skeletal human; however their arrangement is not uniform throughout the entire muscle. When a muscle performs an ECC contraction an uneven load is placed on individual sarcomeres within that muscle. It has been speculated that: greater strain is placed on individual fibres through differences in neural recruitment strategies and uneven force distribution causes some fibres to elongate past their myofilament overlap (Proske and Morgan 1990; Fridén et al., 1983; Fridén & Lieber, 1992). Morgan and colleagues postulated that this overstretching causes susceptible sarcomeres to “pop”, Morgan named this process “*the popping sarcomere theory*” (Morgan et al., 2001). After numerous eccentric contractions, it is postulated that overstretched myofilaments may fail to reintegrate (Proske & Morgan, 2001) accounting for an immediate reduction in maximal voluntary (MVC) contraction force production.

A greater magnitude of damage is induced by contractions performed at a higher velocity, when controlled for the amount of time under tension (Chapman, et al., 2006). Each fibre is thought to be placed under greater mechanical stress when the velocity of the contraction is increased. It is thought that following ECC contractions, actin-myosin bonds are mechanically torn apart as opposed to ATPase enabled disengagement (Edwards, et al., 1977). This mechanical tearing may cause cellular disruption of the sarcomere and allow an efflux of intracellular proteins into the intercellular space. (Armstrong, 1990; Fridén & Lieber, 1998). This overstretching and mechanical tearing is thought to occur on the descending limb of the length tension curve (See figures 2.2 and 2.3) (Gordon et al., 1966). Sarcomeres stretched beyond their overlapping filaments are

no longer able to contribute to force development.

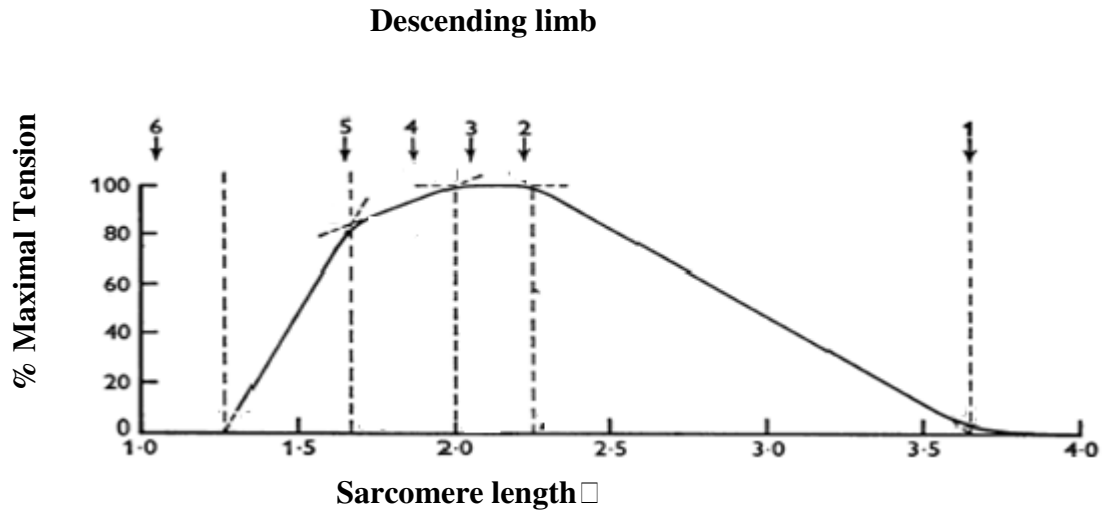


Figure 2.2 The relationship between length and tension in skeletal muscle. Adapted from Gordon et al. (1966), p.185.

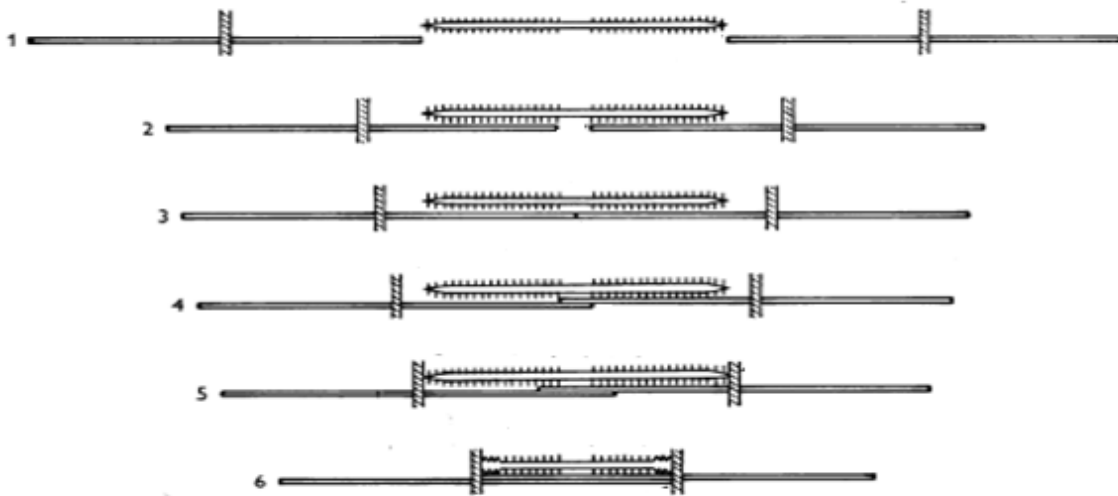


Figure 2.3 Critical stages in the increase of myofilament overlap corresponding to key points (1–6) labeled on the length-tension curve in figure 2.2. Adapted from Gordon et al. (1966), p.186.

2.5.1 Cellular Response to Exercise Induced Muscle Damage.

The “popping sarcomere theory” states during ECC contractions an uneven yield tension of individual sarcomeres causes weaker fibres to lengthen at a much greater rate than adjacent stronger fibres. Under a repeated contraction model it is assumed that some fibres may be stretched beyond the point of any overlap between its actin and myosin filaments (Morgan, 1990). Failure of popped myofibrils to reintegrate places greater load on neighboring myofibrils at the sarcomere, tending to cause a “tearing” of the sarcomere (Morgan et al 1990).

Direct cellular response to tearing is examined using histological analysis of biopsies taken from the affected muscle. Friden’s report was one of the first to identify the evidence of cellular disruption post damage. They observed disturbances in myofibrillar, streaming of Z-band, loss in thick filaments and total Z-band disintegration (Friden et al 1981, 1983). Damage to these areas has lead researchers to postulate that the Z-bands are the “weak link” in the myofibrillar chain (Newham, et al., 1983). Increased synthesis of desmin and titin indicate the presence of damage and reorganization of the cytoskeleton (Friden 1984). It is difficult however, to delineate if the results are indicative of degenerative or regenerative activity. Yu reported that changes detected in desmin cytoskeleton are mainly due to an increased synthesis of desmin and remodeling of myofibrils rather than degeneration (Yu & Thornell 2002). Some evidence of sarcomerogenesis in rabbits (formation of new sarcomeres) has been reported in the whole muscle but individual areas of the muscle respond differently (Butterfield & Herzog, 2006). Sarcomerogenesis has not been reported in human studies as yet. In humans, care must be taken in interpreting the results of muscle biopsies as it has

been reported that the procedure may induce changes within the muscle structure (Malm et al., 2000). The effect of cellular damage following ECC exercise may have a direct effect on athletic performance through reduction in muscle function.

2.5.2 Failure of Excitation Contraction coupling:

Mechanical damage to sarcomeres through tension or shearing stress leads to damage of the contractile proteins. It has been postulated that during ECC contractions there is a failure of actin and myosin filaments to be separated by ATPase, rather they undergo a mechanical tearing (Morgan & Allen, 1999). Disruption to the sarcoplasmic reticulum may increase (Allen, 2001) An increase in membrane permeability is understood to be responsible for increases in intracellular Ca^{++} concentrations (Armstrong, 1990). Ca^{++} concentration increases can contribute to damage by stimulating the release of calcium-activated neutral proteases such as calpain, which have been shown to damage Z-line-associated proteins (Busch, et al., 1972) (Belcastro, 1993). Excitation-contraction coupling failure and titan degradation may occurs following prolonged elevation of Ca^{++} level in toad skeletal muscle from activation of activation of endrogenous calpains (Verburg, et al., 2005). Cell membrane damage also has a Ca^{++} component; removal of extracellular Ca^{++} improves muscle function post EIMD (Zhang, et al., 2008). The full extent of sarcoplasmic reticulum involvement in muscle damage is not known; Neilson et al (2005) reported no changes in function or damage in human skeletal muscle. Yu also reported increases in CK, LDH and reduction in MVC following 3 different modalities inducing EMID and yet reported no signs of sarcoplasmic reticulum damage (Yu et al., 2002).

2.5.3 Inflammation:

Eccentric contractions through the mechanisms described above produce disruption to the cellular structure. Inflammation occurs in response to this disruption by the infiltration of inflammatory cells such as neutrophils and macrophages within the exercised muscle. Neutrophils main function is to contain and destroy damaged tissue through phagocytosis, respiratory burst, and degranulation (Butterfield, 2010). Within 1 hour of EIMD, neutrophil numbers increase, 24 h later neutrophil levels remain elevated but start to decrease and concentrations of macrophages increases (Tidball, 2005). This reaction to EIMD has been implicated in the secondary cytoskeletal disruptions to exercised muscle (Pizza et al., 2005).

One of the main mechanisms for inflammation is the influx of extracellular Ca^{++} in response to ECC exercise. It has been reported that a reduction in membrane integrity and cytoskeletal disruption can be avoided by blocking the influx of calcium via stretch-activated ion channels during ECC exercise (Willems & Stauber, 2005). Currently it is believed that after EIMD pro-inflammatory cytokines are released by fibres; only after membrane disruption, resulting in localization of neutrophils at these injured cells (Tidball, 1995). This view dictates that the inflammatory response occurs after the cellular membrane disruption, however Pizza et al (2002) reported localization of neutrophils within the extracellular matrix of non-damaged skeletal muscle in response to isometric contractions. This evidence suggests the inflammatory response can occur in the absence of or preceding cytoskeletal disruption (Pizza et al., 2002).

2.6 Indirect markers of muscle damage:

Direct analysis of EIMD in animals has been done but it is unknown the extent to which results from animal studies can be generalized to humans. Animal models such as those used by Butterfield et al (2006) involve anesthetizing animals (normally rats, mice or rabbits) attaching them to a fixed apparatus and electronically stimulating a muscle action (Butterfield & Herzog, 2006). After the muscle has been damaged through eccentric repeated contraction the animal is usually destroyed and the muscle dissected for analysis of damage. In human studies direct analysis of cellular damage is achieved via needle muscle biopsies; whereby a small piece of muscle is removed and analyzed. The biopsy technique may itself cause damage and confound any measurement changes observed. As a result, more investigators are using indirect markers of muscle damage to access structural changes in response to EMID. These markers give a view of the systematic response to ECC contraction; CK, LDH and changes in force generation are typical examples of measures used.

2.6.1 Changes in Force Output

A change in force output of the muscle is generally accepted as one of the most reliable ways of measuring the magnitude and time course of EIMD indirectly (Warren, et al 1999). Frequently, the MVC is used as a measurement tool for assessing damage (Warren 1999). Maximal contractions of any type cause an immediate reduction in MVC through fatigue or damage. However, muscle function recovers very quickly post isometric or concentric contractions, whereas repeated ECC contractions triggers a suppression of force output for an extended period of time i.e. 15 days. (Newham et al.,

1987) The extent of functional depression is dependent on the severity of damage. Paulsen et al (2012) suggest grouping results of EIMD in 3 classifications depending on the extent of damage “mild, moderate and severe”. “Mild” EIMD is classified as a below 20% reduction in force-generating capacity (during the first 24 hours) and/or full recovery at 48 hours. “Moderate” is defined as 20-50% force reduction and/or full recovery between 2 – 7 days. “Severe” EIMD is a loss of greater than 50% of force generating capacity, with a recovery period exceeding 7 days (Paulsen et al., 2012). The rationale behind such a classification system is the variability in force-generating capacity to the mode of exercise, and the correlation of MVC to other indirect markers (Paulsen et al., 2012). Small grade such as -8% downhill running and submaximal eccentric contractions of the lower limbs often results in mild EIMD (Byrne & Eston, 2002a; Donnelly et al., 1990). Moderate amounts of muscle damage have been reported post 50 maximal jumps (Albertas, 2010); low number (24) of maximal ECC contraction of elbow flexors (Nosaka et al., 2007); and 10 x 10 bodyweight jumps (Jakeman et al., 2009). Severe damage has been reportedly induced by maximal eccentric contractions of the biceps brachii (Chen, et al., 2010) and marathon running (Sherman et al., 1984). Strength loss post EMID seems to peak within the first 24 h after exercise and recovery (Clarkson & Hubal, 2002) The immediate and prolonged loss of force output in response to eccentric contractions negatively affects athletic ability and is of main concern when training athletic populations.

2.6.2 Blood protein markers:

Blood circulating muscle enzymes are frequently used for assessing and diagnosing EIMD. Lactate dehydrogenase, myoglobin and CK are examples of proteins

tested, with CK being the most popular (Warren et al., 1999). When rapid active lengthening compromises the structural integrity of the muscle sarcolemma, an influx of CK into the bloodstream can occur (Armstrong, 1990). Circulating CK concentrations are a result of two processes (1) what is being produced in the muscle, and (2) the rate of its clearance from the blood stream. There is no way to determine which process is being measured; physical activity and increased blood flow post damage may influence the level of response (Saxton & Donnelly 1995). CK concentrations and time of peak concentration in response to EIMD are largely dependent on the mode of damage. EIMD induced by downhill running, plyometrics, or resistance training show significantly lower concentration (approximately 100 IU to 600 IU/liter) peaking 12-14 h post exercise (Paulsen et al., 2012). Whereas high force, full range, maximal eccentric exercise causes CK levels between 2000 IU and 10,000 IU/liter, peaking 4-6 days post contractions (Clarkson et al., 1992). Using analysis of circulating muscle proteins as an indicator of the magnitude of damage is made particularly difficult due to the large inter-individual variability in response (Nosaka, 1996). This large inter-subject variability means circulating concentrations of CK do not correspond well with the force loss or delayed onset muscle soreness.

2.6.3 Delay onset muscle soreness (DOMS)

Delayed onset muscle soreness (DOMS) is a sensation of dull, aching pain, usually felt during the movement or palpation of the affected muscle (Nosaka et al., 2002). It was first described by Hough in 1902 and is the most commonly used method for evaluating EIMD, with over 70% of studies conducted on humans taking DOMS measurements (Warren et al., 1999). Although it has been extensively researched the

exact mechanisms behind DOMS have not yet been established. DOMS usually presents itself 8-10 h post damage and peaks around 24-48 h post-exercise (Armstrong, 1984; Clarkson et al., 1992; Lieber & Friden, 2002). It has been well established that DOMS is a main outcome of EIMD (Newham 1998; Jones & Round, 1990) and that it is a product of muscle and tendinous damage or ensuing an inflammatory response (Armstrong, 1984; Clarkson et al., 1991; Lieber & Friden, 2002). Time course for DOMS exhibits little to no relationship to severity of muscle damage, CK, changes in bicep/tricep circumference or flexed elbow joint angle (Nosaka, et al., 2002). Functional changes in skeletal muscle are almost immediate and can be suppressed long after DOMS has subsided (Jones et al., 1986). Nosaka et al (2002) illustrated that individuals did not report a greater sensation of soreness when performing exercise protocols that induce a different amounts of damage; 110 subjects were divided in 3 groups who performed 12, 24 or 60 ECC contraction of the elbow flexors. Results showed a difference in other indirect markers of muscle damage such as Maximal isometric force and relaxed joint angle and arm circumference with no statistical difference between groups for DOMS (Nosaka et al., 2002).

Several theories have been put forward to explain the pain associated with delayed onset muscle soreness (DOMS). The combination of the *muscle damage theory* proposed by Hough in 1902; and the *inflammation theory* based on findings that aspects of the inflammatory response are evident following repetitive eccentric muscle action may provide a more complete answer. The muscle damage theory dictates that nociceptors situated in the muscle connective tissue and in regions of arterioles, capillaries and the musculotendinous junction are also stimulated leading to sensation of pain (Cheung, 2003). The presence of delayed onset muscle soreness is thought to adversely affect

dynamic muscle function (Proske et al., 2003). However the time course for initial decrements in function do not mirror the peak in DOMS; meaning the muscle damage theory cannot provide a full explanation for onset of DOMS.

Invasion of inflammatory cells and subsequent swelling as a result of EIMD are accepted as a major contributing factor to DOMS. It is proposed that the swelling associated with EMID increases the internal tissue pressure of muscle (Friden et al, 1986). It is thought that increases in intramuscular pressure, when altered further via contraction or palpation, are adequate to stimulate mechanical nociceptors (Smith, 1991). When cells are altered in response to EIMD, they produce noxious chemicals such as histamines, bradykinins and prostaglandins. The release of these chemicals may be responsible for activation of type III and IV nerve afferent fibres. These fibres carry messages of pain from the muscle to the central nervous system, which leads to the sensation of pain (O'Connor et al., 1999).

More research is needed to elucidate the exact mechanisms causing DOMS and its presence should not be used as a measure for the magnitude, progression or recovery of damage post-eccentric exercise. Many researchers have investigated the effects of different interventions on the sensation of DOMS with varying success (Akamoto, et al 2010: Khamwong, et al 2011: Tseng et al., 2012). Akamoto et al., (2010) reported a significant reduction in DOMS after repeated dumbbell bicep curls at 70% MVC (Akamoto et al., 2010). Following the damaging protocol researches reported that additional muscle actions did alleviate the severity of DOMS, however this was accompanied by a further reduction in MVC (Akamoto et al., 2010).

2.7 Changes in muscle function

Exercise-induced muscle damage (EIMD) in response to eccentric or lengthening contractions generates a series of functional changes to skeletal muscle. Changes in contractile filament overlap, increased serial compliance, compromised force generating capacity and altered neurological control are some of the early events accompanying EIMD (Clarkson et al., 1992; Peake et al., 2005; Proske & Morgan, 2001).

2.7.1 Changes in optimal length

The length-tension relationship of skeletal muscle refers to the amount of force produced by a muscle at a given length (Kats 1939). The “optimal length” refers to the length at which a muscle can produce its maximal force. Optimal muscle length is determined by the joint position (optimal joint angle), which creates the greatest overlap between contractile proteins within sarcomeres. A change in a muscles optimal length leads to a greater reduction in optimal joint angle. Alterations to the length tension relationship was first observed in frog and tortoise muscle following eccentric contractions (Katz, 1939).

Studies in humans have yielded corroborating results (Jones et al., 1997; Brockett et al., 2001; Morgan & Allen, 1999; Proske & Morgan, 2001). Morgan et al (1990) hypothesized that overstretched sarcomeres increase overall series compliance, leading to a shift in length-tension direction to a longer muscle length. If as suggested, the primary cause of force reduction post EIMD was E-C coupling failure (Warren et al 1993); then a muscle would need to increase its overall length to achieve the same contractile fibre overlap (Morgan & Allen, 1999). Figure 2.4 represents this change in torque angle

relationship. The magnitudes of change to the optimal angle are not uniform; a shift to a larger joint angle of 10 – 18 degrees has been reported post-eccentric exercise (Phillippou et al., 2004). The change is thought to occur during or immediately after cessation of exercise; a 10-degree shift in the position at which peak eccentric force is produced immediately following exercise (Donnelly & Brown 2011). Changes to optimal muscle length have been induced by protocols that do not elicit any other indirect markers of muscle damage. In a study by Brughelli (2010) they induced a significant increase in optimal angle of both knee flexors and extensors over a 5-week period without the presence of DOMS (Brughelli et al., 2010). Further current literature supports a rightward shift in optimal angle with a greater loss of strength at a shorter relative muscle length following ECC exercise being reported (Brown & Donnelly, 2011; Byrne et al., 2001).

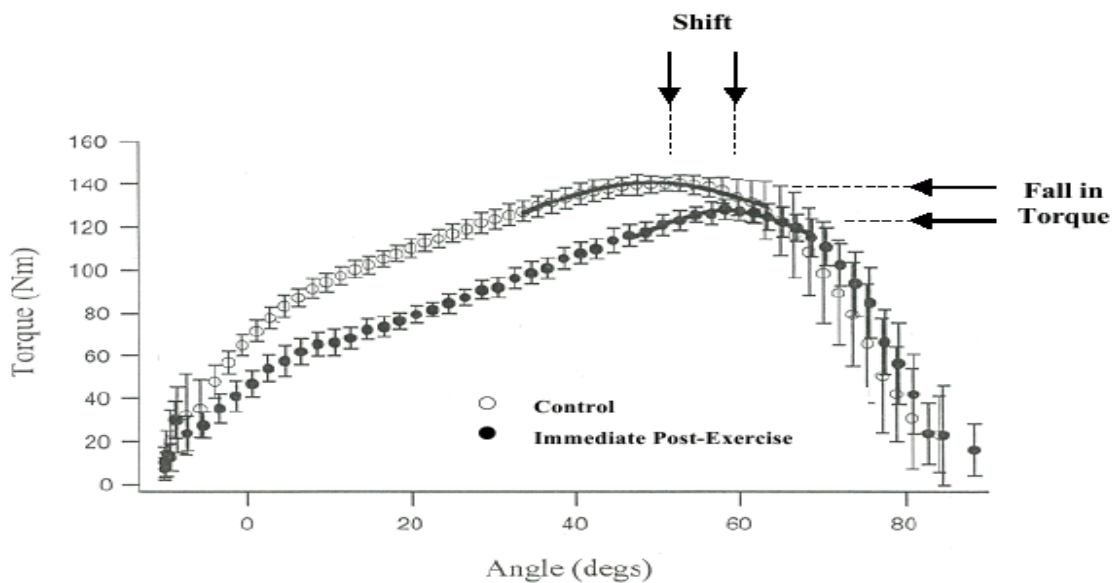


Figure 2.4 Hamstrings angle-torque curves before eccentric exercise (Control) and immediately post exercise. Gaussian curves have been fitted to the top 10% of each curve. Adapted from Brockett et al., (2001). Note the immediate right shift of the angle- torque relationship following eccentric exercise.

2.8 Dynamic Muscle Function

Isometric, isotonic and isokinetic tests of muscle function are effective tools in evaluating the magnitude of EMID (Warren et al., 1999). However they lack any kind of real world application. In any athletic setting, it is of the utmost importance that an athlete can exert the maximum amount of force possible. EIMD negatively affects the force generating capacities of the muscle (Byrne & Eston, 2002b); sprint performance (Twist & Eston, 2005) peak power, (Twist & Eston, 2009) and vertical jump height (Byrne et al., 2001). EIMD induces DOMS and increases the rate of perceived exhaustion, both of which have negative consequences for athletic performance.

2.8.1 Vertical Jump

Several studies have used jump protocols of different intensities as a means of inducing muscle damage because of the large ECC portion (Byrne & Eston, 2002a). Others have used vertical jump as a performance measure post EIMD (Byrne & Eston, 2002a). Results from these studies have provided evidence to suggest that different mechanism involved with each jump may be more or less susceptible to damaging ECC contractions. Typically three different jump types are examined in current research; Counter-movement, Squat and Drop jump. The three different squat variations in jump types allow researchers to examine the effect the stretch-shortening cycle (SSC) has on attenuating the effects of muscle damage (Byrne & Eston, 2002a).

A counter-movement jump (CMJ) employs a loading phase in which the elastic potential of the muscle is utilized in order to produce more force. The pre-activated muscle is first stretched and then followed by the shortening action (Nicol et al., 2006).

When using the SSC as in CMJ model, participants have been able to achieve higher pre-exercise jump heights when compared to using a SJ model. Following EIMD the use of SSC has been shown to attenuate some of the changes in force output (Horita et al., 2003).

The second jump type is Drop Jump (DJ), which employs the greatest amount of SSC. DJ requires the participant to jump down from a predetermined height and then explode maximally upwards. A reported depression in DJ performance for 2 – 4 days following EIMD when SJ returned to baseline after just 10 minutes (Horita et al., 2003). EIMD causes an increase in landing time during a DJ. This is thought to be due to decreases in strength, reflex activity and joint stiffness (Bryne & Easton 2002b).

The third jump type is Squat Jump (SJ); this movement involves squatting down and pausing in squat position before the CON portion of the jump is executed. This protocol does not utilize SSC mechanisms and has been shown to produce less force in the CON phase (Komi, 2000). Squat Jump has been reported to result in a lower initial jump height; and evidence suggests it maybe more susceptible to negative effects of ECC contractions (Bryne & Easton 2002b).

Several researchers have reported a bimodal pattern of recovery for peak power (Horita et al., 1999; Avela et al., 1998; Nicol et al., 1991a). Following an initial decrement in performance found immediately post-exercise, jump performance decreased again some hours afterward, with the greatest decrement in performance occurring 48 h post exercise bout. This second reductions in peak power loosely mirror the development of DOMS suggesting it may have an influence over maximal performance. The reasons

for a different recovery pattern when comparing a static or isometric condition to a ballistic one are not yet fully understood. The stretch-shortening reflex has been postulated to attenuate some of the effects of EMID (Akamoto et al., 2010). However it has also been suggested that subjects may be unwilling to provide maximal effort in the presence of DOMS (Sakamoto et al., 2009).

2.9 Attenuation of Damage and Recovery

Attempts to attenuate the level of damage inflicted by eccentric muscle from subsequent muscle damage contractions have been shown to confer a protective effect (Nosaka & Clarkson 1995) this process is termed the repeated bout effect (RBE). Varying levels of protective effect are conferred after exercise depending on type of the initial exercise bout, maximal ECC only contraction, or high velocity ECC contractions can induce a protective effect for up to a year (Nosaka et al., 2001a). Conversely as little as 2 submaximal eccentric contractions provide a protective effect for approximately 14 days (Nosaka & Newton 2002). Nosaka et al, 2001 reported the greater the magnitude of damage sustained from the initial bout the longer the protective effect is conferred. Other preemptive methods of attenuating EIMD have been investigated; PNF stretching performed prior to a muscle damage protocol involving the forearm muscles attenuated the severity of DOMS, active ROM and MVC (Khamwong et al., 2011). Microwave diathermy heating of the muscle to around 40 degrees Celsius 16 h prior to exercise does not help attenuate the severity in performance of MVC post EIMD; it may however improve the rate of recovery (Nosaka et al., 2007). These strategies rely on re conditioning the muscle in an attempt to minimize the magnitude of EIMD.

Other approaches: focus more on expediting the recovery process after damage, once it has already occurred (Coffey et al., 2004) (Coffey et al., 2004; Crowe et al., 2007). This is important to sporting populations who despite large amounts of physical preparation; the intensity of their respective sporting endeavors will result in muscle damage. Several different recovery strategies have been investigated in an attempt to accelerate the recovery from EIMD such as active recovery (Coffey et al., 2004), temperature regulation be it heat or cold (Jayaraman et al., 2004; Crowe et al., 2007), massage and compression garments (Davies et al., 2009). These recovery strategies usually revolve around the manipulation of blood flow to the damaged muscles. Massage, heat application and exercise area thought to increase blood flow and accelerate the recovery process. Interventions using ice and compression garments are thought to reduce blood flow to the affected muscles and minimize secondary damage induced by neutrophil proliferation.

2.9.1 Effect of Massage on Exercise Induced Muscle Damage

The use of massage to attenuate the indirect markers of muscle damage is a popular modality amongst athletic therapist. Its effectiveness in alleviating DOMS has been established, however no changes in neutrophil or muscle function accompanied the decreased pain sensation (Bakowski et al., 2003). Corroborating these results Zainuddin et al (2005) reported that 10 minutes of massage post-maximal ECC contractions of the elbow flexor attenuated changes in CK, DOMS and swelling without attenuation of any performance markers. Authors postulated this was due to increased blood flow in response to massage; however Shoemaker and Tiidus 1995 reported no changes in localized blood flow of the quadriceps after manual massage. Recently published data

indicates that massage maybe clinically beneficial by reducing inflammation and promoting mitochondrial biogenesis (Crane et al., 2012). Further research needs to examine the exact mechanisms by which massage attenuates EIMD and if it can be used as an effective tool for regaining athletic performance post EIMD.

2.9.2 Cryotherapy, ice and cold water immersions effect on symptoms of exercise induced muscle damage

The effect of cold water immersion, cryotherapy and direct icing in attenuating symptoms of EIMD have also been investigated with varying results (Hauswirth et al., 2011; J R Jakeman et al., 2009; Pointon, et al., 2011). Using different modalities to reduce the temperature of an affected area has been shown to have an analgesic effect (Cheung, et al., 2003), however the effect of cooling on muscle performance is not well established. Icing has been found to have no effect on recovery of muscle function, but may have some effect on blood markers and DOMS (Eston & Peters, 1999; French et al., 2008; Pointon et al., 2011). There is some evidence of increased muscle function following cold-water immersion verses passive recovery (Vaile et al., 2011). Pointin et al., in 2012 reported cold water immersion after collision-based exercise improved acute recovery of MVC (Pointon & Duffield, 2012). Theoretical implications of massage and cooling seem to elicit different responses in tissue blood flow, massage is thought to increase blood flow via vasodilation and cooling restrict blood flow via vasoconstriction. More research with standardized damaging and timing of treatment protocols is needed to further examine the physiological effects of both modalities. Caution should be taken when prescribing ice as a means of recovery as recent evidence indicates it can impedes recovery by delaying the inflammatory and healing response (Tseng et al., 2012).

2.9.3 Effect of wearing compression garments post exercise on recovery

Compression garments worn during exercise and subsequent recovery process has been reported to modulate varying results in symptoms of EIMD. Jakeman et al in 2010 reported compression garments could moderate strength loss and diminish perceptions of soreness following exercise (Jakeman, et al., 2010). These results are supported by John et al (2010) who reported a significant attenuation of damage symptoms following plyometric jumps via the use compression garments (John & Roger, 2010). Contradictory evidence has also been reported citing no change in recovery rates from EIMD by using compression garments. Duffield and colleagues (2010) reported wearing lower limb compression garments had no effect on muscle function, blood lactate or pH levels. This evidence supports data published by French et al in 2008 who reported no attenuation of symptoms of EIMD in young resistance trained men (French et al., 2008).

2.9.4 Employment of an active recovery model to accelerate recovery post exercise

The use of an ART is generally accepted as an effective means of attenuating the severity of, as well as aiding in the recovery from, EIMD. However, there is a dearth of information pertaining to the effectiveness of different activity modalities. Results from existing literature have not come to a consensus. Whereas cryotherapy aims to reduce blood flow to the limbs, ART is designed to increase blood flow in an attempt to aid recovery (Vaile et al., 2011). The transient prophylactic effect of exercise on symptoms of EIMD is term exercise-induced analgesia (EIA). It is characterized by a decrease in severity of DOMS and also a reduced performance measures presumably through fatigue (Sakamoto et al., 2009). This phenomenon must be taken into account when designing the

testing of recovery modalities. If the sequence of testing and treatment is arranged so that performance and pain measures are taken after a treatment of active recovery, it may influence results. In that EIA being a transient effect may influence the testing done immediately after an exercise. The effects of EIA being transient do not reflect overall recovery status of the muscle affected by EIA (Zainuddin, et al., 2006). Experimental evidence supporting the use of an active recovery model has shown some promising signs. Evidence shows that a swimming recovery implemented 10 h after high intensity running session, results in a significant performance on time to fatigue test the following day (Lum, et al, 2010). Evidence to suggest a greater change in CK clearance via the use of active recovery and compression garments. Care should be taken in interpreting these results as aiding recovery from EIMD as CK has been shown to have a poor correlation with performance (Gill, et al., 2006). Conversely 30 minutes of flat running daily, post-downhill running did not aid nor delay recovery (Chen, et al., 2008). Periodization of active recovery in the seven days post-marathon did not aid in recovery of work capacity or muscle peak torque production (Sherman, et al, 1984).

Engaging in resistance training post-ECC contractions has been reported to neither exacerbate nor aid in the recovery process. Research suggests subsequent contractions after EIMD do not exacerbate the levels of damage when using sub-maximal CON and ECC muscle actions were performed (Akamoto et al., 2010; Donnelly, et al 1992; Gulick, et al, 1996; Zainuddin, et al., 2006). Chen & Nosaka, 2006 also reported that engaging in ECC activity 3 days after damaging activity did not induce further damage and nor did it delay recovery. This evidence is contradicted by Sakamoto who in 2009 reported the delay in the recovery process induced by subsequent bouts of exercise in the 7 days post

damage (Sakamoto, et al, 2009). At present no research was found pertaining to the effect of submaximal, concentric only endurance type exercise on damage induced by resistance training.

Purpose:

- The purpose of this study was to determine whether ART would attenuate the severity of indirect markers of EIMD and increase the rate of recovery of muscle function.
- Examine the effect the SSC has on muscle function following EIMD.

Hypotheses

- Participants who engage in an ART will not be as adversely affected by EIMD. Post EIMD 1RM, jump height and rate of perceived exertion will return to baseline values in the CG before the NCG.
- The use of the SSC in counter-movement jump will mean: Squat jump performance will decrease more post EIMD when compared with counter-movement jump performance.

3. Methods:

3.1 Participants

Twelve healthy active men aged 23 ± 1.5 years were recruited from the Western University population. The subjects comprised 2 groups, participants who completed eccentric lengthening contraction and then cycled intermittently for 4 days (CG, N = 5), and participants who completed lengthening contraction followed by a passive recovery (NCG, N = 5). The eligibility criteria for participants were a training frequency of at least 1 leg specific session per week over the past 3 months, have used a Hammer Leg Press before, did not compete in a varsity level sport and had no lower limb injuries. Participants were randomly allocated into each group. The study protocol was approved by the University of Western Ontario Research Ethics Board for Health Science Research Involving Human Subjects (See appendix A) and conformed to the Declaration of Helsinki. Written and oral consent were obtained after the whole procedure was explained and prior to any testing.

3.2 Protocol Overview

Participants reported to St Joseph's Hospital after 7pm on Monday to complete their pre-testing magnetic resonance images (MRI). Prior to the first MRI session, participants were given a MRI screening questionnaire to ensure safety (See appendix B). After participants had completed their scans they were shuttled by an experimenter back to the gym for exercise and testing. Immediately prior to all initial testing protocols participants were run through a familiarization set to ensure maximal effort. Figure 3.1 illustrates the

sequence of tests performed on Day 1. Figure 3.2 represents the sequence of testing for the NCG and the CG completed at 24, 48, 72 and 96 h post lengthening contractions. The testing protocol was arranged in order to control for the fatiguing effect of the MVC testing. The CG completed 36 minutes of moderate intensity cycling after the performance test so that exercise -induced analgesia could not affect performance results.

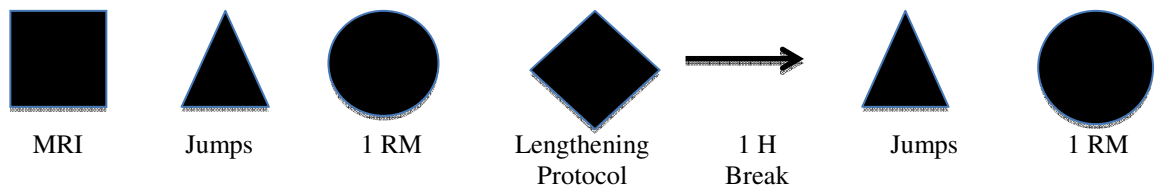


Figure 3.1: Represents the sequence of testing for every participant on day 1.

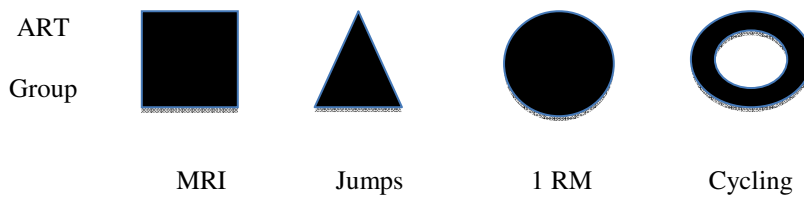


Figure 3.2: Represents the sequence of testing for the 5 ART participants on days 2,3,4 and 5.

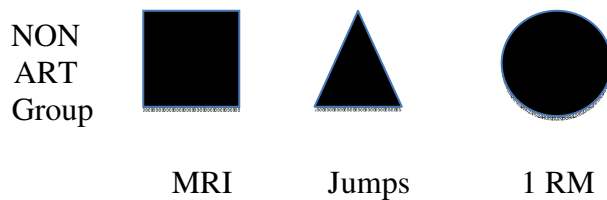


Figure 3.3: Represents the sequence of testing for the 5 non ART participants on days 2,3,4 and 5

3.2.1 Jumps: Counter movement jump - Non-counter movement Jump

Maximal jump height was assessed using a custom-built accelerometer platform. Subjects placed a hernia prevention belt on the hips so that the buckle was situated 2 cm below the umbilicus. The accelerometer was connected to the buckle to ensure when the subjects squatted the cable did not make contact with the body (Fig 3.4).

For each Squat jump subjects were instructed to 1) Keep their hands on their hips. 2) Maintain an upright body position. 3) Keep their toes on the midline tape. 4) Squat down to 90 degrees of knee flexion and hold. 5) On the count down of 3..2..1 jump for maximal height. 6) Trying to jump as vertical as possible making minimal horizontal movement.

For counter movement jumps subjects were instructed to. 1) Keep hands on their hips. 2) Maintain an upright body position. 3) Keep toes on the midline tape. 4) On the count of 3..2..1 squat down to 90 degree and explode straight back up with no pause. . 5) Trying to jump as vertical as possible making minimal horizontal movement. A randomized sequence of jumps was achieved by placing 40 pieces of paper into a box, half with squat jump and half with counter-movement jump written on them. Before each trial a piece was pulled out of a box to determine which jump was first. A total of 6 trials were completed per session 3 squat jumps and 3 counter-movement jumps. A 2-minute rest break was given between each trial. The jump that had the greatest peak height was recorded and analyzed.



Figure 3.4 Experimental set up for Jumps protocol. Linear transducer attached to the waist of the subject.

3.3 One repetition maximum:

Measures of 1RM were obtained using a Hammer strength unilateral leg press machine (fig 3.5). A uniformed spot was placed on the footpad of the machine to ensure that all subjects were similarly positioned. The optimal seat height was achieved by ensuring that the participant's thighs were perpendicular to the ground. This position created a knee joint angle of less than 90 degrees to ensure a greater range of motion while maintaining contact between the participants lower back, buttocks and the seat padding. Participants were instructed on a three second countdown to push the weight until the knees were fully extended. A 3-minute rest was given between each attempt. The starting mass was 22 kg for each leg, and went up incrementally at the tester's discretion until the subject failed twice at a certain weight. The 1RM was then determined to be the last mass that the subject was successfully able to lift through a full ROM. Post-lengthening protocol, every 24 h the subjects retested their 1RM using this protocol.



Figure 3.5 Hammer Strength Leg Press Machine used for lengthening contraction and one repetition maximum.

3.4 Lengthening protocol:

In order to instigate damage participants underwent a ECC protocol consisting of 10 sets of 10 repetitions with a one minute break between each set. This ECC protocol was performed on the same Hammer Strength Leg Press machine used to test 1RM. Once the participant's 1RM was established, 10% of the total 1RM mass was added. Four experimenters lifted the individual weight stacks until the participant's legs were nearly straight, (5 – 10 degrees bend at the knee). The experimenters then released the weights and the participants lowered the weight on the count of 3. The participants were verbally instructed to provide a maximal contraction in attempt to keep the duration of every repetition to 3 seconds; they were also encouraged to lower both legs at the same time. Upon completion of each repetition, the weight was returned to the start position by the experimenters and the participants kept their feet on the pads. The participants were given 5 – 10 trials with no added mass to practice the protocol.

3.5 Cycling (Recovery)

On day one, participants reported to the laboratory to perform a ramp incremental test (20-25 W/min) to the limit of tolerance on a cycle ergometer (model: H-300-R Lode; Lode B.V., Groningen, Holland) for determination of peak VO_2 ($\text{VO}_{2\text{peak}}$) and GET; the ramp portion of the protocol was initiated following 4 minutes of cycling at 20 W. Participants were asked to maintain a cycling cadence between 60-70 rpm throughout. GET was determined by visual inspection as the VO_2 at which CO_2 output (VCO_2) began to increase out of proportion in relation to VO_2 , with a systematic rise in minute ventilation-to- VO_2 ratio and end-tidal PO_2 whereas minute ventilation-to- VCO_2 ratio and end-tidal PCO_2 were stable. On days two, three and four subjects were required to cycle for 36 minutes at 60% of gas exchange threshold.

3.6 MR

Indirect measures of muscle damage were obtained from MRI acquired using a 3.0 Tesla scanner (Siemens Magnetom Verio, Siemens AG, Munich, Germany). Participants were inserted into the magnet feet first, in the supine position, with the mid point on their right thigh (at the halfway point between the greater trochanter and the fibrillar head) iso-centred to the bore of the magnet and flex coil placed over the quadriceps. To ensure minimal movement between scans, the feet and knees of the participants were strapped together using inelastic, Velcro straps. Ten, transverse T2 weighted images were acquired using the following parameters: 3500ms repetition time, ten echos of 7.6ms duration, a 256 x 192 matrix with a field of view of 400mm. Voxel size was 3.1 x 3.1 x 5 mm and slices thickness was 5mm.

3.6.1 Transverse relaxation times

T2 relaxation times were calculated pixel-wise using a combination of manual and semi-automated techniques with open-source OsiriX image processing software (version 3.7, Geneva, Switzerland). The slices representing the largest cross sectional area were used for both T2 relaxation time and Cross-Sectional Area (CSA). A 2.4cm squared region of interest (ROI) was manually outlined in a section of muscle contractile tissue for two sites in the quadriceps. Care was taken to ensure connective tissue or adipose tissue was not included in the ROI. The first of the 10 images was deleted, the ROI was outlined on second image then the ROI was automatically propagated by OsiriX over the remaining eight images that corresponds to the first image, the software then generated a T2 Fit Map for each ROI. The same process was repeated at 24, 48,72 and 96 h post lengthening contractions. T2 times were calculated by subtracting the T2 time for a muscle at a given time point from its corresponding baseline value.

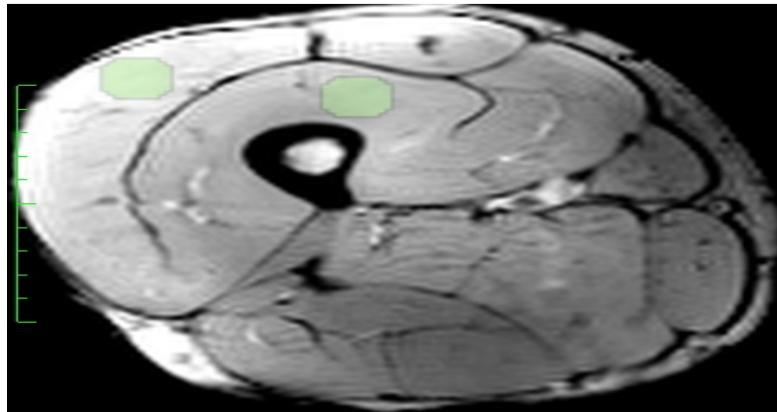


Figure 3.6 Axial Magnetic resonance images of the left thigh; Highlighted regions represent the two sites used to determine transverse relaxation times of the muscle.

3.6.2 Swelling and Cross Sectional Area (CSA)

Total muscle CSA was calculated pixel-wise using a combination of manual and semi-automated techniques with open-source OsiriX image processing software (version 3.7, Geneva, Switzerland). CSA of the quadriceps muscles were calculated at the slice with the largest CSA. Analysis started distally to the knee when the gluteal muscles could no longer be seen. The slice with the greatest CSA was selected; the two-quadriceps muscles were manually outlined with a brush tool and the muscle was automatically filled in. Manual erasing of inter-muscular space and connective tissue was performed to ensure only muscle tissue was measured. The corresponding slice point was selected at each of the four time points 24, 48, 72 and 96 h. Swelling was obtained by subtracting the total CSA of the muscle at each time point from its own baseline value. For example if the CSA of VL at baseline may be 32 cm^2 and at 48 h 35 cm^2 then you would do $35 - 32 = 3 \text{ cm}^2$, this was repeated for all time points. Swelling was originally taken for the four-quadriceps muscle but no difference in CSA was observed for the other two muscles.

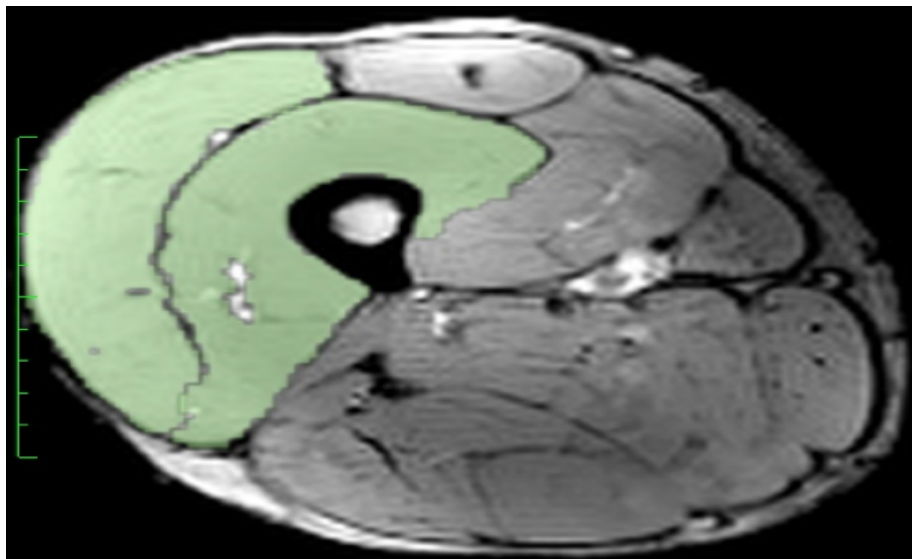


Figure 3.7 Axial magnetic images of the left thigh. Highlighted regions represents the two muscles used to determine swelling

3.7 Rate of perceived pain

A perceived pain rating (PPR) was measured using a Visual Analog Scale (VAS), participants were instructed to indicate how painful performing the trial was. Pain scale ratings were taken at the end of each jump and MVC attempt. Participants were asked to point on a spot on a line between 0 – 10. Zero indicated no pain experienced and 10 represented the worse possible pain (appendices # C). A different line was used to represent each attempt to ensure the participants had no reference from other jumps. Individual PPR was measured after each jump and MVC attempt. The PPR corresponding with the jump and MVC trials used were kept and analyzed.

3.8 Statistics

All data were analyzed using SPSS software (version 18, SPSS Inc. Chicago, Illinois). Univariate analyses of variance (ANOVA) were performed to identify the difference among groups at each time point. A two way repeated measure ANOVA was performed on one repetition maximum; squat jump and counter-movement jump performance. Two ways ANOVA was run between CG and NCG. Two way repeated measure ANOVA were run on T2 and swelling changes in the VL and VI muscles. All ANOVA were performed to determine a between CG and NCG group interaction over time. A Tukey post hoc test was performed when there was a significant between-group effect to determine which groups differed from one another. Pairwise ttests were performed to determine mean difference between each group at each time point. All results are reported in test as group means and level of significance was $p < 0.05$ as well as \pm the standard deviation (SD).

Chapter 4 - Results

4.1 Participant Characteristics

Table 1 represents the means and standard deviations of the two groups for age, height and mass. There was no significant difference for age, height and weight between the two treatment groups ($P > 0.05$).

Table 1. Subject Characteristics

Group	Age (years)	Height (cm)	Weight (kg)
Cycling (n = 6)	23 ± 1.8	182 ± 7	84 ± 10.7
Non-Cycling (n = 6)	23 ± 1.4	176 ± 5.7	83 ± 8

* Sign denotes a statistical significant difference

There were no statistically significant results for any performance measures between the CG and NCG ($P < 0.05$). * symbol denotes a statistical difference in the group relative to its own baseline values. † symbol denotes a statistical difference between the two treatment groups. All bars depicted in the figures represent standard deviations for each treatment group.

4.2 Transverse relaxation time for Vastus Lateralis muscle

Figure 4.1 represents a significant increase in relaxation time for the NCG at all time points compared to baseline ($P < 0.05$). T2 baseline results were 36.41 ± 1.82 and 36.26 ± 1.03 msec for CG and NCG respectively. The greatest increase in T2 was observed at 24 h in the NCG of ~14% this was not significantly different from the CG ($P > 0.05$). A significant difference of ~6% was observed between the two treatment groups at 48 h ($P = 0.03$).

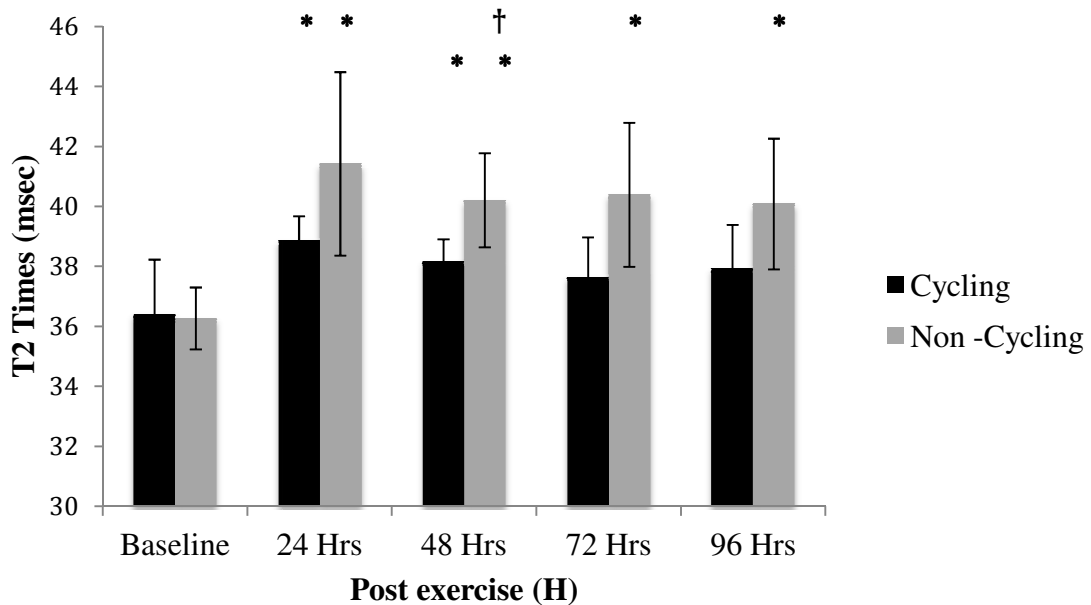


Figure 4.1: T2 relaxation times for the Vastus lateralis muscle for both treatment groups over time. Bars represent SD for each group. † Denotes a statistical difference between the two treatment groups. * Denotes the NCG statistical different from baseline.

4.3 Transverse relaxation time for Vastus Intermedius muscle

Figure 4.2 shows a change in T2 relaxation time for VI; baseline values were 36.2 ± 0.8 and 36.7 ± 0.8 msec for CG and NCG respectively. T2 times for VI changed significantly at 24 h for both treatment groups ($P < 0.05$). The 6.2 % difference between the means of treatment groups is also significant ($P = 0.031$). The NCG mean remained elevated 11.4% above baseline for the entire 96 h. ($P < 0.05$). The CG average returned to baseline by 72 h creating a 10.6% and 9.5% difference between treatment groups at 72 and 96 h respectively ($P = 0.015$), ($P = 0.033$).

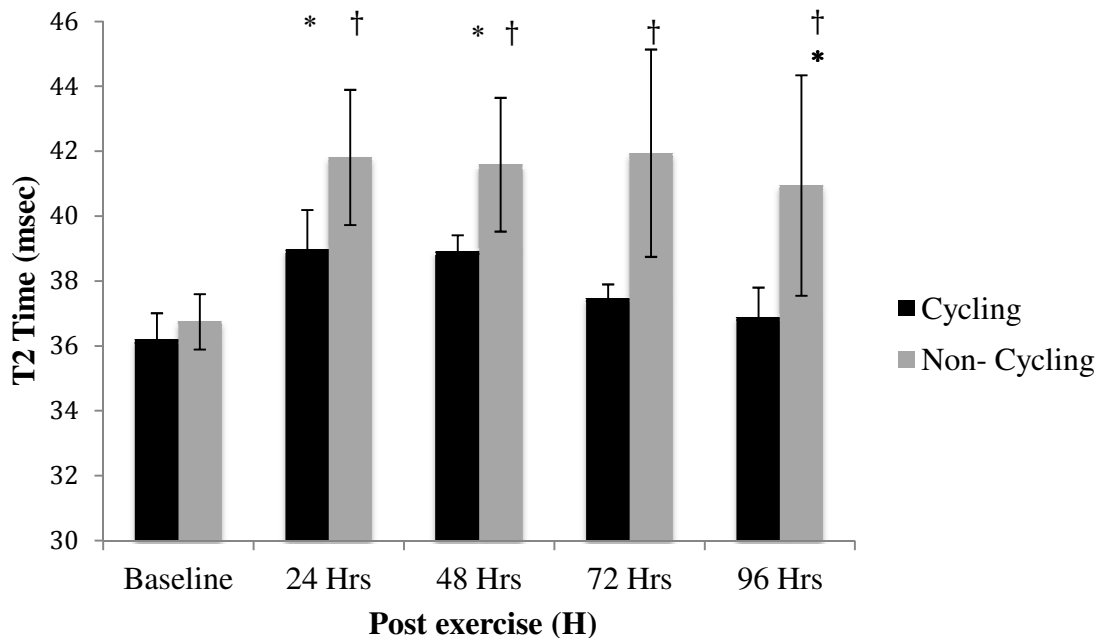


Figure 4.2: T2 times for the Vastus Intermedius between two intervention groups over time. Bars for each group represent SD for each group. † Denotes a significant difference between the two treatment groups. * Denotes a statistical significant

difference for both groups from baseline.

4.4 One repetition maximum

One repetition maximum (1RM) performance decreased significantly $\sim 41 \pm 13.9$ and $\sim 37 \pm 12.61$ kilograms for the CG and NCG respectively one-hour post damage. One repetition maximum performance stays significantly depressed in both groups for 96 h ($P < 0.05$). The greatest decrement in 1RM $\sim 49 \pm 20.5$ and $\sim 52 \pm 37.4$ kg's, were observed at the 48-hour mark for CG and NCG respectively. Figure 4.3 represents no statistically significant difference between the two treatment groups ($P > 0.05$).

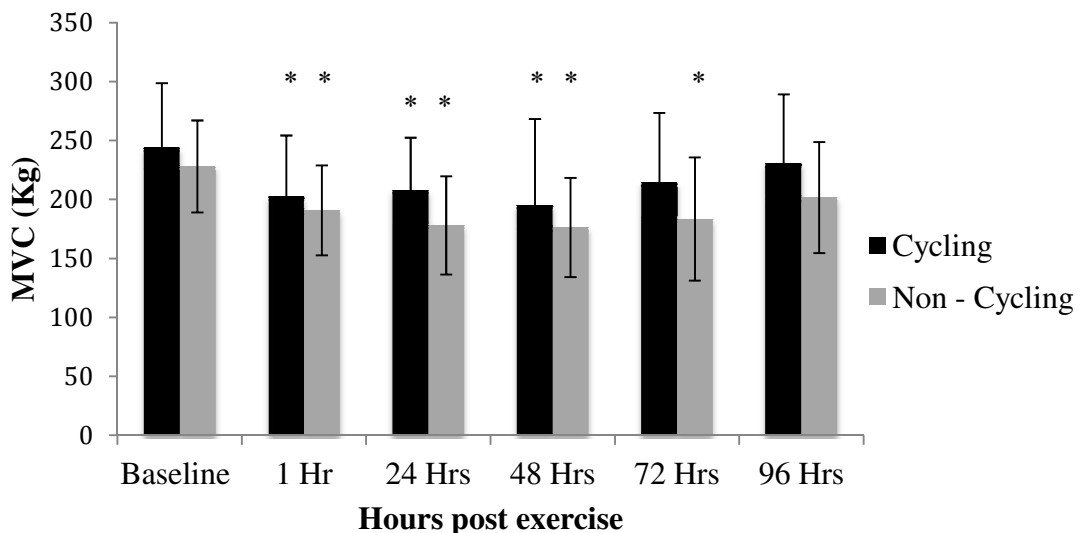


Figure 4.3: Changes in 1RM between groups over time. * Denotes a significant difference relative to base line. Bars represent SD for individual groups.

4.5 Swelling

Figure 4.4 represents changes in swelling for Vastus Lateralis (VL) (Fig 4.4A) and Vastus Intermedius (VI) (Fig 4.4B). Figure 4A shows an increase in CSA at 24, 48 and 72 h for the VL. There was no statistical difference between CG and NCG groups. CSA in both treatment groups remain elevated for 72 h with the highest peak of 1.68 and 1.86 cm squared in both the CG and NCG respectively at 48 h. Figure 4.4B, represents changes in CSA for the VI. CSA increased at time point 24, 48 and 72 h, there were no statistically significant differences between the groups. The greatest increase in CSA of 4.2 cm squared was observed at 48 in the CG. The NCG greatest increase of 2.82 cm's squared was reported at the 48-hr time point.

Figure 4.4 A.

Changes in swelling for the Vastus lateralis in relation to baseline.

*** Denotes a statistical difference relative to baseline. Bars represents SD.**

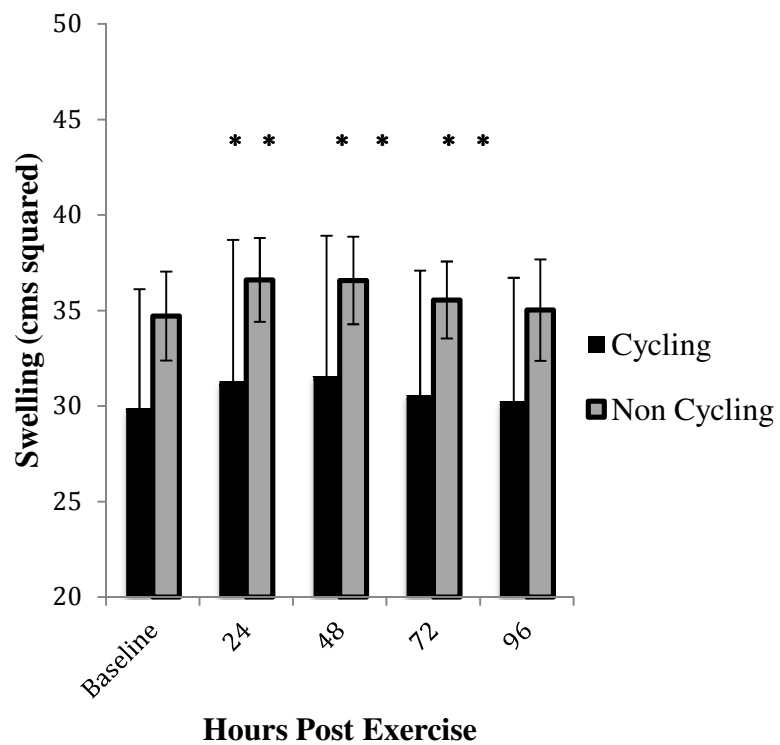
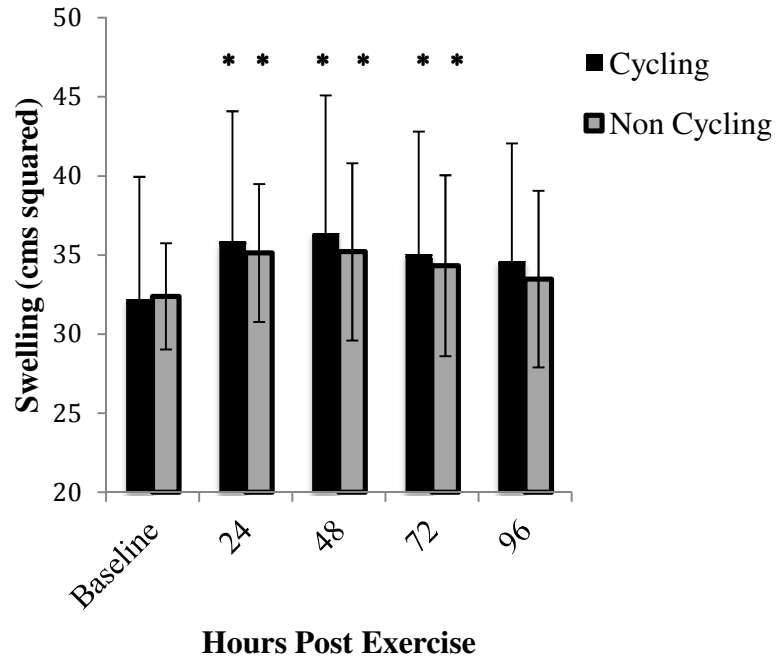


Figure 4.4 B.

Changes in swelling for the Vastus Intermedius in relation to baseline.

*** Denotes a statistical difference relative to baseline. Bars represent SD.**



4.6 Squat Jump

As shown in figure 4.5 Squat Jump (SJ) heights were reduced significantly for CG and NCG post damage ($P < 0.05$). Baseline values for CG and NCG were 42.1 ± 6.8 and 48 ± 6.6 cms. Depression in SJ performance occurred 1 h post damage and remained depressed for 72 hours. The greatest decrement in SJ height was at 48 post-damage for the CG and 72 h for the NCG $\sim 10.5 \pm 6.5$ and $\sim 16.6 \pm 8.2$ cm respectively. There was no statistical interaction between CG and NCG over time ($P > 0.05$).

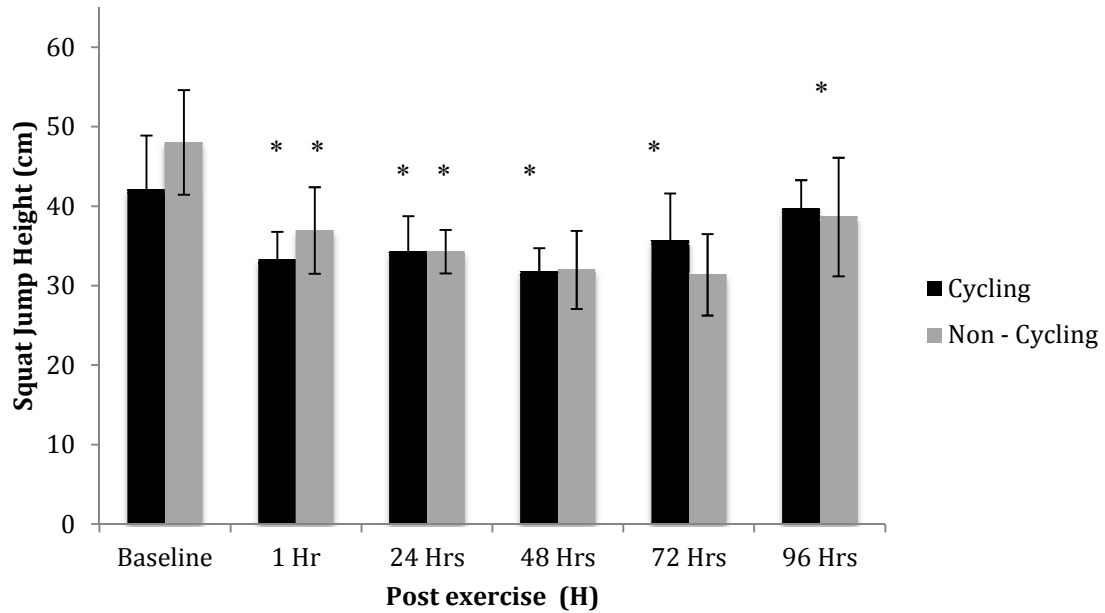


Figure 4.5: Changes in Squat jump height between groups over time. * Denotes a significant change in means for each group relative to baseline. Bars represent SD for individual groups.

4.7 Counter Movement Jump

Counter Movement Jump (CMJ) height also declined significantly in both the CG and NCG post damage ($P < 0.05$). CMJ height in both damage groups significantly decreased 1-h post exercise. The greatest decrement in CMJ occurred at 48 h for both groups $\sim 13.2 \pm 6$ and $\sim 11.5 \pm 5.7$ cm respectively with no between group differences. The non-exercised group showed no statistically significant reduction over the 96 h ($P > 0.05$). There was no statistically significant difference between the CG and NCG at any time point ($P > 0.05$).

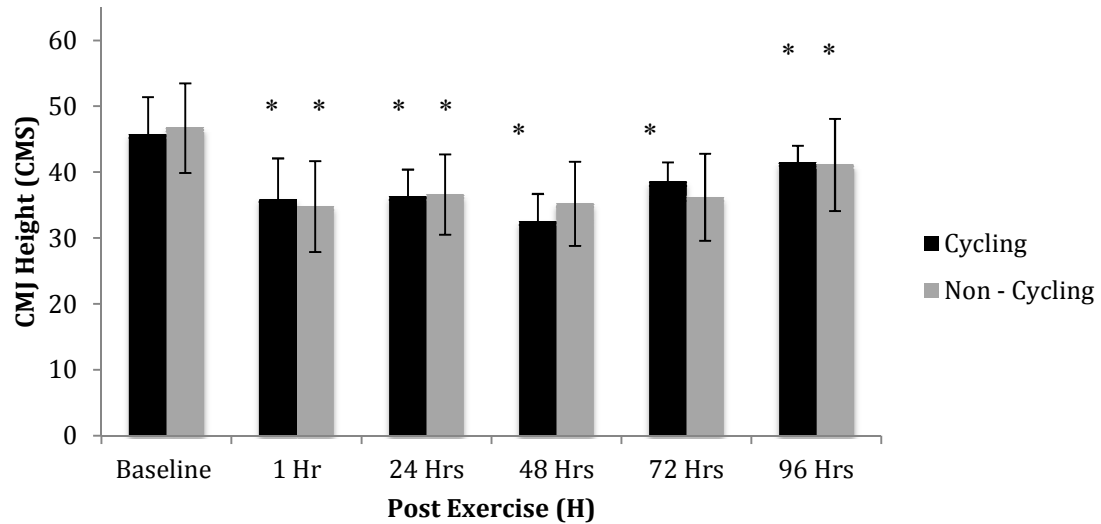


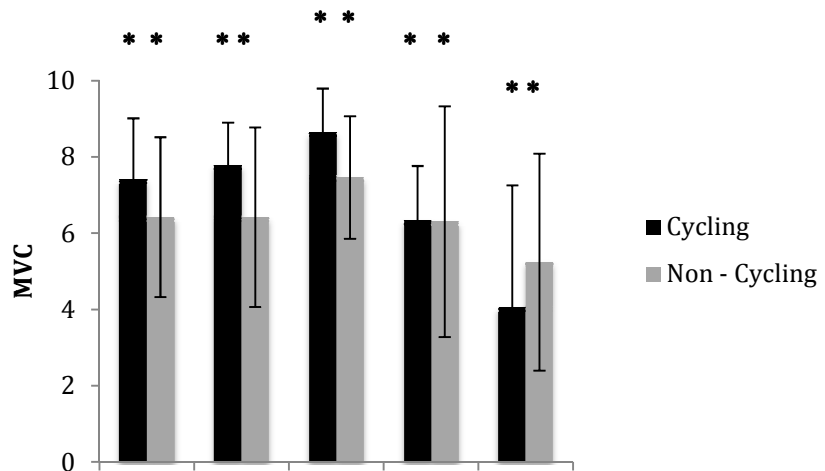
Figure 4. 6: Changes in CMJ height between groups over time. * Denotes a significant change in means for each group relative to baseline. Bars represent SD for individual groups.

4.8 Changes in Rate of Perceived Pain for Squat Jumps, Counter movement Jumps and MVC

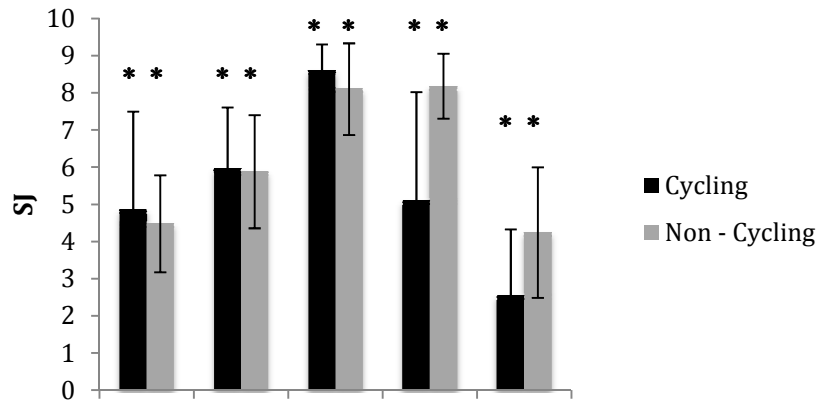
All rates of rate of perceived pain (RPP) values increased significantly from 1 h and peaked at the 48 h mark for all treatment groups on all performance measures post exercise. There was no statistical difference for RPP measures between CG and NCG on SJ, CMJ or 1RM ($P > 0.05$). Figure 4.7 shows a peak in RPP after SJ at 48 h with mean of NCG remaining peaked for 24 h between 48 and 72 hours. All RPP measurements across 3 different movement types were significantly elevated at 96 hours. Overall

changes at 96 h remain significantly elevated relative to baseline for all performance measures. (~ 3.4) ($P < 0.05$), CMJ (~ 3.5) ($P < .05$) and MVC (~ 4.65) ($P < 0.05$).

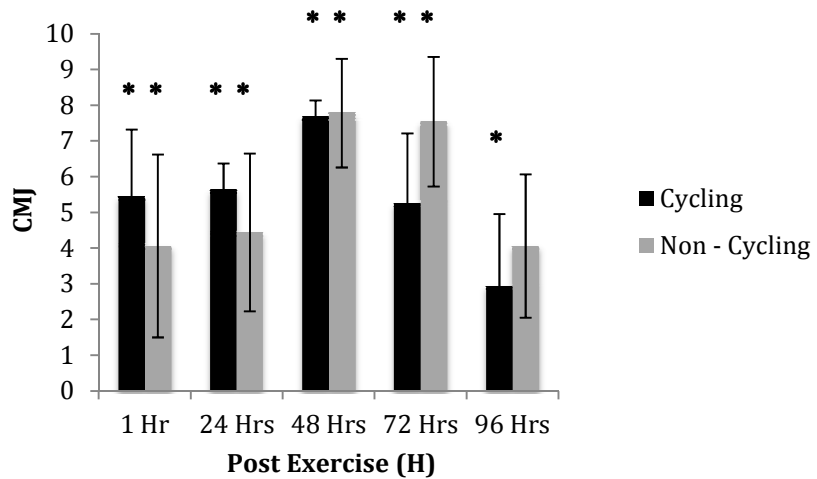
Figure 4.7A.
Changes in (VAS) for One repetition maximum (1RM). * Denotes a significant change in means for each group relative to baseline



B
Changes in VAS
for Squat Jumps
(SJ). * Denotes a
significant
change in means
for each group
relative to
baseline



C
Changes in VAS
for Counter
Movement
Jumps (CMJ). *
Denotes a
significant
change in means
for each group
relative to
baseline



4.9 Comparison on Counter movement jump and One repetition maximum

Figure 4.8, represents a comparison between 2 movements post damage. Both CMJ and MVC significantly decrease 1 h post exercise ($P < 0.05$). The mean performance for both exercises remains depressed for all 96 h with the greatest decrement in performance seen at 48 h. Mean CMJ performance (~73%) decreased post exercise more than MVC (~77%) but it was not significant ($P > 0.05$).

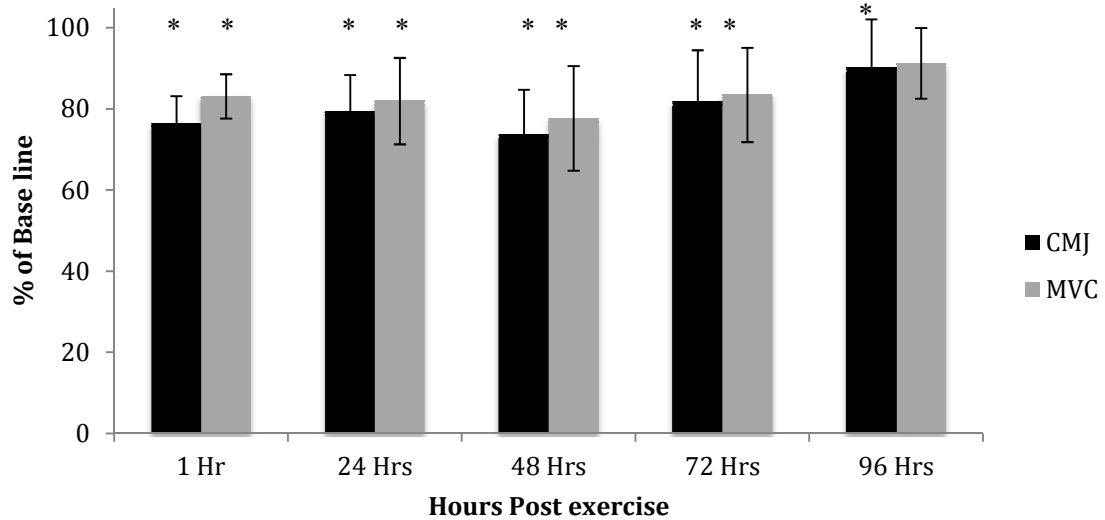


Figure 4.8: Percent change of means for all subjects of Counter Movement Jump and One repetition maximum over time. * Denotes a significant difference relative to baseline. Bars represent SD for individual groups.

4.10 Comparison of Squat Jump versus Counter Movement Jump

Figure 4.9, shows a significant reduction in SJ and CMJ performance for 96 h post exercise ($P < 0.05$). SJ performance decreases linearly from (75%) 1 h post exercise, to (74%) at 24 h and (71%) at 48 h before beginning to recover to (87%) at 96 h. CMJ performance initially reduces to 76% of baseline at 1 h post, increased slightly to 79% at 24 h before reducing again to 73% at 48 h. There is not a statistically significant difference between decreases in performance for the two exercises ($P < 0.05$).

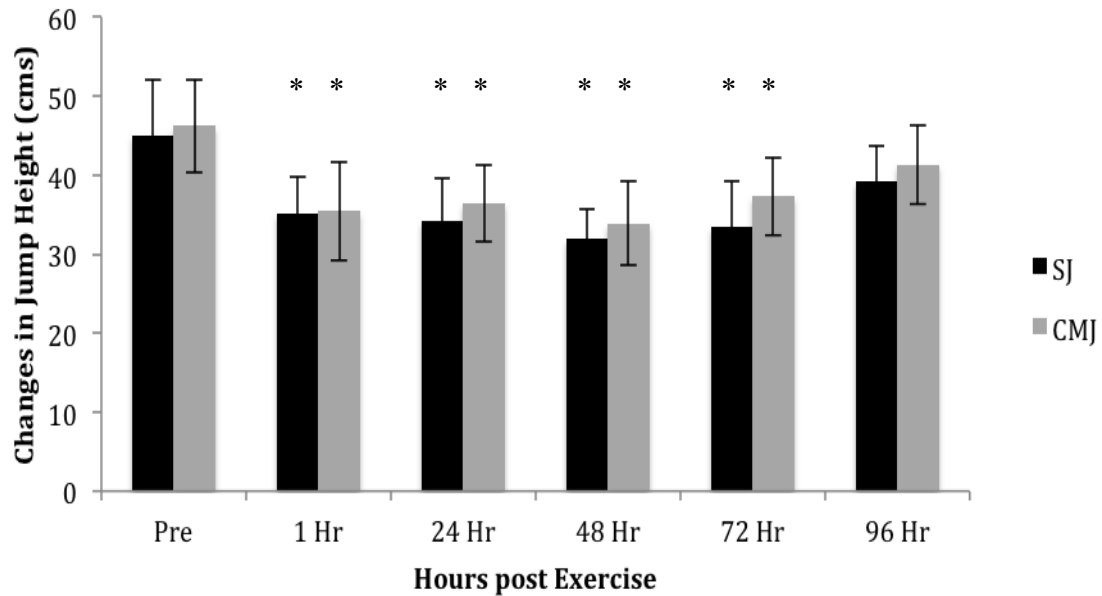


Figure 4.9: Changes in Squat Jumps and Counter Movement Jumps performance over time. * Denotes a difference relative to baseline. Bars represent SD for individual groups.

5. Discussion

The exercise protocol consisting of 10 sets x 10 repetitions of eccentric only contractions at 110% of 1RM resulted in an immediate reduction in all performance measures and increases in indirect markers of EIMD. One repetition maximum, SJ and CMJ heights were all reduced post ECC muscle damaging protocol. It was hypothesized that individuals who underwent active recovery therapy (ART; cycled at 60% of their individual gas exchange threshold, for 36 minutes at 24, 48, and 72 h time points) would

demonstrate faster changes in T2 times back to baseline for both T2 and CSA changes; and a more rapid recovery in performance of 1RM, SJ, CMJ and DOMS. Non-invasive determination of cellular response to ECC contraction; MR T2 relaxation times for both the VL and VI increased post exercise, with a significant between group interactions observed for VI over times point 24 – 96 h ($P < 0.05$). Changes approaching significance were reported at the 24-h time point for T2 time in the VL ($P = 0.064$). Cross sectional area for VL and VI increase significantly at the 24 h time point, and remained elevated for 48 h. However ART had no effect on rate of recovery or the magnitude of changes in swelling. Active recovery induced no significant between group interactions in 1RM, SJ, CMJ or DOMS at any time point. The main finding of this study was that ART had no significant effect on indirect markers of EIMD such as 1RM, jumps and DOMS or their pattern of recovery. This may of particular importance to sports teams that employ ART as a means of preparation for their next sporting event, further activity also did not exacerbate the level of damage or recovery time.

5.1 Evidence of muscle damage:

A significant difference was observed between T2 times for the CG and NCG at 24, 48, 72 and 96 h in the VI muscle. However the initial difference at 24h mark cannot be attributed to the effect of active recovery as the images were taken before the first bout of cycling. It is possible that the ART influenced changes in T2, however the exact mechanism cannot be explained by this investigation. The recovery time between the two

groups differed, with the CG T2 times returning to baseline at 72 h time point in the VI, whereas the NCG remained elevated for the entire 96 h. This may indicate an improved recovery process conferred by cycling. T2 times for all subjects increased significantly relative to baseline post EIMD. The baseline T2 measures taken in this study may not have reflected the T2 times of non-damaged muscle. Literature has reported that T2 times can remain elevated for 2-3 months post ECC contractions (Flexkenstein & Shellock 1992). The present study used participants who regularly engaged in resistance training of the lower body, meaning it is feasible that the baseline numbers may not be a reflection of undamaged tissue as they may be been still elevated from previous bouts of exercise. Resistance training individuals were chosen because they represent a large portion of the population and the reduction in muscle function is of particular importance to them if it has not dissipated before their next competitive event.

We hypothesized that using ART, as a means of increasing blood flow would help to minimize muscle damage and accelerate the recovery process; changes in T2 times were thought to reflect such changes. Results do support this. T2 times are thought to detect pockets of edema within the muscle in response to ECC exercise. Theoretically ART will have increased blood flow to the area and may have aided in the recovery process. Black & McCully, 2008 reported a 40% increase in T2 times after 80 contractions of the knee extensors; Foley et al, 1999 reported a 60% increase in T2 times of the elbow flexor muscles after maximal bicep curls. It is worth noting however that while testing the RBE they reported only a 24% increase in T2 times of the same muscles when the protocol was repeated (Black & McCully, 2008). Changes in T2 times may be directly related to the individuals training history, with muscles that are frequently exposed to ECC contraction as seen in resistance training eliciting smaller responses

despite demonstrating other indirect markers of damage. In a similar study to this one testing the effectiveness of recovery protocol on EIMD; Jayaraman et al, (2004) reported a 20% increase in T2 times accompanying a 40% reduction in MVC. These times remained elevated for the entirety of the study (15 days), and remained unaffected by heat, stretch and heat combine with stretch. Current evidence is not clear on the effect an ART has on the T2 times of damaged muscle.

Swelling or changes in CSA were evident in VL and VI 24 h after ECC contractions. The VI showed the greatest relative increase in CSA: in the CG at the 48 h mark a 4.2 cm² increase was observed equating to a ~ 13.5 % increase in muscle CSA. Current data does not support our hypothesis that; three bouts of cycling performed at 24 h intervals would mitigate some of the damage. Increased blood flow to the affected muscle induced by ART did not reduce the amount of swelling in the CG for VI or VL. Importantly; changes in blood flow to the affected tissue postulated to be induced by ART did not exacerbate the changes in CSA. It could be stated that increasing blood flow to a damaged area may increase the amount of edema through damaged capillaries. The 24 h period between damage and the first bout of cycling may have afforded enough time to ensure increased rate of blood flow did not cause further damage.

This is important, as it has been hypothesized that changes in swelling directly relate to sensation of DOMS as well as perceived ratings of effort during subsequent exercise (Armstrong, 1984). Because of the small number of studies involving such a direct measure of CSA and the large variability in ECC protocol used to induce damage; a pertinent comparison of changes in CSA is difficult. A 6% change in CSA of the VI has been reported after 300 ECC contraction (Takahashi et al., 1994). Comparatively a 13%

change constitutes a large increase in CSA induced by fewer contractions. Interestingly current results match those of Takahashi who found negligible difference in VL CSA when compared to VI post EIMD. The between muscle difference could be due to the respective involvement in the damage movements. If the VL has a greater involvement compared to VI in day-to-day use and/or training movements then it would be harder to damage than the VI. Conversely, if the dominant muscle involved in the ECC contraction were the VI not VL then that would account for the difference in CSA changes.

Delayed-Onset muscle soreness increased significantly post ECC contraction for all subjects. There was no between group interaction for RPP at any time point. Results for both CG and NCG show a rapid increase in rate of pain while completing the performance measure in the magnitude of 80% during 1RM and 40-60% during the jump protocol. This initial increase in pain sensation is not in line with previous reports of DOMS, which have been reported to start 12-24 h post exercise (Nosaka et al., 2002). A possible reason for this change is the large amount of musculature damaged by ECC contraction on a Hammer Leg Press. Participants may have experienced an increased rate of perceived exertion while executing the required performance measures. This increase in rate of perceived exertion may have been reported as a pain measure. Although it has been reported that damaged muscle can still be fully activated voluntarily participants may have been unwilling to fully exert force during the jump protocol as they experienced large amounts of pain upon landing. Results suggest that there is a greater sensation of pain during the 1RM protocol where compared with jumping. The starting position in the Leg Press may have caused a large amount of discomfort on the subsequent testing days. Because 1RM testing was performed prior to cycling in the CG,

no benefit of EIA was reported. The use of ART in alleviating pain sensation after damaging exercise seems to be temporary, with ART having no prolonged effect on the level of DOMS experienced by the CG when compared with NCG.

One repetition maximum is considered one of the most accurate indirect ways of measuring the extent of muscle damage post eccentric exercise (Warren et al., 1999). Changes in 1RM on average decreased to 82% and 83% of baseline values in the CG and NCG group respectively. This reduction in 1RM confirms the presences of damage induced by 100 ECC contractions. Active recovery therapy did not attenuate any of the negative effects of ECC contractions on 1RM performance. It has been postulated that neural factors and increased blood flow with ART will aid in the recovery of muscle function post damage (Sayer et al., 2000). It should be noted however that in that study Sayers et al, did not employ an ART on the same days as testing. A four-day period of ART was undertaken between ECC exercise and testing of rate of recovery. A 4 day period of ART without the presence of further testing may have yielded different results.

In accordance with 1RM data, results from SJ and CMJ for all participants decreased significantly one-h post ECC contraction. One-h post damage SJ performance was reduced ~ 25% for both CG and NCG; further reductions in SJ performance were evident over the next 48 hours. Over the 96 h period cycling did not have a significant effect on the magnitude of damage induced by ECC contraction nor on the rate or mode of recovery. The magnitude of change in SJ performance was greater than that reported by Easton et al., (2003); who reported a loss in SJ performance of between 15% post 100 maximal ECC contractions of the quadriceps muscles. However in this protocol the gluteal and calf muscles were exposed to ECC contractions as well as the quadriceps,

which may have induced damage in these muscles, the was reported by subject in regards to DOMS. The reduction in force output of these muscles may account for the greater reduction in SJ performance when compared with a protocol involving damage to the quadriceps alone. Counter-movement jump performance mirrored that of SJ after ECC contractions; 1 h post damage CMJ performance for participants in the CG, on average decreased 22% and 26% in the NCG. Further reductions were evident over the next 48 h with the greatest depression in performance for both groups at the 48-h mark. The second depression in jump performance may be due to participants unwilling to put in effort while experiencing DOMS. Exercise after EIMD has been shown to provide analgesic effect, however the study protocol dictated that jump height was tested before 1RM and cycling every day. This eliminated the possibility that EIA could affect jump performance. A single bout of cycling performed before the 48-h time point, this one session had no preventative effect on the magnitude of change. Two subsequent bouts of cycling had no effect on the rate of recovery with no difference in jump performance observed between the two groups.

5.2 Effect of Stretch – Shortening Cycling on EIMD

We observed equivocal decrements in force output from explosive movements in SJ or CMJ when compared with a slower 1RM exercise. We hypothesized that movements that utilize the SSC would not be as adversely affected by EIMD. However, there was no difference between a CMJ, SJ slow movement and 1RM tests. Counter-movement jumping utilizes the SSC to store energy and maximize the final CON phase; it

is thought that this stored elastic energy may help provide some protection from EIMD (Komi, 2000). Contrary to our hypothesis CMJ and SJ performance were equally as affected by 100 ECC contractions. Interestingly despite the SSC reported aid in athletic performance, there was no difference in baseline numbers between CMJ and SJ performance, and CMJ performance had not returned to baseline at 96. One reason for this could be that participants need to practice the jump protocol in order to fully utilize the elastic stored energy afforded by the pre-stretch of SSC jump. Alternatively the testing procedure was not stringent enough to ensure no SSC was used in the SJ test meaning both jumps used a similar model; we tried to control for this by removing attempts that visibly exhibited a SSC. No video or was taken but that may have helped to control for this variable.

Both 1RM and SJ tests did not use the SSC and were purely concentric in nature. Byrne and Easton (2002) theorized that ballistic movements would not be as affected by the symptoms of EIMD as isometric or slow velocity movements, we found equivocal decrements in performance in response to 100 ECC contractions. The recovery patterns observed may have been a product of similar testing procedure. Research has used isometric and isokinetic test of muscle function post ECC contraction (Power et al., 2012), whereas in the current study; the two tests used for 1RM and SJ did not control for velocity of contraction or range of motion. These two variables could have a large impact on the results of the aforementioned tests. One repetition maximum results from this study were equivocal to Byrne and Easton following at slow (50 degrees/sec) velocities after 100 ECC only maximal contractions (Byrne et al., 2001). Conversely the over 20% reduction in SJ and CMJ height do not match Byrne and Easton results from 2002; where following 100 squats at 70% of the participants body mass jump heights only reduced 10

– 15% at 24 h (Byrne & Eston, 2002a). This maybe due to above 110% of 1RM we used to induce damage compared with the 70% of bodyweight used in that experiment; however it should be noted the changes in 1RM were comparable to that found to Byrne and Eston. As far as we are aware this is the first study to investigate the effect of an active recovery on vertical jump performance following EIMD. Although in both SJ and CMJ the CG had an equivocal decreases in performance to the NCG over the first 48 hours; it seem to recovery faster. Although it was not statistically significant it maybe a product of lower subject numbers in both groups. The effect of EIMD on jump performance may represent a more sports specific movement we compared to an isometric or isokinetic test of muscle function.

5.3 Pattern of recovery

A bimodal model of recovery has been reported before in performance measures of SJ (Byrne & Easton 2002). It is characterized by an initial depression in muscle function immediately after exhaustive ECC muscle contraction, followed by a slight increase in performance (typically tested 24 h mark) and then further reduction at 48 h. Present results are in agreement with Byrne and Easton (2002), all performance measure followed the same bimodal recovery pattern in the 96 h after ECC contractions. Unlike Byrne et al., (2001) who reported a linear recovery of MVC back to baseline, our results followed a bimodal pattern of recovery. Further reductions in 1RM performance were observed between the 24 and 48 h time points. This could be due to the nature of 1RM testing. The current protocol used a leg press, which unlike a dynamometer does not control for changes in velocity or movement. Testing apparatus of this nature may be of

greater importance to strength and conditioning or sports coaches who do not have the funds, time or access to laboratory grade equipment. Hammer Leg Press was effective in assessing changes in 1RM and is a more cost effective way of measuring changes in 1RM post damage. The recovery models for both exercise groups differed slightly but were not statistically different at any time point. It was postulated that three bouts of cycling; being non-ECC in nature and only of a moderate intensity would aid in accelerating healing by increasing blood flow and not causing further damage. However current results indicate that in this population after maximal ECC contraction 3 bouts of moderate intensity cycling had limited effect on the rate of 1RM recovery and caution should be taken prescribing cycling as means of aiding in recovery of the indirect markers of muscle damage.

The second depression in jump performance at around the 48 h mark may have been due to a combination of DOMS and swelling. Participants in our study may have been reluctant to give maximal effort on the jumping attempts as they were in a great deal of pain, especially upon landing. By Peilson's (2012) definition of the severity of muscle damage our results sit on the border between a Mild and Moderate levels of damage according to the decline in MVC. Mild amounts of damage have been reported after several different exercise types such as 60 minutes of downhill walking (Nottle & Nosaka, 2005), a 42 km run (Petersen et al., 2007) and 30 minutes of downhill running at a -15% grade (Chen et al., 2008). This level of damage induced by non-eccentric only, single joint activity may allows us to assume that the changes in muscle function reported here maybe of the same magnitude as those experienced after a sporting competition.

5.4 Conclusion

In the current investigation it was hypothesized that individuals who cycled at 90% of their individual gas exchange threshold, for 30 minutes at 24, 48, and 72 h time points would demonstrate a faster recovery of 1RM, SJ, CMJ and DOMS. It appears that in a moderately trained population three bouts of moderate intensity cycling following 100 ECC only contractions has no effect on 1RM, SJ or CMJ height, Swelling or DOMS. MRI T2 changes did show some improvement in the CG and further investigation is needed to delineate the exact mechanism of change in T2. Our results do not support that of Sayers et al., (2003) who reported improved muscle function recovery with both immobilization and ART; these results suggest that more than one mechanism may be responsible for regulation of the rate of recovery. In some circumstances ART alone may not be enough to appreciably improve recovery of muscle function that has been damaged. It is important to note that in sporting contexts a performance increase of as small as one or two percent can have a large influence on contextual results. Importantly this research provides further evidence that while subsequent exercise may not aid in recovery it also doesn't prolong the recovery process. The presence of EIA induced by subsequent recovery activity maybe enough to warrant the utilization of ART. Delayed onset muscle soreness can be debilitating, and effect rate of perceived exertion for resistance and aerobic activity. This may be of particular importance to sports that use a periodized training calendar, as many sports require a simultaneous combination of resistance training and cardiorespiratory training.

5.5 Limitations

An insufficient the sample size may have resulted in limited statistical power to detect small, but significant changes in performance measures among the three groups.

Although other studies have found significant difference in muscle function using a similar sample size, the difference in protocol may explain the lack of results. Therefore, its possible that the sample size used of this study resulted in a type II error. In addition, the testing measures used may not have been as sensitive to change as other laboratory techniques. For example, 1RM was measured directly using leg press (increments of 5kgs), and not a standard dynamometer. The linear transducer has been shown to be an effective tool in testing maximal jump height however it does not provide sufficient information to determine if a participant did not use the SSC, this has the potential to effect the difference between SJ and CMJ. T2 relaxation times were used as an evaluation tool of cellular response to ECC contraction. Some research has suggested that T2 times can stay elevated for up to 3 months after training (Foley et al., 1999), this study used resistance trained individual; meaning their baseline measure may not have been accurate. Although ART effected T2 changes; this effect was not accompanied by changes in performance measures. Changes in T2 times may represent structural changes in response to ART but such changes have limited practical significance when presented on their own.

5.6 Future direction

Future investigation involving a much larger sample size with a more stringent training history is needed. This would allow us to compare results of individuals against others who exhibit the same magnitude of change post EIMD. Because there is a large inter-subject variability in response to a structured intervention the effectiveness of a recovery intervention on “high or low responders” would be of benefit. Furthermore,

future studies may choose to place more stringent parameters on the 1RM and jump testing. Investigation of the effects of SSC on indirect markers of muscle damage post EIMD shows some promise, however tests need to be used that ensure no SSC is utilized by the participant. All of these strategies would potentially reveal subtle benefits of a recovery intervention as well any affect using a SSC has on athletic performance post EIMD.

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Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. A.A. Vandervoort

Review Number: 16118

Review Level: Full Board

Review Date: May 19, 2009

Protocol Title: Short-Term Muscle Fatigue in Older Adults and The Potential Related Benefits on Increasing Force-Producing Capacity on Fatigue Resistance

Department and Institution: Physical Therapy, University of Western Ontario

Sponsor: NSERC-NATURAL SCIENCES ENGINEERING RESEARCH COUNCIL

Ethics Approval Date: July 29, 2009

Expiry Date: September 30, 2013

Documents Reviewed and Approved: UWO Protocol and Letter of Information and Consent Form dated June 22, 2009, and Poster

Documents Received for Information:

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request Form.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the HSREB except when necessary to eliminate immediate hazards to the subject or when the change(s) involve only logistical or administrative aspects of the study (e.g. change of monitor, telephone number). Expedited review of minor change(s) in ongoing studies will be considered. Subjects must receive a copy of the signed information/consent documentation.

Investigators must promptly also report to the HSREB:

- a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
- b) all adverse and unexpected experiences or events that are both serious and unexpected;
- c) new information that may adversely affect the safety of the subjects or the conduct of the study.

If these changes/adverse events require a change to the information/consent documentation, and/or recruitment advertisement, the newly revised information/consent documentation, and/or advertisement, must be submitted to this office for approval.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

Chair of HSREB: Dr. Joseph Gilbert

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Use of Human Subjects - Ethics Approval Notice

Principal Investigator: Dr. G. Marsh

Review Number: 17762

Review Level: Full Board

Review Date: January 25, 2011

Approved Local # of Participants: 90

Protocol Title: Comparison of bone geometry and muscle volume in the legs of young, old and very old females versus males using magnetic resonance imaging (MRI).

Department and Institution: Kinesiology, University of Western Ontario

Sponsor:

Ethics Approval Date: March 09, 2011

Expiry Date: August 31, 2015

Documents Reviewed and Approved: UWO protocol (including instruments noted in Section B.1), Letter of Information and Consent Form and Advertisement

Documents Received for Information:

This is to notify you that The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/CH Good Clinical Practice Practices: Consolidated Guidelines and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this REB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request Form.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the HSREB except when necessary to eliminate immediate hazards to the subject or when the change(s) involve only logistical or administrative aspects of the study (e.g. change of monitor, telephone number). Expedited review of minor change(s) in ongoing studies will be considered. Subjects must receive a copy of the signed information/consent documentation.

Investigators must promptly also report to the HSREB:

- a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
- b) all adverse and unexpected experiences or events that are both serious and unexpected;
- c) new information that may adversely affect the safety of the subjects or the conduct of the study.

If these changes/adverse events require a change to the information/consent documentation, and/or recruitment advertisement, the newly revised information/consent documentation, and/or advertisement, must be submitted to this office for approval.

Members of the HSREB who are named as investigators in research studies, or declare a conflict of interest, do not participate in discussion related to, nor vote on, such studies when they are presented to the HSREB.

Chair of HSREB: Dr. Joseph Giben
 FDA Ref #: IRB 00380640

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APPENDIX C
MRI SCREENING QUESTIONNAIRE



NAME: _____
 AGE: _____
 HEIGHT: _____
 WEIGHT: _____

- | | | |
|---|-----|----|
| 1. Have you had a previous MRI?..... | YES | NO |
| 2. Have you ever had a metallic object in your eye? | YES | NO |
| 3. Is there any chance you might be pregnant? | YES | NO |
| 4. Do you have any of the following? | | |
| • HEART | YES | NO |
| PACEMAKER/WIRES/STENT/DEFIBRILLATOR..... | YES | NO |
| • BRAIN ANEURYSM CLIPS. | YES | NO |
| | YES | NO |
| • SHUNT/SURGICAL CLIPS. | YES | NO |
| | YES | NO |
| • SHRAPNEL/BULLETS. | | |
| | YES | NO |
| • DENTURES. | | |
| | YES | NO |
| • INTRA-UTERINE DEVICE | YES | NO |
| (IUD)..... | YES | NO |
| • OTHER IMPLANTED DEVICES (HEART VALVES. | | |
| • EAR IMPLANTS. PROSTHESES, EYE | | |
| SPRINGS)..... | | |
| • MEDICATION PATCHES (NICODERM, HABITROL, | | |
| • TRANSDERM-NITRO. ETC). | | |
| | | |
| • BODY PIERCING. | | |
| | | |
| • PERMANENT TATTOO, EYELINER. | | |
| | | |
| 5. Please list surgeries on the following: | YES | NO |
| • Head _____ | | |
| • Neck _____ | | |
| • Spine _____ | | |
| • Chest _____ | | |
| • Abdomen _____ | | |
| • Upper/Lower _____ | | |
| • Other _____ | | |
| 6. Are you claustrophobic?..... | | |

PLEASE REMOVE ANY JEWELLERY, METAL ON CLOTHING AND FROM POCKETS

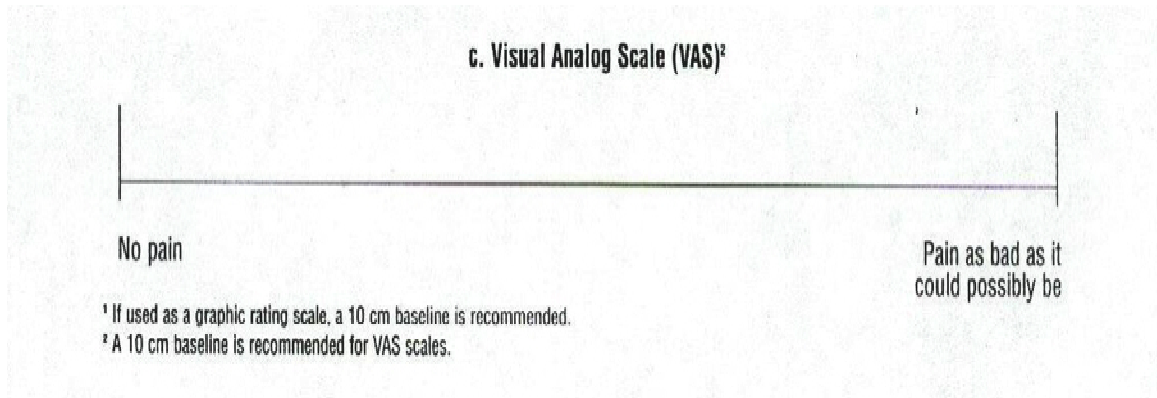
Participant's Signature: _____

Date: _____

MR operator's Signature: _____

THIS FORM MUST BE COMPLETED BEFORE YOU CAN UNDERGO YOUR MRI SCAN

APPENDIX D
VISUAL ANALOG SCALE



CIRRICULUM VITAE

Name: Brendan P. Major

Post-secondary Education and Degrees: Masters of Science (Supervisor: Dr. Greg Marsh)
Western University
London, Ontario, Canada
2011 - 2013

Bachelor of Exercise Science
Victoria University
Footscray, Victoria, Australia
2008 – 2010

Related Work Experience: Teaching Assistant
A) Introduction to research design
B) Physical Fitness Appraisal
Western University
2011 - 2013

Graduate Assistant Strength and Conditioning Coach
Western University Athletics
2010 - 2013