A comparison of flexibility training and the repeated bout effect as priming interventions prior to eccentric training of the knee flexors.

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Abstract

Performance of a series of eccentric contractions produces adverse effects including muscle weakness, delayed onset muscle soreness (DOMS), fluid accumulation and decreased muscle function. The repeated bout effect is a physiological adaptation observed when a single-bout of eccentric exercise protects against muscle damage from subsequent eccentric bouts. Similar to the repeated bout effect, increases in flexibility have been linked to attenuations in acute muscle damage, muscle fatigue and strength loss after eccentric exercise. **Purpose**: The purpose of this study was to examine the muscle physiological responses to eccentric strength training after first priming the muscles with either a period of static flexibility training or a single intense bout of eccentric exercise performed weeks earlier; and compare these to the responses from eccentric strength training when no prior intervention is administered. **Methods**: Twenty-five participants were randomly assigned to a flexibility (F) (n=8), a single-bout (SB) (n=9), or a control (C) (n=8) group. The design consisted of two 4-week phases; 1) priming intervention, 2) eccentric training. The priming intervention included static stretching (3x/week; 30mins/day) (F), a single-bout of eccentric exercise (SB) or no priming intervention (C). All groups proceeded to complete eccentric training of the knee flexors using isotonic contractions (%load progressively increased over training period) on a dynamometer following the priming intervention phase. Testing was completed at baseline, post-priming intervention and post-eccentric training, in conjunction with data being collected during the acute eccentric training phase (0hr, 24hr, 48hr; post-bout 1 and 4). Dependent measures included muscle thickness, isometric maximal voluntary contraction (MVC), eccentric and concentric MVC, optimal angle, active range of motion (ROM), passive ROM, maximal power, electromyography (EMG) and delayed onset muscle soreness (DOMS).

Results: Acute data during the eccentric training phase revealed a significant reduction in DOMS for both the F and SB groups compared to the C following the first bout of eccentric exercise ($p<0.05$). The F also had reduced soreness in comparison to both the SB and C post fourth bout of eccentric exercise ($p<0.05$). The F group demonstrated attenuated loss in isometric strength (post fourth bout) and maximal power (post first bout) during eccentric training compared to the C group ($p<0.05$). However, there was no significant difference between groups across all dependent variables following the eccentric training phase.

Conclusion: This is the first study to directly compare the protective effects observed with static flexibility training to that of a single-bout of eccentric exercise throughout a subsequent eccentric training regime. Although differences in muscle soreness, strength and maximal power occurred during the acute stages of eccentric training, there appeared to be no significant advantage of either protective priming method at the end of eccentric training.

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Dedication

I dedicate the completion of my Master's thesis to my best friend and wife Samantha. Without her incredible patience and support during the pursuit of my academic goals, I would have never seen my graduate degree to completion. I am forever grateful for your love.

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Glossary of Terms

Active Range of Motion (Active ROM): The range of motion achieved by ones ability to assume extended joint positions and then maintain them using only the tension of the agonists and synergists while the antagonists are being stretched.

Concentric: Muscle action in which tension causes visible shortening in the length of the muscle; positive work is performed.

Eccentric: Muscle action in which tension is developed in the muscle and the muscle lengthens due to the force applied exceeds the momentary force the muscle can dynamically generate; negative work is performed.

Flexibility: The range of motion available to a joint or a series of joints to move freely without restriction.

Force-Velocity Relationship: The relationship between the tension development in the muscle and velocity of shortening or lengthening.

Hypertrophy: An enlargement or growth of tissue caused by an increase in the size of the muscle cells.

Isokinetic Exercise: An exercise in which concentric muscle action is generated to move a limb against a device that is speed controlled. Individuals attempt to develop maximum tension through the designated range of motion at the specified speed of movement.

Isometric: Muscle action in which tension develops but no visible or external change is seen in joint position; no external work is produced.

Isotonic Exercise: An exercise in which an eccentric or concentric muscle action (or both) is generated to move a specified weight through a range of motion with the velocity free to vary. Also known as velocity dependent contractions.

Length-Tension Relationship: The relationship between the length of the muscle and the tension produced by the muscle; highest tensions are developed slightly past resting length.

Optimal Angle: The angle at which peak torque is achieved during a muscle contraction.

Parallel Elastic Component: The passive component in a muscle model that behaviorally develops tension with elongation.

Passive Range of Motion (Passive ROM): The range of motion achieved by ones ability to assume extended joint positions and then maintain them through external manipulation, such as gravity or manual manipulation.

Power: The product of force and velocity.

Range of Motion: The measurement of movement around a joint.

Repeated Bout Effect: Refers to the protective effect of a single bout of eccentric exercise on a subsequent bout of eccentric exercise. Typically, the subsequent bout of eccentric exercise is accompanied by reduced muscle soreness, quicker recovery of strength, and decreased markers of muscle damage.

Sarcomerogenesis: When sarcomeres are added longitudinally to a muscle fiber without changing the overall fiber length. These additional sarcomeres will create a shift in the muscle's length-tension curve towards a longer length.

Visual Analogue Scale (VAS): A psychometric response scale used as a measurement instrument for subjective characteristics (i.e. muscle soreness).

Chapter 1: Scientific Outline

1.1 Introduction

Utilizing eccentric exercise as a strength training method is often used in clinical and performance settings as research has shown that it increases muscle strength, flexibility and cross-sectional area (Farthing & Chilibeck, 2003; Nelson & Bandy, 2004). However, repeated eccentric contractions can lead to exercise-induced muscle damage (EIMD) and it is well documented to produce adverse effects including muscle weakness, delayed onset muscle soreness (DOMS), fluid accumulation and decreased muscle function (Chapman et al., 2006; Krentz & Farthing, 2010; Peake et al., 2005). It has also been proposed that the microscopic damage suffered by the muscle fibers following a period of unfamiliar eccentric exercise can potentially lead to a more severe strain injury (Brockett et al., 2001; Proske et al., 2004). Adverse symptoms of eccentric exercise typically subside following a week of recovery, however, methods aimed at reducing the extent of muscle damage and preventing prolonged DOMS are extremely important for athletes maximizing their performance during subsequent training sessions, potentially reducing risk of significant muscle strain injury and for the general population to remain physical active on a daily basis (Cheung et al., 2003; Brockett et al., 2001; Proske et al., 2004).

One method of protecting against muscle damage commonly observed following multiple bouts of eccentric exercise is known as the “repeated bout effect” (Nosaka & Clarkson, 1995). The repeated bout effect refers to the protective effect of a single bout of eccentric exercise on a subsequent bout of eccentric exercise and has been shown to last up to several weeks and possibly up to 6 months (Nosaka et al., 2001). Typically, the subsequent bout of

eccentric exercise is accompanied by reduced muscle soreness, quicker recovery of strength, and decreased markers of muscle damage (Chapman et al., 2006). Eccentric exercise has been implemented into the training programs of Australian Rules football (AFL) players, and the incidence of hamstring strains appears to have been reduced from previous seasons (Proske et al., 2004).

Similar to the repeated bout effect, recent evidence has indicated that increased flexibility has been linked to attenuation of acute eccentric EIMD, muscle fatigue and strength loss (Chen et al., 2011). This apparent protective effect of enhanced flexibility might be related to a reduction in muscle stiffness (McHugh et al., 1999b). This finding might suggest that prolonged flexibility training and eccentric training may share similar mechanisms for prevention against subsequent muscle damage.

Could one of these protective training modalities become the preferred solution for preventing the muscle damage incurred by eccentric exercise and potentially help maximize subsequent training periods for athletes and the general population alike? Is one method of preventing eccentric EIMD more beneficial than the other? Answering these questions could help uncover better strategies for preventing eccentric EIMD that could become a critical component to training and rehabilitative programs used by health and exercise professionals.

1.2 Literature Review

1.2.1 Eccentric Exercise and Muscle Damage

Muscles generate tension when they contract against resistance in order to maintain joint positions, raise a segment or object, or lower or control a segment. An isometric contraction occurs when a muscle actively contracts and develops tension with no visible or external change in joint position (Komi, 1984). If a muscle visibly shortens while generating tension actively, the

muscle action is termed concentric (Komi, 1984). In concentric joint action, the net muscle forces producing movement are in the same direction as the change in joint angle, meaning that the agonists are the controlling muscles. Eccentric contractions are defined as muscle activities that occur when the force applied to the muscle exceeds the momentary force the muscle can dynamically generate (Lindstedt et al., 2002). This results in the muscle being forcefully lengthened due to the high external load. An eccentric muscle contraction can be completed with a greater load than can be lifted using a concentric contraction (Kraemer et al., 2006; Hollander et al., 2007). Fewer motor units are recruited during eccentric contractions when compared to equally loaded concentric contractions (Moritani et al., 1987). Consequently, eccentric contractions are more metabolically efficient and can produce the same force output with less oxygen consumption, sympathetic activation and blood lactate levels when compared to concentric contractions (Carrasco et al., 1999). Muscles use eccentric contractions primarily to help decelerate, brake or absorb energy (Vogt & Hoppeler, 2014).

Eccentric exercise is often used as a strength training modality in which repeated eccentric contractions are done consecutively. This type of exercise offers a unique and potentially beneficial form of exercise for maintaining and improving health (Isner-Horobeti et al., 2013) and has been shown to increase muscle strength, flexibility and cross-sectional area (Farthing & Chilibeck, 2003; Nelson & Bandy, 2004). However, eccentric exercise also unfavourably results in muscle fiber damage to the muscle and is commonly referred to as EIMD (Hyldahl & Hubal, 2014). Eccentric exercise results in greater muscle damage than either concentric or isometric contractions; theorized to be associated with higher load per fiber experienced throughout the lengthening muscle movement (Clarkson & Hubal, 2002).

While performing a series of eccentric contractions, the sarcomeres progressively become disrupted as a result of being overstretched. The disruptions spread to other sarcomeres within a myofibril, and across to other myofibrils, resulting in damage to the membrane; including the sarcoplasmic reticulum, transverse tubules or the sarcolemma (Proske & Allen, 2005). Telling signs that EIMD has occurred following eccentric exercise are the presence of disrupted sarcomeres in myofibrils and interference with the excitation-contraction (E-C) coupling system (Proske & Morgan, 2001). Accompanying this disruption is the opening of cation channels followed by an uncontrolled release of Ca^{2+} into the sarcoplasm. This increase of sarcoplasmic Ca^{2+} triggers proteolysis, which is associated with fiber breakdown and repair (Proske & Allen, 2005). Increased muscle stiffness following eccentric EIMD has been shown to cause alterations in EMG muscle activity (DeVries, 1966; McGlynn et al., 1979). These alterations in EMG activity are likely caused by the changes to the sensitivity of the muscle spindles of the damaged muscle and can lead to interference in sensory inputs (Ia and II afferent fibers), which in turn increases the number of motor-units recruited at rest (Torres et al., 2013) and increases muscle stiffness. A number of acute adverse effects and consequences have been observed following eccentric muscle actions that can include loss in muscle force production, DOMS, fluid accumulation and disturbances in proprioception (Chapman et al., 2006; Krentz & Farthing, 2010; Peake et al., 2005).

The decline in muscle force production following bouts of eccentric exercise is one of the most commonly observed indirect markers of EIMD. A significant reduction in force has been observed following eccentric exercise ranging from 15-70% of pre-exercise values and the loss can persist up to 2 weeks (Clarkson & Hubal, 2002). The extent of the force loss and the time for full recovery post-eccentric bout is dependent upon the intensity, mode and novelty of the

exercise (Newhan et al., 1987; Eston et al., 2000). These losses have been shown to be greater in muscles of the upper body than the lower body (Hyldahl & Hubal, 2014).

Muscle soreness has been extensively studied and observed following eccentric exercise (Clarkson & Hubal, 2002). Muscle soreness usually peaks at 24hr and 48hr post-eccentric exercise and will usually resolve within 5-7 days (Clarkson et al., 1992; Ebbeling & Clarkson, 1989); however, the adverse effects have been ameliorated through a protective response observed during subsequent bouts of eccentric exercise following the original exercise stimulus (Nosaka & Clarkson, 1995). This protective adaptation observed with eccentric exercise is commonly referred to as the repeated bout effect.

1.2.2 The Repeated Bout Effect

The repeated bout effect refers to the protective effect of a single bout of eccentric exercise on a subsequent bout of eccentric exercise when performed within 6 months of the original bout even when no additional exercise is performed between bouts (Clarkson et al., 1992; McHugh et al, 1999a). Typically, the subsequent bout of eccentric exercise is accompanied by reductions in muscle soreness, strength loss, and circulating muscle proteins associated with muscle damage, such as blood creatine kinase (CK) which is virtually unchanged following the second bout (Chapman et al., 2006, Clarkson & Tremblay, 1988).

Researchers are uncertain as to the mechanisms that underlie the phenomenon of the repeated bout effect. Some theories point towards neural mechanisms being responsible for the reduction in EIMD and related symptoms (Nosaka & Clarkson, 1995). Early theories suggest that uneven recruitment of motor neurons during a bout of eccentric exercise could lead to the rupture of muscle fibers and subsequent eccentric exercise training might reduce the damage incurred (Hough, 1902). Differences in motor unit activation have been observed between

repeated bouts of eccentric exercise as evident by increases in electromyography (EMG) amplitude (McHugh et al., 2001). An increase in EMG amplitude and torque following a repeated bout of eccentric exercise would indicate that the contractile workload was distributed across a greater number of muscle fibers (Hortobagyi et al., 1996). Additionally, an increase in motor unit synchronization is evident as indicated by a decrease in firing frequency of the content EMG signal (Warren et al., 2000). Median frequency decreases following a repeated bout of eccentric exercise in the tibialis anterior, which could indicate an increase in slow-twitch motor unit recruitment or an increase in motor unit synchronization (Warren et al., 2000). Dartnall et al. (2011) found an increase in motor unit synchronization following a single bout of eccentric exercise. This evidence of neurological adaptation was credited for reducing the amount of muscle damage following a second bout of eccentric exercise. The repeated bout effect has also been shown to have specificity effects as evident during contractions performed at the same velocity, demonstrating further support for the neural adaptation theory (Barss et al., 2014).

Although neural changes such as synchronicity of motor unit firing (Hortobagyi et al., 1998) and altered recruitment patterns (Golden & Dudley, 1992) have been observed, the primary argument refuting a neural mechanism mediating the repeated bout effect is that a reduction in damage markers of EIMD, such as CK, during a second bout of eccentric exercise was still observed when exercise was solely invoked by electrical muscle stimulation (Aldayel et al., 2010; Black & McCully, 2008). The use of electric muscle stimulation would trigger a targeted muscle contraction during exercise that would bypass neural control and thus refutes the neural recruitment mechanism theories previously suggested. Therefore the central nervous

system probably plays a minimal role in the adaptations observed and peripheral adaptations play a more prominent role with regards to the repeated bout effect (Nosaka et al., 2002).

An alternative theory as to the mechanism of the repeated bout effect points toward mechanical adaptations protecting the myofibrils against disruption and subsequent damage (McHugh et al., 2003). An increase in both dynamic and passive muscle stiffness has been observed with eccentric training (Pousson et al., 1990; Reich et al., 2000). The effects in muscle stiffness have been attributed to both an increase in the stiffness of intramuscular connective tissue and a change in the cytoskeletal proteins responsible for maintaining sarcomere alignment and structure. An increase in intramuscular connective tissue following a damaging bout of eccentric exercise is suggested to protect against damage from repeated eccentric bouts due to the increased connective tissue's ability to dissipate stress across the myofibrils (Lapier et al., 1995). Disruption of the cytoskeletal protein desmin is one of the primary events of eccentric EIMD. An animal study showed an increase in desmin and subsequently, the remodeling of the cytoskeleton 3-7 days following eccentric contractions (Barash et al., 2002). This mechanical reinforcement of the cytoskeleton was suggested to protect against excessive sarcomere strain in subsequent bouts. The theory of cytoskeletal reinforcement through an increase in desmin has been refuted in one study (Sam et al., 2000). Mouse muscles that lacked desmin actually demonstrated less cytoskeletal disruption. It was theorized that the muscle lacking desmin was less susceptible to damage during an eccentric contraction due to less dynamic muscle stiffness.

Contradictory literature suggests that more passive muscle stiffness actually increases the likelihood of muscle damage occurring with eccentric exercise (McHugh et al., 1999b). The passive stiffness of a skeletal muscle has been measured in humans according to the relationship between joint torque and range of motion during passive stretch (Gadjdosik, 1991). McHugh et

al. (1999b) showed that subjects with greater passive muscle stiffness experienced more pain and tenderness, as well as greater losses in strength and increases in CK activity than subjects who had more compliant hamstrings. McHugh et al. (1999b) theorized that the reason for the increased muscle damage in subjects with stiffer muscles might be explained by tendon-aponeurosis mechanics during eccentric contractions. McHugh et al. (1999b) proposed that as the stiff muscles are actively lengthened, strain is transferred from a rigid tendon-aponeurosis complex to the coinciding muscle fibers, resulting in myofibrillar strain. On the contrary, in compliant muscles, the tendon-aponeurosis complex is able to absorb lengthening, which in turn limits the amount of myofibrillar strain. This theory suggests that muscle stiffness primarily reflects the extensibility of the connective tissue elements in parallel with the muscle fibers (parallel elastic component) (Lieber & Fridén, 1993). The more compliant tendon-aponeurosis complex would therefore allow the muscle fibers to shorten or remain relatively constant in length despite the muscle-tendon unit lengthening during an eccentric contraction (Roberts et al., 1995).

Another proposed mechanism that could be primarily responsible for the repeated bout effect is a physical restructuring of muscle following an initial bout of eccentric exercise (McHugh, 2003). It has been suggested that stress-susceptible muscle fibers may incur permanent damage that could cause these fibers to be damaged selectively beyond repair by an initial exercise bout. Removal of these fibers and replacement with stronger fibers during the repair process would strengthen the structure for future challenges (Armstrong, 1990; Armstrong et al., 1991).

Further structural changes observed during the adaptation process following the first bout of eccentric exercise involve the integration of additional sarcomeres in series to existing muscle

fibers (Morgan & Allan, 1999). One of the more attractive theories to explain the repeated bout effect is that extra sarcomeres are added longitudinally to a muscle fiber without changing the overall fiber length and this process is referred to as sarcomerogenesis. This adaptation results in individually shorter sarcomeres in relation to a corresponding muscle fiber length (Proske & Morgan, 2001). These additional sarcomeres will create a shift in the muscle's length-tension curve towards a longer length (Brockett et al., 2001). Therefore, any subsequent stretching across a given section of the contracting muscle will result in a shorter, initial sarcomere length. This reduces strain to individual sarcomeres during muscle lengthening due to their greater number in series. Consequently it is less likely for individual sarcomeres to be stretched to the point of disruption and EIMD following the repeated eccentric bout would be significantly reduced.

Regardless of the mechanism for explaining the repeated bout effect, the protective adaptations incurred following a single bout of eccentric exercise are of particular importance in the realm of preventing muscle damage; however, the repeated bout effect may not be the only method in protecting a muscle against subsequent damage during eccentric exercise. As discussed in the next section, comparable results have been uncovered with chronic flexibility training and stretching.

1.2.3 Chronic Static Stretching

Chronic static stretching and flexibility training (over several weeks) have been linked with many performance benefits and no current studies report that it impedes or diminishes performance (Shrier, 2004; Kokkonen et al., 2007). Previous research indicates that the primary benefit of regular stretching is an increase in strength; with reported increases in hamstring isokinetic torque, trunk strength and bench press performance (Worrell et al., 1994; Handell et al., 1997; Godges et al., 1989; Wilson et al., 1994). Other performance benefits include

decreased sprint time (Dintman, 1964), increased vertical jump power (Hunter & Marshall, 2002) and increased velocity of concentric contractions (Hortobagyi et al., 1985). Chronic stretching has also been shown to increase concentric and eccentric maximum voluntary contraction (MVC) (Shrier, 2004). Markers of hypertrophy and muscle growth such as increased muscle protein synthesis have been documented following progressive stretch overloading (Antonio & Gonvea, 1993).

Similar to the repeated bout effect, recent evidence indicates that increased flexibility is effective for reducing the negative symptoms associated with eccentric EIMD (Chen et al., 2011). Chen et al. (2011) investigated the effects of chronic static stretching or proprioceptive neuromuscular facilitation (PNF) on an acute maximal eccentric exercise bout. Following 8 weeks of flexibility training, the static stretching and PNF groups had smaller changes in muscle soreness, functional decline, plasma CK activity and strength loss following acute maximal eccentric exercise. The researchers concluded that the protective effect created by flexibility training was likely associated with the longitudinal increase in the number of sarcomeres in series and/or changes in musculo-tendon compliance. These ideas coincide with McHugh et al. (1999b) who suggested that the apparent protective effect of enhanced flexibility might be connected to a reduction in muscle stiffness. They reported that subjects whose hamstrings were more compliant (i.e. less stiff) had significantly less symptoms of muscle damage after maximal eccentric exercise than those whose hamstrings were stiff.

There are at least two other studies that have investigated the effect of flexibility training on eccentric EIMD (Eston et al., 2007; LaRoche & Connelly, 2006). After 5 weeks of PNF training of the knee flexors, Eston et al. (2007) reported that flexibility training increased range of motion (ROM) and enhanced recovery of isometric strength at a long muscle length but it had

no effect on muscle soreness or isometric strength at a short muscle length following eccentric exercise. LaRoche and Connelly (2006) had subjects perform either 4 weeks of static or ballistic stretching and found that neither training method affected muscle soreness or stiffness after maximal eccentric exercise. These studies conflict with both McHugh et al.'s (1999b) theory that more compliant muscles are less susceptible to muscle damage, and with the results from the above-mentioned Chen study where flexibility training significantly affected the amount of muscle damage incurred by the hamstrings.

Static stretching has been previously documented to significantly shift the optimal angle to a longer muscle length (Ferreira et al., 2007; Chen et al., 2011). Optimal angle is defined as the joint angle at which peak torque is achieved during a muscle contraction (Brockett et al., 2001) and is often measured as the average peak torque across several repeated concentric muscle contractions (see figure 1.1 for example of optimal angle graph). Worrell et al. (2001) reported 30° as the angle at which peak torque is generated during prone knee flexion when the hip joint is fixed at 0°. Older studies report similar findings with knee flexion angle of peak torque in the range of 10° to 30° (Inman et al., 1981). However, optimal angle for peak torque can vary for movements such as knee flexion depending on testing conditions (i.e. hip fixed at 0° vs 90° during isokinetic dynamometry) due to the muscle's mechanical properties (i.e. length-tension relationship) (Krishnan & Williams, 2014). Animal studies have reported that shifts in optimal angle to a longer muscle length, attenuate the effects of eccentric EIMD (Lynn et al., 1994; Lynn et al., 1998). Shifts in the optimum angle for peak torque are often obtained by subtracting the optimum angle before the exercise from that measured after the exercise (Brockett et al., 2001). Similar to Chen et al. (2011), McHugh et al. (1999b) theorized that one of the mechanisms underlying chronic stretching's protective adaptation is an increase in optimal

angle of peak torque related to a longitudinal increase in the number of sarcomeres in series (i.e. sarcomerogenesis).

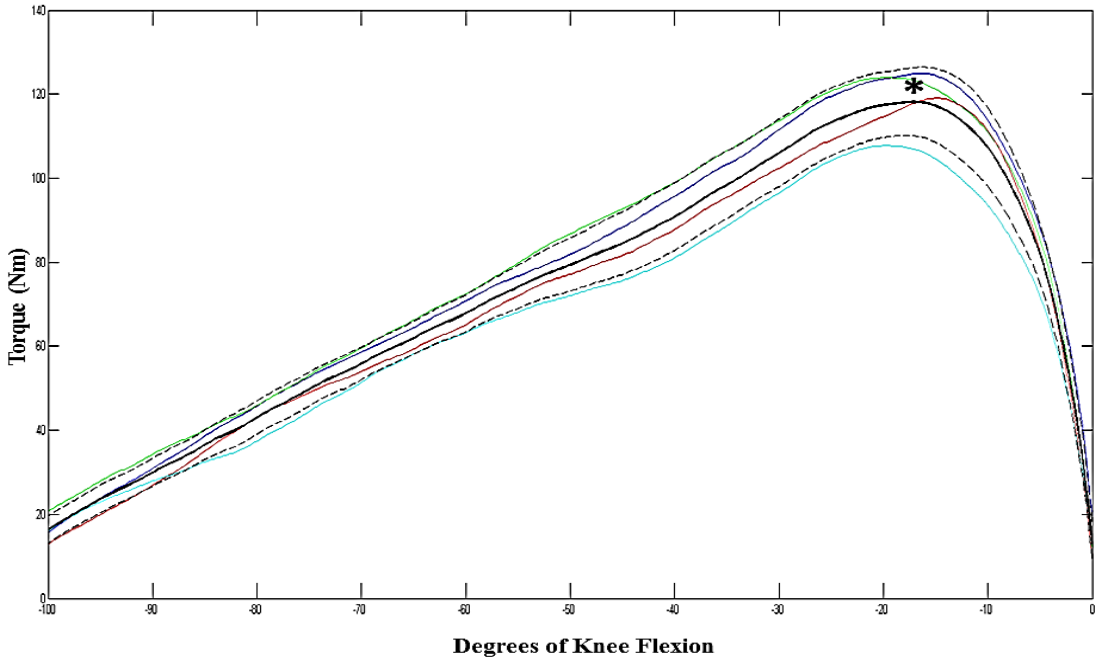

Figure 1.1 Example trace of optimal angle curves during concentric knee flexion in prone position (isokinetic dynamometry at 60°/s)

*Indicates mean peak torque during knee flexion (i.e. optimal angle).
——— Dark, bold curve is average trace curve across multiple concentric repetitions.
------- Dashed line indicates SD ±
Data on graph obtained from a participant during a single testing time point from present study.

Sarcomerogenesis remains the most likely mechanism by which optimal angle and ROM increases following either eccentric or static stretching exercise. Nelson and Bandy (2004) investigated the effectiveness of eccentric training and compared it to static stretching activities on the hamstring muscles in high school males. They found no significant difference between eccentric training and static stretching for increasing hamstring flexibility, and both groups showed significant improvement in flexibility when compared to the control group. The authors suggested that prolonged flexibility training and eccentric training might result in similar

adaptations. Static stretch training and eccentric training both shift the muscle length tension curve (an indication of sarcomerogenesis) which optimizes generation of torque at more extended joint positions and limits the potential for muscle damage providing protection for a subsequent bout of eccentric exercise (O'Sullivan et al., 2012, Kilgallon et al., 2007). The largest difference between the two types of exercise is the amount of DOMS and muscle damage experienced following eccentric exercise in comparison to static stretching (Proske & Morgan, 2001).

Following a thorough examination of the literature, it appears that no study has directly compared the repeated bout effect to the protective effects of chronic flexibility training. Similarly, although several studies have examined either the repeated bout effect or flexibility training with subsequent isolated bouts of eccentric exercise, no study has examined the protective effect of a single intense bout of eccentric exercise or a period of flexibility training for a subsequent period of eccentric strength-training. Nelson and Bandy (2004) is the only study to have compared static stretching and eccentric training but solely focused their examination on how these two training methods affected flexibility and ROM. Further, no study has examined the potential performance benefits that these protective methods have for the acute phase of an eccentric strength-training program. The prophylactic effects associated with both the repeated bout effect and flexibility training for subsequent acute eccentric exercise could potentially be used to help maximize or expedite gains during a chronic eccentric strength-training program.

Training interventions or specific methods used to protect against subsequent eccentric EIMD, as well as maximize performance benefits from subsequent eccentric strength training, will be referred to as "muscle primers" throughout this thesis document. A muscle that has been primed for eccentric training would be predicted to have attenuated DOMS (i.e. indirect marker

of EIMD), power loss; shift the optimal angle to a longer muscle length; and an increase in the rate and/or magnitude of strength gain and hypertrophy. If both methods prove proficient at priming a muscle for eccentric strength training, it would be interesting to further investigate whether the magnitude of effect is greater for one priming method over the other.

Based on previous literature, both muscle priming modalities seem to offer similar protective benefits with eccentric exercise and since there is no evidence yet to suggest that one method would be superior over the other, it is justified to predict that both priming methods will share similar performance outcomes following a full phase of eccentric strength-training.

1.3 Purpose Statement and Hypothesis

1.3.1 Purpose Statement

The purpose of this study is to examine the muscle physiological responses to eccentric strength training after first priming the muscles with either a period of static flexibility training or a single intense bout of eccentric exercise performed weeks earlier; and compare these to the responses from eccentric strength-training when no prior intervention was administered.

1.3.2 Hypothesis

1) Both muscle-priming methods would offer a greater protective effect during the acute stage of eccentric strength training compared to the control group. The groups that receive a muscle priming intervention will exhibit less muscle soreness and smaller decreases in isometric strength and maximal power output compared to the control group in the first 2 weeks of eccentric-strength training.

2) The enhanced protective effect realized by the muscle priming groups during the acute stages of eccentric strength-training would facilitate greater training induced increases in muscle strength, hypertrophy, muscle activation and maximal power output, and shift the optimal angle

to a longer muscle length following the completion of the period of eccentric strength-training; compared to the control group.

Chapter 2: Methods

2.1 Study Design

This study employed a between-within subjects design. For the initial pre-testing session, participants were randomly assigned to one of three experimental groups. Each group received a different priming intervention preceded by a subsequent period of eccentric training specifically targeting the knee flexors muscle group (hamstrings). The groups consisted of a flexibility (n=8; 5 male, 3 female), a single-bout (n=9; 3 male, 6 female), or a control (n=8; 4 male, 4 female) group. Dependent measures of muscle performance and damage included muscle thickness, isometric MVC, eccentric and concentric MVC, optimal angle, active ROM, passive ROM, maximal power, EMG and DOMS. Each full testing period (baseline (pre), post-priming intervention (mid) and post-eccentric training (post)) was completed within a single lab testing session. For each testing session, muscle thickness was measured first followed by flexibility measures of active ROM and passive ROM, then maximal power, and finally a randomized order for testing isometric, concentric, and eccentric MVC. After completing baseline measures for muscle performance, each group proceeded to the priming intervention phase of the study, which occurred across a 4-week period. The flexibility group completed a 4-week period of static stretching. The single-bout group performed a single, intense bout of eccentric exercise after baseline testing, and then measures for DOMS, isometric MVC and maximal power were completed immediately post eccentric bout, at 24 and 48 hours, followed by 4 weeks of recovery. Following baseline measures, the control group received no priming intervention throughout this 4-week period. At the end of the 4-week

priming intervention stage, all groups were re-tested for the same dependent measures as used during baseline testing. All groups then proceeded to complete a 4-week phase of eccentric training. All groups were tested briefly (DOMS, isometric MVC and maximal power only) immediately after the first and fourth eccentric training bouts with follow up testing at 24 and 48 hours following these training bouts. Finally, all groups were tested for all dependent measures again at the end of the 4-week eccentric training phase. Refer to figure 2.1 for a complete breakdown of the study design and timeline.

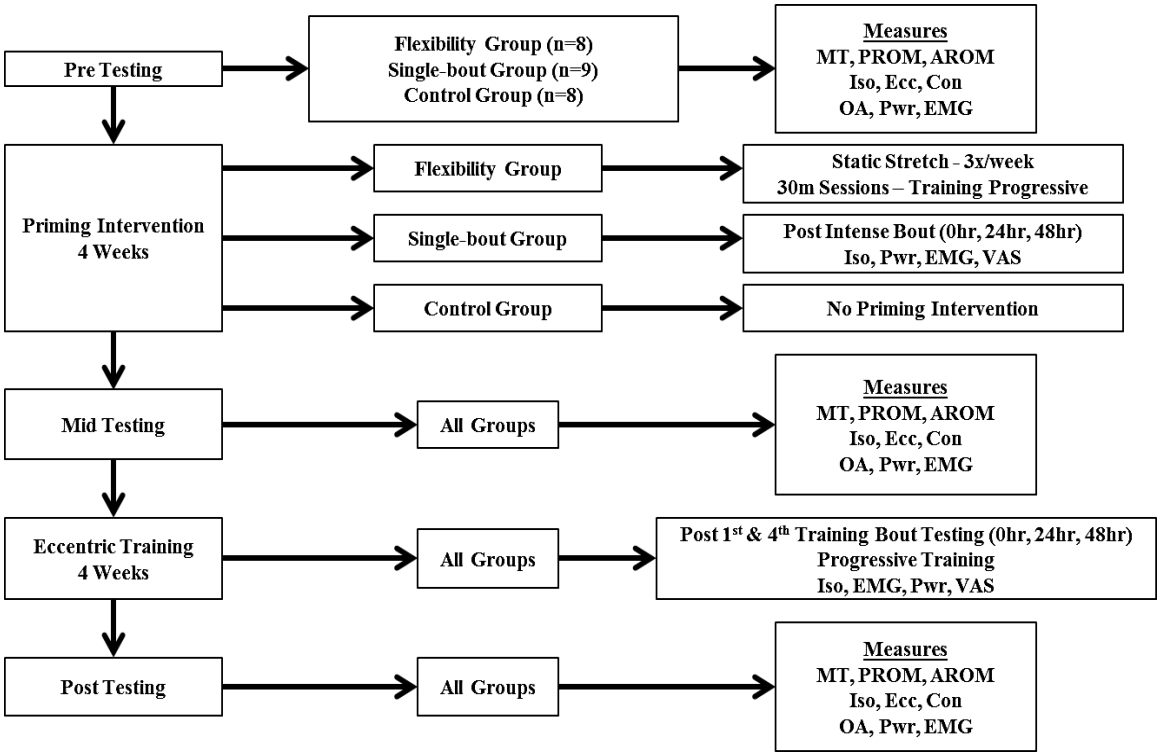

Figure 2.1 Study design & timeline

Dependent Variables: Muscle Thickness (MT), Passive Range of Motion (PROM), Active Range of Motion (AROM), Isometric MVC (Iso), Eccentric MVC (Ecc), Concentric MVC (Con), Optimal Angle (OA), Maximal Power (Pwr), Electromyography (EMG)

2.2 Participants

A sample of 29 volunteer participants from the University of Saskatchewan community was initially recruited for the study. Online advertisements and oral presentations were used to recruit the participants. Four individuals dropped out of the study and did not complete the final testing protocol (two participants dropped out due to scheduling conflicts with school or employment, and two for personal reasons). A final sample of 25 participants (male: n=12; and female: n=13) between the ages of 19-34 completed the entirety of the study. This study was approved by the University of Saskatchewan biomedical review board for research in human subjects (Appendix A), and all subjects gave their written informed consent (Appendix B) prior to data collection. Participants' mean (±SD) height, body weight, age and footedness score was 173.8 ± 7.8 cm, 68.6 ± 16.5 kg, 22.5 ± 4.2 years and 9.5 ± 4.0 respectively. Footedness was determined using a 10-item version of the Waterloo Footedness Questionnaire (WFQ) (Appendix D). Scores on the WFQ can range from -20 to +20 where negative score indicate left-footedness and positive scores indicate right-footedness. Participants' descriptive statistics and characteristics are show in table 3.1.

Sample size calculations were run using G*Power3.1 (Faul et al., 2009) based on the data from a previous unpublished pilot study done in our lab (Leslie et al., 2011 unpublished). Based on an effect size estimate of 0.25, and 80% power with an alpha level = 0.05, sample size calculations for each variable were run using a 3 (group) x 3 (time) ANOVA and assuming necessary adjustment for violations of sphericity. The required sample size for the between group design was estimated at n=12 per group. All participants were required to meet three criteria in order to participate in the study. First,

participants had limited prior resistance training experience (i.e. resistance training in previous year = < 4 months, lifetime = < 36 months), with no prior history of eccentric training or structured flexibility training of the knee flexors. Participants were asked to avoid flexibility and eccentric training of the knee flexors throughout the duration of the study. If participants were engaging in any current resistance, flexibility or cardiovascular training program, they were encouraged to continue with no increases to the volume (frequency or intensity) or changes to the type of exercises. No new resistance, flexibility or cardiovascular training program could be started other than that required for the study. Second, participants were otherwise healthy and injury free, with no history of knee, thigh, and hip or back problems within the last full calendar year prior to the study. Each participant completed a questionnaire regarding their previous training experience and history of injury (Appendix E) to ensure they met the previously stated inclusion criteria. Third, the participants needed limited passive ROM of the knee flexors. This was important to avoid the possibility of ceiling effects influencing the magnitude of change from flexibility training. Limited passive ROM was defined as 30° knee-extension deficit with the hip at 90° (Bandy & Irion, 1994). Any volunteers not meeting the 3 inclusion criteria were excluded from participating in the study.

2.3 Testing Procedures

2.3.1 Muscle Thickness

Muscle thickness of the biceps femoris long head (BF^{LH}) was measured using a portable ultrasound scanner (LOGIQ e BTO8, GE Healthcare, Milwaukee, Wisconsin, USA) equipped with a linear (38.4 mm) 12Hz transducer (resolution 0.3 mm) to determine if changes in muscle thickness occurred in response to both the flexibility and

the eccentric exercise training. The BF^{LH} was selected as the most clinically relevant, as most hamstring muscle strain injuries involve the BF^{LH} muscle (Askling et al., 2007) and it is easily measured with ultrasound due to its superficial location within the hamstring muscle group. Four longitudinal images of the BF^{LH} were taken while the participant was lying in the prone position, and the average of the two closest measurements was used for analysis. Additional measurements were taken if any two of the four measurements were not within 1mm (Farthing & Chilibeck, 2003). The best images were selected as determined by the visual clarity of the structures. Muscle thickness was defined as the mean of the vertical distances delimited by superficial and deep aponeuroses measured at both image extremities (Lima et al., 2015). The probe was placed at 50% of thigh length, defined as the distance from the greater trochanter to the popliteal crease. Anatomical landmarks and probe positioning was marked on transparent paper at baseline testing (Farthing & Chilibeck, 2003). This ensured consistent probe placement during subsequent testing time points. Muscle thickness measures have been found to be reliable in our lab across various muscle groups of the upper and lower body (Farthing & Chilibeck, 2003; Krentz et al., 2008; Krentz & Farthing, 2010). The coefficient of variance (CV) for the current muscle thickness protocol for the BF^{LH} muscle was 1.7%.

2.3.2 Electromyography

Muscle activation was assessed using EMG during isometric MVC, eccentric MVC and concentric MVC tests, as well as during the maximal power tests to determine the changes in muscle activation throughout the study. A Delsys EMG (Bagnoli-2 or Bagnoli-4, Delsys Inc., Boston, MA) system was used to assess muscle activation during the maximal strength and power tests on each testing occasion. EMG surface electrodes

were placed over the BF^{LH} on the designated testing leg. The electrode was placed at 50% on the line between the ischial tuberosity and the lateral epicondyle of the tibia (SENIAM, 1996). A reference electrode was place on the lateral malleolus to serve as a common ground for the EMG signal. The overall amplification or gain was 1K for the agonist muscle (knee flexors). The system noise was <1.2 μV (rms) for the specified bandwidth. The electrodes consist of two silver bars (10 X 1mm diameter) spaced 10mm apart, with a common mode rejection ratio (CMRR) of 92 dB. Raw EMG signals (in mV) were converted to root mean square (RMS) using MATLAB (version 7.3.0). RMS was used to obtain an estimate of the amplitude of the signal, measured as a function of time and calculated using a defined window. EMG activation collected during dynamic contraction tests (eccentric MVC, concentric MVC, maximal power) was normalized to the isometric MVC. The CV for normalized EMG activation during eccentric MVC, concentric MVC, and maximal power tests was 13.7%, 13.1%, 20.5%, respectively.

2.3.3 Active Range of Motion

Active ROM of knee flexors was assessed using a classic sit-and-reach test protocol as outlined by the Canadian Physical Activity Training for Health Manual (PATH). The CV for the classic sit-and-reach test for knee flexor extensibility was 6.8%. In order to reduce the variability and standard error of measurements, participants were given a five-minute warm-up on a stationary bike as differences in body temperature have been shown to effect muscle flexibility (Dixon & Keating, 2000). Participants sat straight legged with the soles of their feet flat against the Flexometer. Participants were instructed to keep knees fully extended, then with arms evenly extended forward, placing one hand over the other. With palms pronated downwards, the participant pushed the

sliding marker as far down the measuring scale until a minor discomfort was felt. They held the reach for approximately two seconds. The highest score achieved from three trials was recorded.

2.3.4 Passive Range of Motion

Passive ROM of the knee flexors was assessed using passive straight leg raise test on a dynamometer (Humac NORM, CSMi, Stoughton, MA). The CV for the passive straight leg raise test, using the dynamometer, was 1.6%. The participant was placed in a supine position with legs straight on the dynamometer table. A trained examiner palpated the greater trochanter to ensure that the hip joint approximate center was properly lined up with the dynamometer axis of rotation. The contralateral straight leg was held in place with a Velcro strap to enable maximum stability in all planes of motion and the tester controlled the pelvis to prevent posterior pelvic tilt. A long attachment bar was used and secured to the testing leg posterior and superior calcaneus. The leg was passively lifted into hip flexion and the examiner kept the knee joint fixed in extension. The end range of the straight-leg raising was determined by the examiner's perception of a firm resistance to stretch and/or the palpable onset of posterior pelvic tilt (Ayala et al., 2011). The passive ROM was measured by the dynamometer and recorded.

2.3.5 Strength and Power

All strength and power measures were completed using isokinetic dynamometry (Humac NORM, CSMi, Stoughton, MA) to assess the changes in strength and power throughout the study. Participants were placed in a prone hamstring curl position with their pelvis and testing leg secured to the dynamometer using the manufacturer's specifications and the non-testing leg was placed in full knee extension and the hip at 0°

of extension/flexion on a separate chair for comfort for all strength and power tests. A five-minute warm up on a stationary bike was given prior to any strength and power testing. A brief familiarization period in which participants performed several sub-maximal contractions to help minimize the effect of learning was given prior to each strength and power test.

2.3.6 Isometric Maximal Voluntary Contraction

Maximal isometric knee flexor torque (Nm) measures were taken at baseline, post-priming intervention phase and post-eccentric training phase. All groups were also tested for isometric MVC strength immediately after the 1st and 4th eccentric training bout with follow up testing proceeding at 24 and 48 hours following these training bouts. The single bout group was also tested for isometric MVC strength immediately post single eccentric bout, and at 24 and 48 hours post single bout. During isometric MVC testing, the knee was positioned at 30° flexion from full extension, while the hip remained at 0° of extension/flexion, which is the angle of peak force production for the knee flexors during isometric MVC in the prone position (Pincivero et al., 2003). Optimal angle for peak torque of the knee flexors with a hip angle at 0° occurs at angles ranging from 10° to 30° (Inman et al., 1981). Three isometric MVCs were performed, each being held for three seconds with concurrent verbal encouragement, followed by a one-minute rest period and feedback of performance. The peak of the three contractions was used during analysis. The CV for isometric MVC strength of the knee flexors was 5.2%.

2.3.7 Eccentric Maximal Voluntary Contraction

Maximal eccentric knee flexor torque (Nm) measures were taken at baseline, post-priming intervention phase and post-eccentric training phase. With an unloaded

limb, the testing knee was moved to110° flexion from full extension (0°) at which time a maximal contraction of the knee flexors was initiated. The maximal voluntary eccentric contraction was performed at a velocity of 30°/sec (0.52rad/sec). Three eccentric MVCs were performed with concurrent verbal encouragement, followed by a one-minute rest period and feedback of performance. The peak of the maximal eccentric contractions was used for analysis. The CV for eccentric MVC strength of the knee flexors was 6.8%.

2.3.8 Concentric Maximal Voluntary Contraction & Optimal Angle

Maximal concentric knee flexor torque (Nm) measures were taken at baseline, post-priming intervention phase and post-eccentric training phase. Optimal angle data were extracted from the torque tracing during the concentric MVC. Beginning from full knee extension (0°), participants performed several (min of 3; max of 6) voluntary maximal concentric contractions, throughout a ROM of 110° of knee flexion, at constant velocity of 60°/s (1.05rad/sec) (Brockett et al., 2001). In order to determine the optimal angle, the torque values during knee flexion were extracted in a time series and ordered according to knee angle. A spline interpolation function was used to resample the data from each trial at regular joint angle intervals so that different trials could be averaged at the same angular positions across the movement. Values were plotted on an averaged curve across repetitions. The peak torque of the concentric MVC was also used for analysis. The CV for concentric MVC strength of the knee flexors and optimal angle determination was 7.2% and 16.0%, respectively.

2.3.9 Maximal Power

Velocity-dependent contractions (concentric knee flexion), using the “isotonic” mode on the dynamometer, were performed at baseline, post-priming intervention phase

and post-eccentric training phase to measure maximal power. All groups were also tested for maximal power immediately after the first and fourth eccentric training bout with follow up testing proceeding at 24 and 48 hours following these training bouts. The single-bout group was also tested immediately post single eccentric bout, and at 24 and 48 hours post single bout. Velocity-dependent contractions were performed with a load of 20% of isometric MVC. Three maximal effort contractions were performed with concurrent verbal encouragement, followed by a two-minute rest period and feedback of performance. Maximal power (W) was calculated using the velocity and torque signals from the velocity-dependent contractions and was then used for analysis. The CV for maximal power from the velocity-dependent contractions was 7.7%.

2.3.10 Muscle Soreness

A subjective scale of muscle soreness was used to assess DOMS. A visual analog scale (VAS) was used consisting of a 100mm line where 0mm indicated, “not sore at all” and 100mm indicated “very, very sore”. VAS has been used as a reliable method for measuring muscle soreness following eccentric bouts (Chapman et al., 2006; Mattacola et al., 1997). The participants were asked to gently flex and extend the unloaded knee, as well as perform toe touches to stretch the knee flexors, as well as self-palpation along the surface of the hamstrings. Participants then indicated the degree of soreness experienced, recorded on the VAS. A rating of muscle soreness was done immediately after the first and fourth eccentric training bout with follow up testing proceeding at 24 and 48 hours. The single-bout group was also asked to rate soreness immediately after the single eccentric bout performed after baseline testing, and at 24 and 48 hours post single bout.

2.4 Training Phases

2.4.1 Priming Intervention: Flexibility Training

Static flexibility training was completed three times per week for four weeks (n = 12 sessions) with 48 hours rest between sessions. Participants in the flexibility group performed static stretching of the knee flexors, under supervision by the researcher, preceded by a five minute warm up on a stationary bike. A supine, straight leg hamstring stretch was used where one hip was flexed, with knee in full extension, while the opposite leg laid flat on the floor in full extension. The leg being stretch was moved slowly to the point that the participant indicated a feeling of mild discomfort or until the researcher felt a distinct resistance to stretch, and then held in place against a doorframe. The researcher ensured that pelvis maintained a neutral position with both hips equally on the floor. The total time under tension for each session was fifteen minutes per leg. The length of time each rep was held was progressively increased during the training period, beginning with five sets of three-minute holds and ending three sets of five-minute hold. During the stretch sessions, participants' hip angle was decreased upon verbal indication that no stretch discomfort was being felt throughout the repetition. Thirty-seconds of rest was given between repetitions. Both legs were stretched to avoid any asymmetries or injuries following training.

2.4.2 Priming Intervention: Single Eccentric Bout

A single eccentric bout was completed four weeks prior to the beginning of the eccentric training phase. Participants in the single-bout group performed the eccentric exercise on a dynamometer using the identical prone knee flexor ("hamstring curl") position as used for the strength and power measures. The single session was preceded by

a five-minute warm-up on a stationary bike. Six sets of eight repetitions of voluntary velocity-dependent contractions (eccentric knee flexion) with a load of 80% of isometric MVC (using the "isotonic" mode on the dynamometer) were performed with a one-minute rest in between sets. The researcher assisted in flexing the knee to 110° flexion and then at this point, participants initiated a near-maximal or maximal (once fatigued) contraction of the knee flexors to slowly lower the load down until full knee extension (0°) was reached. The process was the repeated for each rep and both legs were trained to avoid any strength asymmetries. The participants were tested at 0hr, 24hr and 48hr for muscle soreness, isometric MVC and maximal power. This process was completed in order to ensure the single intense bout was adequately damaging and participants were experiencing normal recovery. Participants then rested and recovered for four weeks following this single intense bout of eccentric exercise.

2.4.3 Eccentric Training

Eccentric training was completed by all groups, three times per week, with 48 hours rest between sessions, for four weeks (n = 12 sessions) and performed on a dynamometer using the identical prone knee flexor ("hamstring curl") position as used for the strength and power measures. Training sessions were preceded by a five-minute warm-up on a stationary bike. Initially, three sets of eight repetitions of voluntary velocity-dependent contractions (eccentric knee flexion) with a load of 80% of isometric MVC (using the "isotonic" mode on the dynamometer) were performed with one minute of rest between sets. The researcher assisted in flexing the knee to 110° and then at this point, participants initiated a near-maximal or maximal (once fatigued) contraction of the knee flexors to slowly lower the load down until full knee extension (0°) was reached.

The process was the repeated for each rep and both legs were trained to avoid any strength asymmetries. The training volume was progressive across the four eccentric training weeks. The sets were progressed by one extra set each session up to a maximum of six sets. At that point, the percentage load of isometric MVC was progressively increased weekly (week two = 80%, week three = 85%, week four = 90%).

2.5 Data Acquisition

Custom software in LabVIEW (version 8.6) was used to obtain torque, position, velocity, and EMG signals simultaneously. All channels were acquired at a sampling rate of 1,000 Hz. An analog-to-digital converter (model PCI-6034E, National Instruments, Austin, TX) was used to convert the analog signals from each device to digital signals displayed in LabVIEW. Acquisition data files were extracted for offline analysis using MATLAB (version 7.3.0).

2.6 Statistical Analysis

Prior to detailed analyses of the intervention effects, all dependent measures were tested for normality using tests of skewness and kurtosis, and baseline differences between groups were examined using one-way ANOVA. The acute response to the single intense bout of eccentric training for the single bout group was analyzed using one-way repeated measures ANOVA.

2.6.1 Acute Responses to Eccentric Training Data

To determine the effects that different types of priming interventions had during the first two-weeks of eccentric training, data were analyzed using one between-subjects factor of *Group* and one within-subjects factor of *Time*, for each dependant variable (isometric MVC, maximal power). Separate 3 (*Group*; flexibility group, single-bout

group, control group) x 3 (*Time*; 0hr, 24hr, 48hr) repeated measures ANCOVA was completed, with the respective mid-testing data point (pre-eccentric training) for each dependent variable serving as a covariate. The mid-testing data point was used as a covariate because MVC and maximal power measure were not taken immediately before the first and fourth bout of training.

Acute muscle soreness data were found to violate the assumption of normality (flexibility group had skewness score of 1.573 (*SE* = 0.75) at 0h post fourth-bout, both the flexibility and single-bout group had skewness scores of 2.45 (*SE* = 0.75) and 2.30 (*SE* = 0.69) 48h post fourth-bout respectively) due to floor effects. Therefore, to determine the effects of the priming interventions on muscle soreness during the first two-weeks of eccentric training, data were analyzed using non-parametric tests for related and independent samples. Differences between time points (0, 24, 48 hrs) for each group were examined using Friedman's ANOVA by ranks. Differences between groups (flexibility group, single-bout group, control group) at each time point were examined using Kruskal-Wallis ANOVA. Differences between week 1 and 2 pooled across groups at each time point were examined using Wilcoxon Signed Rank test.

2.6.2 Post-Eccentric Training Data

To determine the effects of the priming interventions following subsequent eccentric training, data were analyzed using one between-subjects factor of *Group* and one within-subjects factor of *Time* for six-dependant variables (muscle thickness, passive ROM, active ROM, optimal angle, maximal power, EMG). For each dependent variable, separate 3 (*Group*; flexibility group, single-bout group, control group) x 3 (*Time*; pre, mid, post) repeated measures ANOVAs were completed.

For the three dependent variables measuring maximal strength (isometric MVC, eccentric MVC, concentric MVC), data were analyzed using a 3 (*Group*; flexibility group, single-bout group, control group) x 3 (*Time*; pre, mid, post) x 3 (*Type*; concentric, eccentric, isometric) repeated measures ANOVA.

If significant interactions or main effects were identified from the ANOVA tests, simple main effects analysis continued using one-way ANOVA and Bonferroni adjusted post hoc tests where appropriate. Post hoc testing for non-parametric tests included Mann-Whitney U and Friedman's ANOVA (using 2 levels) where appropriate. Despite the number of separate analyses for dependent variables, significance was accepted at $p < 0.05$, without further adjustment for Type I error. All values are expressed as mean ± standard deviation. Statistical analysis was completed using SPSS version 21 (Chicago, IL).

Chapter 3: Results

3.1 Participant Characteristics & Training Experience

No differences were observed between groups for age, height, weight or Waterloo footedness score, and there was no difference between groups for resistance training experience; both within the past year or lifetime (Table 3.1).

Table 3.1 Participant characteristics

Characteristics	Pooled Participants (n=25)	Flexibility Group (n=8)	Single-bout Group (n=9)	Control Group (n=8)
Age (Years)	22.5 ± 4.2	22.5 ± 4.6	24.2 ± 5.0	20.6 ± 1.2
Height (cm)	173.8 ± 7.8	174.3 ± 9.1	171.2 ± 8.3	176.3 ± 5.4
Weight (kg)	68.6 ± 16.5	70.1 ± 17.2	67.2 ± 16.7	68.5 ± 17.6
Waterloo Footedness Questionnaire	9.5 ± 4.0	8.9 ± 5.8	10.0 ± 3.6	9.6 ± 2.7
Resistance Training in Previous Year (months)	4.0 ± 3.9	5.6 ± 3.9	2.2 ± 3.8	4.4 ± 3.6
Resistance Training in Lifetime (month)	21.5 ± 21.0	27.8 ± 25.0	15.8 ± 20.9	21.6 ± 17.4

Data listed as means ± standard deviation

3.2 Acute Response to the Single Eccentric Bout

For the single bout group, the intense bout of eccentric exercise (serving as the priming intervention) resulted in significant declines in isometric MVC (19.2%, $p<0.05$) and power (14.6%; $p<0.05$) and increases in VAS score (69.8 ± 20.4mm; $p<0.05$) in the first 48 hours post eccentric exercise as indicators of muscle damage.

3.3 Acute Responses to Eccentric Training

3.3.1 Muscle Soreness

Week 1

The Kruskal-Wallis ANOVA for ranked data revealed a significant difference in muscle soreness between groups at 24hrs post first bout, $\chi^2(2)=7.387$, $p<0.05$, and at 48hrs post first bout, $\chi^2(2)=15.728$, $p<0.001$. Post-hoc analysis using Mann-Whitney *U* tests revealed that both the flexibility and the single-bout group had significantly lower soreness scores at 24hrs post first bout ($p<0.05$) and 48hrs post first bout ($p<0.01$) in comparison to the control group. The Friedman's two-way ANOVA by ranks revealed a significant time main effect for muscle soreness, $\chi^2(2)=16.887$, $p<0.001$. There was a significant increase in muscle soreness over time pooled across groups. When splitting the data by group, the Friedman's two-way ANOVA by ranks indicated that the control group was the only group to experience a significant increase in muscle soreness during the first week, $\chi^2(2)=10.129$, $p<0.01$. Refer to figure 3.1 for a graph of changes in muscle soreness during week one of eccentric training.

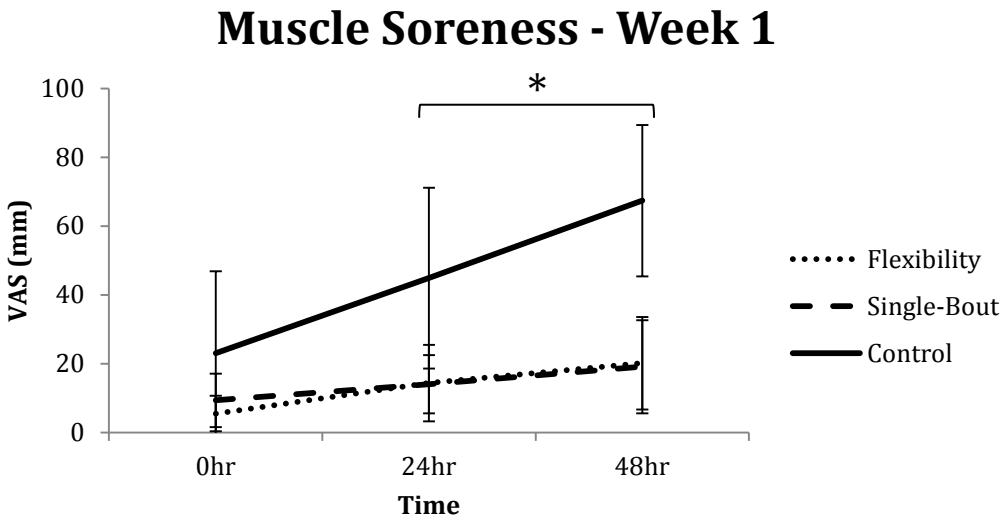

Figure 3.1 Muscle soreness - week 1 over time

* Control sig. different than flexibility & single-bout ($p<0.05$)
Data listed as means ± standard deviation

Week 2

The Kruskal-Wallis ANOVA for ranked data revealed a significant difference in muscle soreness between groups immediately post fourth bout, $\chi^2(2)=12.822$, $p<0.01$, at both 24hrs, $\chi^2(2)=15.421$, $p<0.001$ and 48hrs post fourth bout, $\chi^2(2)=9.471$, $p<0.01$. Post-hoc analysis using Mann-Whitney *U* tests revealed that the flexibility group had significantly lower soreness scores in comparison to the single-bout and control group immediately post fourth bout ($p<0.01$), 24hrs post fourth bout ($p<0.01$) and 48hrs post fourth bout ($p<0.01$ vs. single bout; $p<0.05$ vs. control). The Friedman's two-way ANOVA by ranks revealed a significant time main effect for muscle soreness, $\chi^2(2)=8.321$, $p<0.05$. There was a significant decrease in muscle soreness over time pooled across groups. Overall, soreness was significantly reduced for all groups from week 1 to week 2 ($p<0.001$). Refer to figure 3.2 for a graph of changes in muscle soreness during week two of eccentric training.

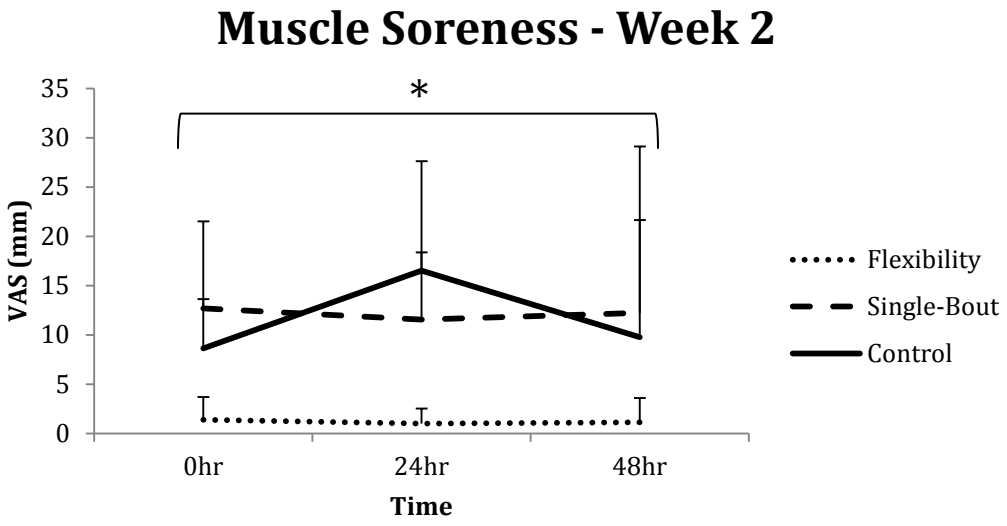

Figure 3.2 Muscle soreness – week 2 over time

* Flexibility sig. different than single-bout & control ($p<0.05$)
Data listed as means ± standard deviation

3.3.2 Maximal Power

Week 1

The omnibus ANCOVA revealed a significant group main effect, $F(2, 20)=4.704$, $p<0.05$, partial $\eta^2= 0.320$. The flexibility group had significantly greater maximal power compared to both the single-bout and the control group pooled across time. There was no significant interaction, $F(4, 40)=1.412$, $p=0.247$, partial $\eta^2= 0.124$, or time main effect, $F(4,40)=1.989$, $p=0.150$, partial $\eta^2= 0.090$, to report for maximal power post first bout. Refer to figure 3.3 for a graph of changes in max power during week one of eccentric training.

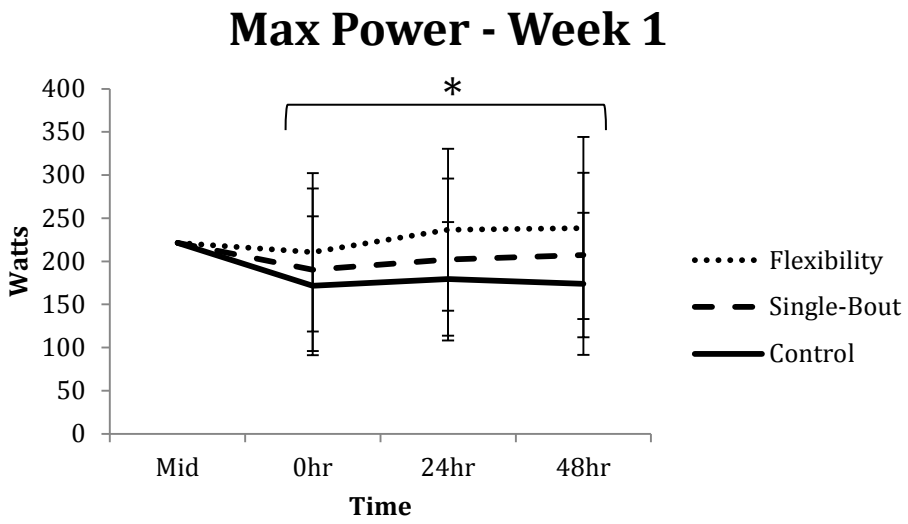

Figure 3.3 Maximal power – week 1 over time

* Flexibility sig. different than single-bout & control pooled across time ($p<0.05$)
Power data are ANCOVA adjusted means, with Mid-testing values as the covariate
Data listed as means ± standard deviation

Week 2

The omnibus ANCOVA revealed no significant interaction, $F(4, 36)=0.870$, $p=0.491$, partial $\eta^2= 0.088$ and no significant group main effect, $F(2, 18)=1.162$, $p=0.335$,

partial η^2= 0.114, or time main effect, $F(2,36)=1.419$, p=0.255, partial η^2= 0.073, to report for maximal power post fourth bout.

3.3.3 Isometric MVC

Week 1

The omnibus ANCOVA revealed no significant interaction, $F(4, 42)=1.631$, p=0.184, partial η^2= 0.134, no significant group main effect, $F(2, 21)=2.090$, p=0.149, partial η^2= 0.166, or time main effect, $F(2, 42)=0.910$, p=0.410, partial η^2= 0.042, to report for isometric MVC post first bout.

Week 2

The omnibus ANCOVA revealed a significant group x time interaction, GG $F(3.1, 30.9)=3.726$, $p<0.05$, partial η^2= 0.271. Post-hoc analysis using the Bryant-Paulson procedure revealed that the isometric MVC for the control group was significantly lower than the flexibility group immediately post fourth eccentric bout ($p<0.05$). The control group had recovered significantly in isometric strength 24hrs post fourth bout ($p<0.05$) and 48hrs post fourth bout ($p<0.05$) compared to immediately post fourth bout. Refer to figure 3.4 for a graph of changes in isometric MVC during week two of eccentric training.

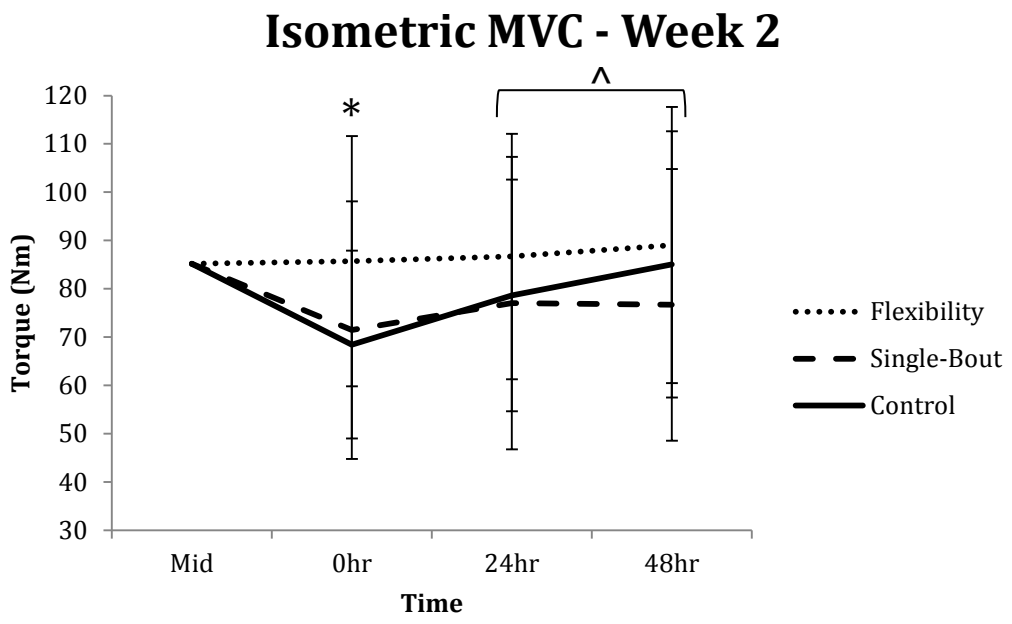

Figure 3.4 Isometric MVC torque – week 2 over time

* Flexibility sig. different than control (p<0.05)
^ Control sig. different than 0hr (p<0.05)
Torque data are ANCOVA adjusted means, with Mid-testing values as the covariate
Data listed as means ± standard deviation

3.4.4 Electromyography

Week 1

The omnibus ANCOVA revealed no significant interaction, GG F(2.6, 26.5)=0.894, p=0.446, partial η^2= 0.082, and no significant group main effect, F(2, 20)=0.096, p=0.909, partial η^2= 0.009, or time main effect, GG F(1.3, 26.5)=0.146, p=0.775, partial η^2= 0.007, to report for the EMG recorded during the velocity dependent contractions post first bout. Average normalized EMG values were 1.2 ± 0.3 at 48hrs post first bout.

Week 2

The omnibus ANCOVA revealed no significant interaction, F(4, 36)=0.315, p=0.866, partial η^2= 0.034, and no significant group main effect, F(2, 18)=0.875,

p=0.434, partial η^2= 0.089, or time main effect, F(2, 36)=1.239, p=0.302, partial η^2= 0.64, to report for the EMG recorded during the velocity dependent contractions post fourth bout. Average normalized EMG values were 1.2 ± 0.5 at 48hrs post fourth bout.

3.4 Post-Eccentric Training

3.4.1 Muscle Thickness

There were no baseline differences between groups to report for muscle thickness. The omnibus ANOVA revealed a significant time main effect, F(2, 44)=36.836, p<0.001, partial η^2= 0.626.There was a significant increase in muscle thickness over time pooled across groups. There was no significant interaction, F(4, 44)=2.219, p=0.082, partial η^2= 0.168, or group main effect, F(2, 22)=2.066, p=0.151, partial η^2= 0.158, for muscle thickness. Refer to figure 3.5 for a graph of changes in muscle thickness.

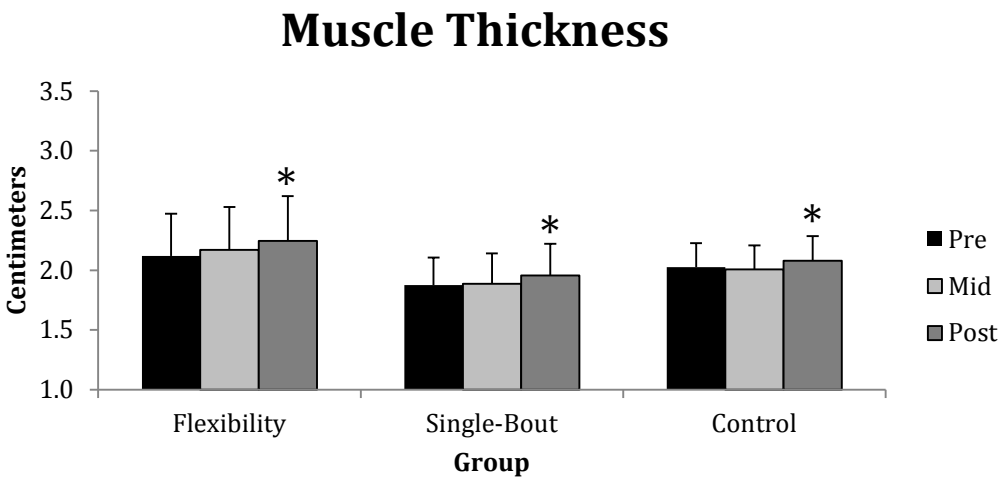

Figure 3.5 Muscle thickness vs group

*Sig. main effect of time pooled across groups (p<0.001)
Data listed as means ± standard deviation

3.4.2 Active Range of Motion

There were no baseline differences between groups to report for active ROM. The omnibus ANOVA revealed a significant group x time interaction, GG $F(2.7, 30.1)=16.025$, $p<0.001$, partial $\eta^2= 0.593$. Bonferroni adjusted post-hoc analysis revealed an increase in active ROM for the flexibility group following flexibility training ($p<0.001$). The flexibility group decreased in active ROM following the eccentric training phase ($p=0.001$) but remained significantly greater than baseline values ($p<0.01$). Refer to figure 3.6 for a graph of changes in active ROM.

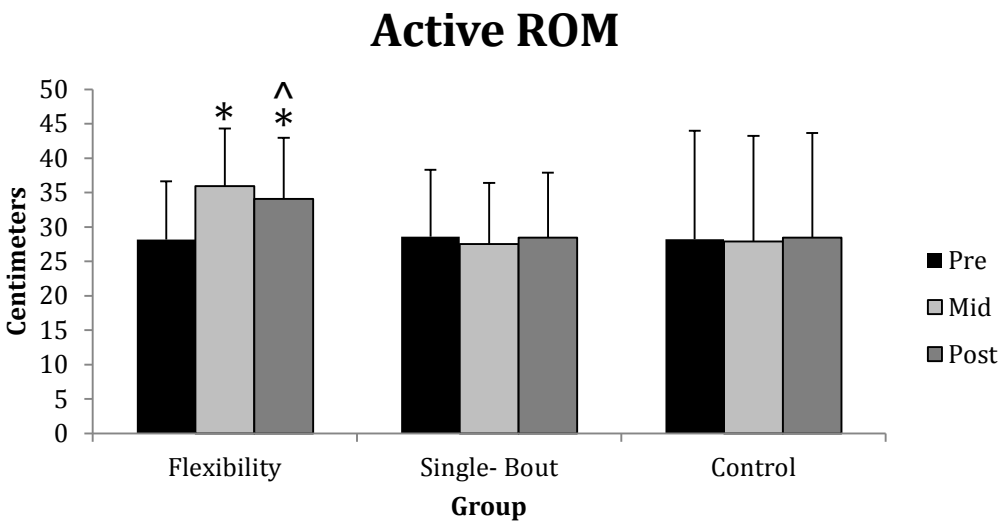

Figure 3.6 Active ROM vs group

* Sig. different than previous time point ($p<0.01$)
^ Sig. different than Pre ($p<0.01$)
Data listed as means ± standard deviation

3.4.3 Passive Range of Motion

There were no baseline differences between groups to report for passive ROM. The omnibus ANOVA revealed a significant group x time interaction, GG $F(3.2,$

34.7)=22.518, $p<0.001$, partial $\eta^2= 0.672$. Bonferroni adjusted post-hoc analysis revealed that the flexibility group increased in passive ROM following flexibility training ($p<0.001$). The flexibility group decreased in passive ROM following the eccentric training phase ($p<0.05$) but remained significantly greater than baseline values ($p=0.001$). The control group increased in passive ROM following the eccentric training phase ($p<0.005$). Refer to figure 3.7 for a graph of changes in passive ROM.

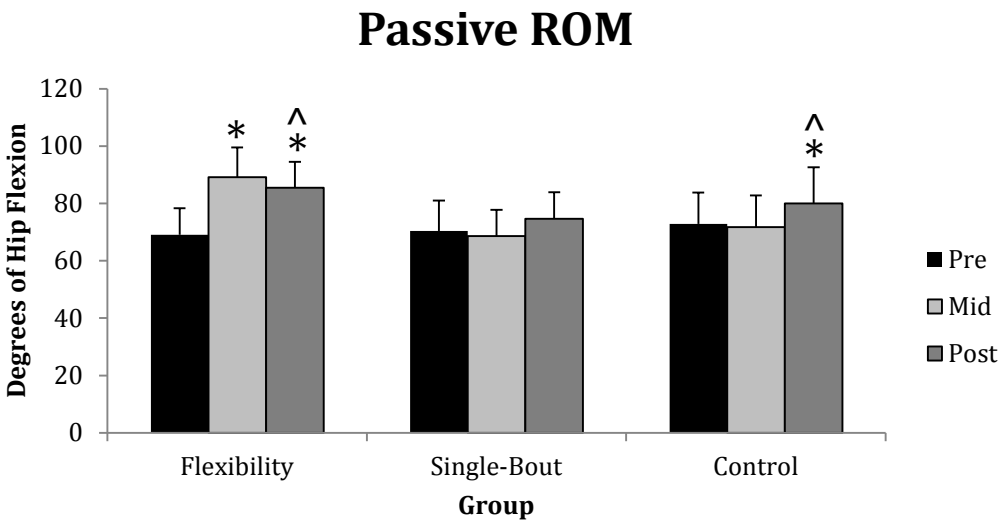

Figure 3.7 Passive ROM vs group

* Sig. different than previous time point ($p<0.01$)
^ Sig. different than Pre ($p<0.05$)
Data listed as means ± standard deviation

3.4.4 Optimal Angle

There were no baseline differences between groups to report for optimal angle. The omnibus ANOVA revealed a significant time main effect, $F(2, 42)=4.818$, $p<0.05$, partial $\eta^2= 0.187$. There was a significant shift in optimal angle towards a greater muscle length over time pooled across groups. There was no significant interaction, $F(4, 42)=1.999$, $p=0.112$, partial $\eta^2= 0.160$, or group main effect, $F(2, 21)=1.557$, $p=0.234$,

partial η^2= 0.129, for optimal angle. Refer to figure 3.8 for a graph of changes in optimal angle.

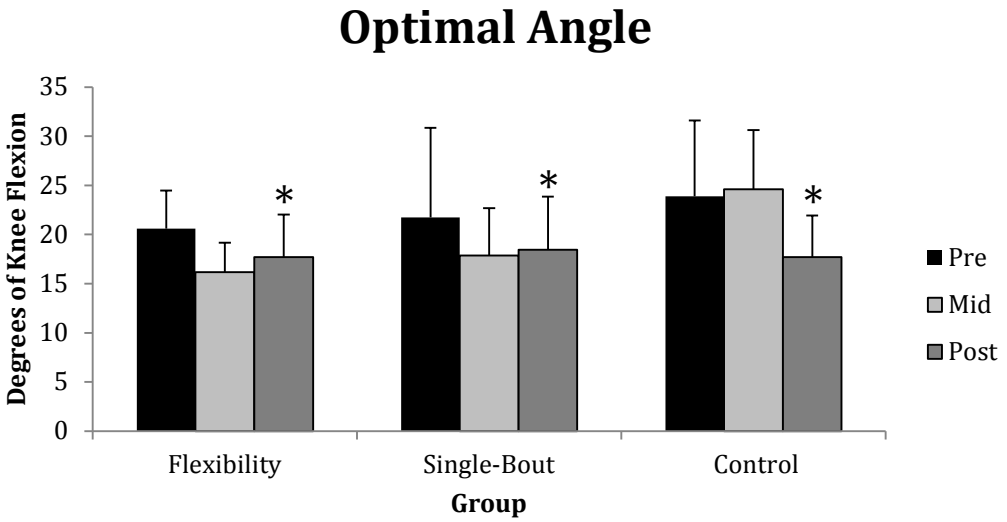

Figure 3.8 Optimal angle vs group

* Sig. main effect of time pooled across groups (p<0.05)
Data listed as means ± standard deviation

3.4.5 Maximal Voluntary Contractions

There were no baseline differences between groups to report for MVC. The omnibus ANOVA revealed a significant type x time interaction $F(4, 88)=9.375$, p<0.001, partial η^2= 0.299. Bonferroni adjusted post-hoc analysis revealed that eccentric, concentric and isometric MVC strength all increased following eccentric training phase (p<0.001). Further comparison using difference scores (from baseline to post-testing) for all three contraction types using a repeated measures ANOVA with Bonferroni adjusted post-hoc analysis revealed that eccentric strength increased to a greater extent than both concentric and isometric strength following eccentric training (p<0.01). There were no other significant interactions for MVC (refer to table 3.2).

Table 3.2 Torque value (Nm) for contraction type over time

	Ecc MVC (30°/s)*			Iso MVC			Con MVC (60°/s)		
Group	***Pre***	***Mid***	***Post***	***Pre***	***Mid***	***Post***	***Pre***	***Mid***	***Post***
F	107.3 ± 28.4	113.3 ± 34.6	127.0 ± 37.3	93.4 ± 23.6	95.0 ± 30.1	101.9 ± 28.8	87.9 ± 21.8	92.6 ± 28.1	98.2 ± 25.7
SB	89.2 ± 27.9	93.0 ± 33.6	108.4 ± 37.8	75.9 ± 28.2	77.1 ± 31.3	88.4 ± 26.4	74.3 ± 31.1	74.3 ± 27.8	81.9 ± 27.0
C	99.5 ± 30.2	100.6 ± 30.5	115.5 ± 32.2	81.6 ± 21.9	84.6 ± 22.0	92.6 ± 22.3	81.5 ± 25.2	80.0 ± 22.9	85.0 ± 24.8

* Sig. type x time interaction pooled across groups (p<0.001);
Ecc MVC increased more than both Con and Iso (p<0.01).
F = Flexibility; SB = Single-bout; C = Control
Data listed as means ± standard deviation in Newton Meters

3.4.6 Maximal Power

There were no baseline differences between groups to report for maximal power. The omnibus ANOVA revealed a significant time main effect, GG F(1.6, 34.8)=6.282, p<0.01, partial η^2= 0.222. There was a significant increase in maximal power over time pooled across groups. There was no significant interaction, GG F(3.2, 34.8)=0.336, p=0.809, partial η^2= 0.030, or group main effect, F(2, 22)=0.751, p=0.483, partial η^2= 0.064, for maximal power (refer to table 3.3).

Table 3.3 Maximal power over time

	Maximal Power (W)		
Group	***Pre***	***Mid***	***Post***
F	242.9 ± 97.8	253.3 ± 114.3	270.0 ± 110.0*
SB	197.1 ± 80.0	192.5 ± 91.4	217.9 ± 87.2*
C	209.3 ± 101.0	207.7 ± 84.4	223.7 ± 81.2*

* Sig. main effect of time pooled across groups (p<0.01).
Data listed as means ± standard deviation

3.4.7 EMG Muscle Activity - Eccentric MVC

There were no baseline differences between groups to report for muscle activation (RMS amplitude normalized to peak isometric MVC at baseline) during eccentric MVC. The omnibus ANOVA revealed a significant time main effect for muscle activation during eccentric MVC, $F(2, 44)=4.423$, $p<0.05$, partial $\eta^2= 0.167$. There was a significant decrease in muscle activation during eccentric MVC over time pooled across groups. There was no significant interaction, $F(4, 44)=0.524$, $p=0.719$, partial $\eta^2= 0.045$, or group main effect, $F(2, 22)=0.211$, $p=0.811$, partial $\eta^2= 0.019$, for muscle activation during eccentric MVC (refer to table 3.4).

3.2.8 EMG Muscle Activity - Concentric MVC

There were no baseline differences between groups to report for muscle activation (RMS amplitude normalized to peak isometric MVC at baseline) during concentric MVC. The omnibus ANOVA revealed a significant time main effect for muscle activation during concentric MVC, $F(2, 44)=7.374$, $p<0.01$, partial $\eta^2= 0.251$. There was a significant decrease in muscle activation during concentric MVC over time pooled across groups. There was no significant interaction, $F(4, 44)=0.778$, $p=0.546$, partial $\eta^2= 0.066$, or group main effect, $F(2, 22)=0.463$, $p=0.635$, partial $\eta^2= 0.040$, for muscle activation during concentric MVC (refer to table 3.4).

3.4.9 EMG Muscle Activity - Velocity Dependent Contractions

There were no baseline differences between groups to report for muscle activation (RMS amplitude normalized to peak isometric MVC at baseline) during velocity dependent contractions. The omnibus ANOVA revealed a significant time main effect for muscle activation during max velocity dependent contractions, $F(2, 44)=6.893$, $p<0.01$,

partial η^2= 0.239. There was a significant decrease in muscle activation during velocity dependent contractions over time pooled across groups. There was no significant interaction, $F(4, 44)=0.462$, $p=0.763$, partial η^2= 0.040, or group main effect, $F(2, 22)=0.089$, $p=0.915$, partial η^2= 0.008, for muscle activation during maximal velocity dependent contractions (refer to table 3.4).

Table 3.4 Normalized EMG for strength measures over time

	Ecc MVC (30°/s)			Con MVC (60°/s)			Velocity-Dependent		
Group	***Pre***	***Mid***	***Post***	***Pre***	***Mid***	***Post***	***Pre***	***Mid***	***Post***
F	1.2 ± 0.3	1.2 ± 0.4	1.0 ± 0.2^	1.0 ± 0.3	1.0 ± 0.3	0.9 ± 0.2*	1.2 ± 0.3	1.2 ± 0.4	1.1 ± 0.3*
SB	1.2 ± 0.3	1.1 ± 0.3	1.1 ± 0.2^	1.0 ± 0.2	1.0 ± 0.2	0.9 ± 0.1*	1.2 ± 0.4	1.1 ± 0.2	1.0 ± 0.1*
C	1.3 ± 0.4	1.1 ± 0.2	1.1 ± 0.2^	1.1 ± 0.3	1.1 ± 0.3	0.9 ± 0.2*	1.2 ± 0.3	1.2 ± 0.2	1.0 ± 0.1*

* Sig. main effect of time pooled across groups ($p<0.01$).
^ Sig. main effect of time pooled across groups ($p<0.05$).
Data listed as means ± standard deviation in arbitrary units.

Chapter 4: Discussion

The purpose of this study was to examine the muscle physiological responses to eccentric strength training after first priming the muscles with either a period of static flexibility training or a single intense bout of eccentric exercise performed weeks earlier; and compare these to the responses from eccentric strength training when no prior intervention was administered. The primary hypothesis was that both muscle priming methods would offer a greater protective effect during the acute stage of eccentric strength training compared to the control group. The groups that received a muscle priming intervention were predicted to exhibit less muscle soreness and smaller decreases to isometric strength and maximal power output compared to the control group in the first 2 weeks of eccentric-strength training. The main finding was that during the acute stages of eccentric strength-training, the two groups that received a priming intervention exhibited significantly reduced acute muscle soreness (Figure 3.1 and 3.2), along with evidence that the flexibility group better maintained maximal power output and isometric MVC (Figure 3.3 and 3.4) and offered enhanced protection against muscle soreness compared to both groups (Figure 3.1 and 3.2). This finding partially confirms our primary hypothesis and offers new insight into the differences between both priming modalities.

Our secondary hypothesis was that the enhanced protective effect realized by the muscle priming groups during the acute stages of eccentric strength-training would facilitate greater training induced increases in muscle strength, hypertrophy, muscle activation and maximal power output, and shift the optimal angle to a longer muscle length following the completion of the period of eccentric strength-training, compared to

the control group. Contrary to the secondary hypothesis, any protective effects realized by both priming interventions offered no greater increases in muscle strength, hypertrophy, muscle activation, or maximal power and no differences in optimal angle were detected compared to receiving no intervention prior to eccentric training. The lack of discrepancy amongst groups upon completion of eccentric strength training was not anticipated.

Upon review of current literature, this study appears to be the first objective investigation to compare static flexibility training and a single intense bout of eccentric exercise as muscle priming interventions subsequent to chronic eccentric training, and to compare the protective effects of the two. Therefore, the results are novel with regards to current research in the area of protecting against EIMD, eccentric exercise training, the repeated bout effect and static flexibility training. Due to the unique nature of the design, it is challenging to directly link the findings with previous literature; however, the results support previous theories surrounding the protective nature of the repeated bout effect and static flexibility training prior to subsequent eccentric training and their known ability to help reduce muscle soreness and functional decline during the acute stages of eccentric exercises (Chen et al., 2011; McHugh et al., 1999b; McHugh, 2003).

The present study is also the first to examine repeated bouts of eccentric exercise following static flexibility training. Although static flexibility training appeared to provide greater protection against eccentric EIMD in the acute stages of eccentric training, the data does not support any further performance benefits after eccentric strength training as evidence of a muscle priming effect. One possibility is that the stretching period was either too short, or did not involve enough training volume. Since

previous studies typically only observe a single acute bout of eccentric exercise following flexibility training (Chen et al., 2011), it is difficult to conclude whether the shorter period of static flexibility training was insufficient to induce lasting differences between groups following the completion of the eccentric strength training phase. Chen et al. (2011) showed that both static stretch and PNF training shifted the optimum angle of the knee flexors to a longer muscle length and this shift in optimal angle prior to an acute bout of maximal eccentric exercise was effective in attenuating eccentric EIMD compared to a control group. Similar to Chen et al. (2011), the present study used a total time under tension of 15 minutes per leg during each static flexibility training session; however, the participants of Chen et al. (2011) performed static flexibility training 3 times per week for 8 weeks, compared to the present study where static flexibility training was 3 times per week for only 4 weeks. The shorter training period could be theorized as one of the reasons for the lack of difference observed between groups following the eccentric strength training phase, but this is unlikely because there were significant increases in straight leg hip flexion for the flexibility group (~20°; Figure 3.7) similar to that which Chen et al. (2010) reported during their study (~24°), despite having four fewer weeks of static flexibility training. Secondly, previous studies that have used a shorter duration stretching period still reported significant increases in ROM of the knee flexors (Worrell et al., 1994; Handel et al., 1997; Kokkonen et al., 2007). For example, Worrell et al. (1994) reported that static stretching increased ROM by 8°-10° after only 3 weeks (~1 $min \cdot d^{-1}$, 5 $d \cdot wk^{-1}$). The aforementioned evidence indicates that the results following eccentric training were unlikely to be related to a lower volume of flexibility

training. More likely the benefits of flexibility training prior to eccentric training are only realized in the acute phase of training within the first 2 weeks.

Following the first bout of eccentric exercise during the eccentric strength-training phase, both the flexibility and single-bout group reported significantly lower muscle soreness values compared to the control group (Figure 3.1). This finding confirms previous research surrounding the repeated bout effect (Clarkson & Hubal, 2002; Nosaka & Clarkson, 1995) and that chronic stretching prior to a maximal bout of eccentric exercise attenuates EIMD that can lead to muscle soreness (Chen et al., 2011; McHugh et al., 1999b). A shift in optimum angle of the knee flexor muscles to a longer muscle length following static flexibility training has been correlated to reductions in muscle soreness following an acute eccentric exercise bout (Chen et al., 2011). Interestingly, a single acute bout of eccentric exercise is sufficient enough to create a similar protective effect to that which is observed with flexibility training; indicated by a long-lasting shift in the muscle's optimum length for a contraction (Brockett et al., 2001). The current project is the first study to directly compare the two priming methods and show a similarity in the protective effects (reduced soreness) following a bout of eccentric exercise. Although there was no significant difference in optimal angle following the priming intervention, a shift towards a longer muscle length was evident for both the flexibility (~5°) and the single-bout group (~4°) while the control group experienced relatively no change (+0.7°; Figure 3.8). Following 6 weeks of static stretch training, Ferreira et al. (2007) reported a shift of the optimum angle of the knee flexors to a longer length (~4°), while Chen et al. (2011) reported ~10° shift in optimal angle following 8 weeks of flexibility training. The present study also showed no differences in optimal

angle between groups after the eccentric training phase, largely because the control group showed an optimal angle increase to a longer muscle length similar to what the other groups experienced after the priming phase. Therefore, it appears the reductions in acute soreness (protective effects) early in training for the intervention groups did not translate into any differences in post training outcomes.

A possibility that could explain why there was no difference between groups for isometric strength could be the joint position for which isometric MVC was measured. A standard knee angle of 30° of knee flexion was used to measure isometric MVC throughout the study. If the optimal angle had shifted to a longer muscle length following the priming phase for both the single-bout and flexibility group, then the angle at which isometric peak torque would be found would have shifted as well. It is well known that both chronic flexibility training and acute bouts of eccentric exercise shift the optimum angle of the knee flexors to a longer muscle length (Chen et al., 2011; Brockett et al., 2001). With the shift in optimal angle, a standardized isometric testing angle across the duration of the study could have missed significant changes in isometric MVC both during and post eccentric exercise training because the testing angle was sub-optimal. No adjustments were made to the isometric testing angle in relation to the observed changes in optimal angle, in part due to time constraints with an already large number of measures. With regards to the current data, the reduced muscle soreness following the first bout of eccentric exercise (Figure 3.1) could be attributed to a shift in optimal angle towards a longer muscle length (Figure 3.8) during the priming intervention but lack of statistical evidence makes this theory difficult to support. This remains an important point for future research.

Static flexibility training appeared to aid in attenuating the changes in the overall maximal power of the knee flexors during the first week of eccentric strength training compared to both the single-bout and control group (Figure 3.3). This is consistent with the results of Kokkenen et al. (2007) who reported increases in power following 10 weeks of static flexibility training using a variety of static stretching exercises for all the major lower-extremity muscle groups. They speculated that the improved power values following flexibility training were strongly related to the observed improvement in eccentric and concentric strength. Likewise, improving muscle strength with several sessions of static flexibility training is not a novel finding. Worrell et al. (1994) reported increases in maximal voluntary eccentric and concentric torque following just several days of static stretch training. Our current data provides evidence for increases in isometric, eccentric and concentric MVC, as well as maximal power following eccentric training when values were pooled across groups for each dependent variable (Table 3.2 and 3.3), but there were no differences between groups. Of note for future research, the flexibility group showed non-significant increases in both eccentric (~6Nm) and concentric (~5Nm) torque (Table 3.2), as well as maximal power (~10W) (Table 3.3) following static flexibility training, and prior to eccentric training. It has been speculated that increases in muscle length can lead to increases in the forces produced at a given shortening velocity (Kokkenen et al., 2007). The present study cannot conclude whether changes in overall strength and maximal power were related to the observed attenuation of changes in maximal power during the first week of eccentric strength training (Figure 3.3). Future research may use this unique finding regarding the advantages static flexibility training have over a single-bout of eccentric exercise as a springboard for

further investigation into acute differences between these two priming mechanisms and to determine the repeatability and reliability of these findings.

Our current study found muscle thickness not to be significantly different between groups following the muscle-priming phase, but there was a significant increase in muscle hypertrophy after eccentric training pooled across groups ($p<0.001$; Figure 3.5). There is some speculation in the literature that increases in strength following flexibility training could be the result of muscle hypertrophy (Kokkenen et al., 2007). The alteration in the connective tissue has also been linked to increased muscle damage resulting from intense bouts of acute static stretching (Smith et al., 1994). Passive stretching has been shown to increase creatine kinase enzyme activity, a marker of muscle damage during exercise, by 62% after approximately 20 minutes of static stretching (Smith et al., 1993). Similar to eccentric training, acute stretching has also been shown to stimulate protein synthesis in human skeletal muscle (Fowles et al., 2000). Fowles et al. (2000) indicated a 28% increase in fractional protein synthesis rate in a passively stretched limb versus a control limb, although the findings were not statistically significant. In an animal study involving stretching of the soleus muscles of rats, Stauber et al. (1994) found muscle mass to be increased by 13% and fiber area by 30%. Similarly, Coutinho et al. (2004) reported a 16% increase in rat soleus fiber area following chronic stretching of the muscle. Regrettably, no measures of muscle damage markers, such as creatine kinase, were taken while participants performed static flexibility training in our study, so it is impossible to conclude that the passive stretching technique used was intense enough to induce muscle damage. The low sample size made it difficult to detect any small differences in muscle hypertrophy between groups. Even with an effect size of partial $\eta^2=$

0.16 for the group x time interaction, observed power was just 55%. Future research examining direct measures of changes in hypertrophy and muscle damage will help in uncovering whether cellular changes are potential mechanisms for the protective effects produced by these two priming methods.

Repeated eccentric exercise bouts have been shown to increase ROM and shift the optimal angle to a longer muscle length similar to those changes associated with chronic flexibility training (Nelson & Bandy, 2004; Potier et al., 2009). When directly comparing the effects that static stretching and eccentric training have on the flexibility of the knee flexors muscle group, Nelson and Bandy (2004) reported that both types of training realized larger increases in ROM when compared to the control group. Potier et al. (2009) also studied the knee flexors and after a training period found that the eccentric trained group had greater increases in ROM, with fascicle length changes twice as large as the change reported in non-exercise control group. In the current study, with all groups training at the same volume and intensity during the eccentric strength training phase, one might conclude that any distinct advantages that either priming intervention had over control in the acute phase of training were insignificant upon completion of 4 weeks of training. Arguably, the control group experienced a repeated bout effect following the first eccentric exercise session; evident by the noticeably reduced muscle soreness during the second week of training (Figure 3.2). The control group also experienced a significant increase in passive ROM after eccentric training (Figure 3.7) comparable to the two previously aforementioned studies. There appears to be a strong correlation in previous literature concerning shifts in optimal angle and joint ROM (Chen et al., 2011), alluding to the possibility that the control group was exposed to the same attenuating effects of

repeated eccentric exercise after an initial bout of eccentric exercise as that of the single-bout group. The control group finished with identical optimal angle values as the flexibility group after eccentric training at 17.7° ± 4.3°, and a similar optimal angle muscle length to the single-bout group (18.5° ± 5.4°; Figure 3.8). This suggests that several repeated bouts of eccentric exercise occurring within the first week of a progressive training program induces protective effects similar to prior static flexibility training or a single bout of damaging exercise performed weeks earlier, and yield similar outcomes at the end of subsequent eccentric training. This theory cannot be supported by previous literature, as this is the first study to directly look at the changes in optimal angle using various priming methods and its effect on subsequent bouts of eccentric exercise.

A somewhat puzzling finding is that normalized EMG activation amplitude (normalized to isometric MVC) during the eccentric and concentric MVC actually decreased significantly after eccentric training (Table 3.4). Increases in muscle strength are commonly attributed to increased neural drive to agonist muscles, as measured by EMG activity (Gabriel et al., 2006). The majority of eccentric and concentric training studies report increased EMG activation after training (Hortobagyi et al., 1997; Carvahlo et al., 2014; Aagaard et al., 2000), but not all studies show this (Krentz et al., 2010; Seger & Thorstensson et al., 2005).

It is difficult to determine the reasoning behind the significant decrease in EMG activity following eccentric training based on the statistical evidence in the current study. It might be speculated that the isotonic training method used during the eccentric training phase did not reflect the isokinetic testing measures used for eccentric and concentric

MVC. Previous research has suggested that there is a strong correlation between strength gains and the specificity of the training mode (Tomberlin et al., 1991; Duncan et al., 1989). Another possibility for the significant decrease in EMG activity could be accredited to measurement error and methodological limitations. Seger and Thorstensson (2005) indicated that performing a comparison of absolute EMG levels before and after a training period requires identical recording conditions, which is very difficult to accomplish, especially without normalizing to the maximal compound action potential (M-wave). Normalizing EMG data to the M-wave, instead of normalizing to the isometric MVC at a fixed joint angle, may have helped clarify the confusing EMG amplitude data because M-Wave is a more stable measure when interpreting maximal muscle activity during dynamic contractions. Regardless of methodology, it is important to note that previous studies of EMG amplitude using voluntary contractions (Heckathorne & Childress, 1981) and evoked responses (Garland et al., 1994) have demonstrated that even when motor unit activity is held constant, changes in muscle length affect the EMG amplitude. Worrell et al. (2001) recommends using multiple normalizing isometric contractions performed at various points in the range of motion when testing dynamic muscle contractions. While there are some limitations related to the interpretation of the surface EMG amplitude and frequency data (Farina et al., 2010), the aforementioned explanations are purely speculative in nature and it remains highly perplexing as to the reason for the significant decrease in EMG muscle activity following eccentric strength training as it does not coincide with previous research (Hortobagyi et al., 1997; Carvahlo et al., 2014; Aagaard et al., 2000).

The mechanisms by which flexibility training and a single-bout of eccentric exercise attenuate the damaging effects of subsequent eccentric exercise are still unclear. One of the most common mechanistic theories emerging in both repeated bout effect and chronic flexibility literature is that which credits the increase in the number of sarcomeres in series for protecting the muscle against eccentric EIMD (Brockett et al., 2001; Proske & Morgan, 2001; McHugh et al., 1999b; Chen et al., 2011). Animal studies have stated that static stretching throughout a 3 week period of the latissimus dorsi or soleus muscles resulted in a 4%–25% increase in the number of sarcomeres in series (Coutinho et al., 2004; Cox et al., 2000). The theory is that the tension experienced by the muscle during intense eccentric contractions is decreased when the number of sarcomeres in series is increased. This adaptation is thought to lessen the severity of muscle damage occurred during the contractions (McHugh et al., 1999b). As previously stated, a shift of the optimum angle to a longer muscle length and the number of sarcomeres in series are thought to be directly related (Proske & Morgan, 2001). In the present study, the optimal angle of the knee flexors after static flexibility training indeed shifted to a longer muscle length, but this was not significantly different than the other two groups (Figure 3.8). When groups were pooled across time, a shift towards a longer muscle length following eccentric training provides some indirect evidence of an increase in the number of sarcomeres; although this was not directly measured in this study. Further studies are necessary to specifically investigate changes in sarcomere number in series after flexibility and/or eccentric exercise in humans and how this affects changes in optimal angle after subsequent eccentric strength training. An alternate theory, previously suggested by McHugh et al. (1999b), is that compliant hamstrings are less susceptible to

muscle damage after eccentric exercise. They hypothesized that the tendon–aponeurosis complex of less stiff or more flexible muscles can absorb the intense lengthening induced by eccentric exercise and limit muscle strain and damage. Unfortunately, the current project did not include any measures examining the muscle-tendon complex of the knee flexors. The current data suggests the need for further studies investigating the behavior of the muscle-tendon complex following static flexibility training and a single-bout of eccentric exercise and examine their comparable interactions with subsequent eccentric exercise training.

4.1 Limitations and Future Research

Several limitations should be considered when critically examining the study. One limitation was the small sample size. The small sample size rendered the study underpowered when performing statistical analysis on some variables (i.e. muscle hypertrophy, optimal angle). The failure to detect many significant interactions between groups could be due to few participants with each group (F: n=8; SB: n=9; & C: n=8). The estimated required sample size based on our preceding power calculations was n=12 per group. Due to the time constraints of the demanding training program, and some participant dropouts, fulfilling the required sample size proved to be a challenge. Future research using a larger sample sizes is needed to determine whether the findings of the present study can be confirmed and whether further differences between priming interventions exist.

Secondly, although there was no significant difference between groups at baseline, the unequal distribution of males and females created a notable difference in baseline values between groups, as well as the standard deviations within a group (e.g.,

see Table 3.1 and 3.2). Previous research has identified that there is a difference in flexibility between sexes (Blackburn et al., 2004) and that men and women respond differently to eccentric exercise–induced muscle damage (Sewright et al., 2008; Fredsted et al., 2008; Hubal et al., 2008). Therefore, it is necessary to either investigate the differences between males and females using a comparable experimental design, or restrict the sample to one sex or the other and conduct parallel studies.

Another limitation of the current study is that the participants were a sample of convenience of injury-free university students between 19 and 34 years of age. It is also important to note that the sample included participants who did not participate in any regular strength or sport specific training, in an attempt to help minimize confounding factors. Therefore, the current findings are most applicable to a similar demographic, and the implementation of these findings into a practical situation requires caution. Further research is needed to determine if an older population, such as elderly subjects, or a well-trained population, would realize similar protective effects from the priming interventions during the acute stages of eccentric training. Likewise, the present study only examined the effects of priming interventions using the knee flexors muscle group. Future research is needed to determine whether other muscle groups (i.e. triceps brachii, quadriceps, gastrocnemius) respond differently to eccentric training after being exposed to similar priming interventions of either flexibility training or a single eccentric exercise bout.

There were a couple limitations directly related to the collection and interpretation of muscle EMG amplitude data. As previously mentioned, not having used M-wave to normalize the MVC data proved it difficult to acquire reliable data with the knee flexors muscle group. Secondly, technical equipment challenges could have potentially led to the

uncharacteristic results in muscle activity. A malfunction in the primary four lead EMG (Bagnoli-4, Delsys Inc., Boston, MA) system approximately half way through data collection forced the use of an older 2-lead Delsys EMG system; however, the same electrodes were used. Following repair, the four lead EMG (Bagnoli-4, Delsys Inc., Boston, MA) system was once again used in data collection. This difference in EMG system may have increased variability in EMG signal amplitude.

The standardized angle at which isometric MVC was measured throughout the study might have limited the detection of important acute changes during eccentric training. As stated earlier in the discussion, with the shift in optimal angle, a standardized isometric testing angle across the duration of the study could have missed significant changes in isometric MVC both during and post eccentric exercise training because the testing angle was sub-optimal. If the optimal angle was measured consistently throughout the acute stages of eccentric training, then perhaps a more accurate angle for testing isometric MVC could have been measured. Due to time constraints and training demand, it was difficult to realistically accommodate this more challenging but potentially more accurate method of assessing acute difference between groups.

Finally, no direct measures of muscle damage were taken throughout the study. Biochemical markers of muscle damage during the acute stages of eccentric exercise could have provided further insight into differences between groups that were not observed with subjective measures of muscle soreness. Similarly, no neurological, molecular or cellular measures were taken in the study. Therefore no definitive conclusions can be made regarding the mechanisms associated with the priming intervention effects observed. Future research measuring neurological, molecular and

cellular responses is needed to determine the mechanistic reasons for the similarities observed between chronic flexibility training and eccentric strength training.

4.2 Practical Application

The results of the present study offer important new insights into the practical application of the protective effects of both chronic flexibility training and the repeated bout effect to offset the damaging consequences of eccentric exercise training. The results may be the most applicable in the realm of injury prevention and rehabilitation, specifically with regards to preventing hamstring strains in high-speed sports, like soccer and sprinting in track and field. Hamstring strains are among the most prevalent soft-tissue injuries in sports, such as Australian football and athletics (Stanton & Purdam, 1989; Seward et al., 1993). Reoccurrence of these traumatic injuries for previously injured hamstrings is approximately 16-54% (Timmins et al., 2015). Most hamstring injuries appear to occur when high-speed running is involved (Brooks et al., 2006). During this complex high-speed movement, the hamstring, specifically the biceps femoris long head, experience tremendous amounts of eccentric force and loading patterns (Askling et al., 2007). Epidemiological evidence suggests that hamstring strains occur most frequently during an eccentric contraction when the muscle is forcefully lengthened while contracting (Kujula et al., 1997). It has been proposed that the microscopic damage suffered by the muscle fibers following a period of unfamiliar eccentric exercise can potentially lead to a more severe strain injury (Brockett et al., 2001; Proske et al., 2004). Reduced muscle soreness, which is often associated with eccentric EIMD, was evident in both the chronic flexibility group and the single bout group compared to the control group in the present study. Those athletes who have optimal angles of torque at shorter

muscle lengths are most at risk for experiencing eccentric EIMD compared to those with an optimal angle at longer muscle lengths (Proske et al., 2004).

Athletes who have experienced a previous hamstring strain have been found to have significantly shorter optimal angle muscle lengths compared to uninjured hamstrings (Proske et al., 2004). Although the present study did not show significant differences in optimal angle between all three groups, changes in optimal angle occurred across all groups following eccentric exercise and all groups completed the study with similar optimal angles. Optimal angle shifts were observed following just a single bout of eccentric training similar to the flexibility group which trained three times a week for 30 minutes each session (15 $min \cdot leg^{-1}$). Other studies have shown that a single bout of eccentric exercise can shift an optimal angle by about 7° immediately after the first session of eccentric exercise (Proske et al., 2004). Eccentric training is also associated with other performance benefits, which include improvements in peak torque and jump performance (Clark et al., 2005) and reduced pain and disability (Kingma et al., 2007; Young et al., 2005).

Another practical application of the findings from the current study is the benefit that both these protective modalities possess for enhancing training programs for athletes. The adverse effects of eccentric exercise persist for approximately a week following a single bout. The uncomfortable nature of DOMS can make it difficult for athletes or general fitness enthusiasts to continue training during the acute phase following muscle damage. A phase of muscle priming would be tremendously advantageous for any athletes that experience high eccentric stresses during their sport or training. For example, a baseball pitcher requires the latissimus dorsi, posterior deltoid and infraspinatus

muscles to contract eccentrically to slow down the rapid horizontal adduction of their arm during the deceleration phase of throwing (Escamilla & Andrews, 2009). During their initial throwing sessions late in the winter, DOMS and micro-damage is experienced following these sessions. A priming intervention of eccentric exercise a few weeks earlier would help reduce adverse effects, thus allowing the athlete to maximize early training sessions.

The similarities between groups following eccentric strength training regardless of priming intervention, indicates that both methods offer similar protective advantages during the acute stages of eccentric training and similar outcomes following the completion of 4 weeks of eccentric strength training. Also, there was evidence that flexibility training offered superior protection against muscle soreness, as well as better maintenance of maximal power and isometric MVC during the acute stages of eccentric strength training compared to a single bout of eccentric exercise. With the low sample size and lack of supporting research for the benefits of chronic flexibility training, and the overwhelming amount of evidence for the benefits of eccentric exercise, no firm conclusions on the advantages of prior flexibility training can be made. Further research needs to be done to examine the intriguing benefits and applications of chronic flexibility training prior to eccentric strength training.

Based on the current results and previous literature, athletes looking to prevent hamstring muscle strains or rehabilitate previous hamstring strains or wish to prevent early eccentric EIMD during the early stages of off-season training or the competition season, a priming intervention would be extremely beneficial weeks prior, should consider eccentric exercise as their preferred mode of training. High-level athletes have

extremely demanding schedules throughout a season and maximizing gains in the least amount of time is favored. As evident in preceding literature, and by the similarity in training responses between priming interventions in the current study, it is recommended that even though a single bout of eccentric exercise is not as effective as chronic flexibility training for improving joint ROM and reducing acute soreness, the other advantages of eccentric training suggest that including an eccentric component to every athletic strength and conditioning program is of fundamental importance.

Chapter 5: Summary and Conclusion

5.1 Summary

The present study examined the muscle physiological responses to eccentric strength training after first priming the knee flexor muscles with either a period of static flexibility training or a single intense bout of eccentric exercise performed weeks earlier; and compared these to the responses from eccentric strength training when no prior intervention was administered. During the acute stages of eccentric training, the two groups that received a priming intervention exhibited significantly reduced acute muscle soreness; along with evidence that the flexibility group better maintained maximal power output and isometric MVC and offered enhanced protection against muscle soreness compared to both groups. These protective benefits of the priming intervention during the acute stages did not translate into enhanced performance benefits following the completion of an eccentric training program. The groups that performed the priming interventions experienced no greater increases in muscle strength, hypertrophy, muscle activation or maximal power and no differences in optimal angle were evident compared to the groups that received no intervention prior to eccentric training, despite the fact that both priming interventions offered significant reductions in muscle soreness during the initial stages of eccentric strength training. This study appears to be the first objective investigation to compare static flexibility training and a single intense bout of eccentric exercise as muscle priming interventions subsequent to chronic eccentric training. Therefore, the results are novel with regards to current research in the area of protecting

against EIMD, eccentric exercise training, the repeated bout effect and static flexibility training.

5.2 Conclusion

It appears that any indicators of performance benefit or protection against eccentric EIMD incurred by the priming interventions are washed out following a 4-week period of eccentric strength training. The protective effect conferred by the flexibility training and eccentric exercise and the similarities observed across groups following eccentric strength training, is likely to be associated with the addition of sarcomeres and/or changes in tendon compliance, but these theories should be investigated further as neither were directly measured in this study. Future studies that assess the potential shared mechanisms of the protective effects of flexibility training and the repeated bout effect are warranted. The results of the current study suggest that the flexibility training provides similar effects to that observed with the repeated bout effect; however, the apparent lack of differences at the end of eccentric training, regardless of priming intervention, raises interesting questions about the practical utility that these priming methods have on an eccentric exercise based training program. The potential applications of using priming methods to prevent muscle damage during the acute stages of eccentric training or exercise could be used by athletes and general population to help attenuate early performance impairments. Rehabilitative and injury prevention application could potentially be utilized by exercise and health professionals as an essential component to their training methods but further field testing and research in the area of injury prevention is required.

References

1. Aagaard, P., Simonsen, E. B., Andersen, J. L., Magnusson, S. P., Halkjaer-Kristensen, J., Dyhre-Poulsen, P. (1985). Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. *Journal of Applied Physiology*, *89*, 2249-2257.
2. Aldayel, A., Jubeau, M., McGuigan, M. R., Nosaka, K. (2010). Less indication of muscle damage in the second than initial electrical muscle stimulation bout consisting of isometric contractions of the knee extensors. *European Journal of Applied Physiology*, *108*, 709-717.
3. Antonio, J., Gonyea, W. J. (1993). Progressive stretch overload of skeletal muscle results in hypertrophy before hyperplasia. *Journal of Applied Physiology*, *75*, 1263-1271.
4. Armstrong, R. B. (1990). Initial events in exercise-induced muscular injury. *Medicine and Science in Sports and Exercise*, *22*, 429-435.
5. Armstrong, R. B., Warren, G. L., Warren, J. A. (1991). Mechanisms of exercise-induced muscle fiber injury. *Sports Medicine, 12*, 184-207.
6. Askling, C. M., Tengvar, M., Saartok, T., Thorstensson, A. (2007). Acute first-time hamstring strains during slow-speed stretch: clinical, magnetic resonance imaging, and recovery characteristics. *American Journal of Sports Medicine, 35*, 1716-1724.
7. Ayala, F., Sainz de Baranda, P., De Ste Croix, M., Santonja, F. (2011). Criterion related validity of four clinical tests used to measure hamstring flexibility in futsal

players. *Physical Therapy in Sport*, *12*, 175-181.

8. Bandy, W. D., Irion, J. M. (1994). The effect of time on static stretch on the flexibility of the hamstring muscles. *Physical Therapy*, *74*, 845-850.

9. Barash, I. A., Peters, D., Friden, J., Lutz, G. L., Lieber, R. L. (2002). Desmin cytoskeletal modifications after a bout of eccentric exercise in a rat. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, *283*, R958-R963.

10. Barss, T., Magnus, C., Clarke, N., Lanovaz, J., Chilibeck, P., Kontulainen, S., Arnold, B., Farthing, J. (2014). Velocity-specific strength recovery after a second bout of eccentric exercise. *Journal of Strength and Conditioning Research*, *28*, 339-349.

11. Black, C. D., McCully, K. K. (2008). Muscle injury after repeated bouts of voluntary and electrically stimulated exercise. *Medicine and Science in Sports and Exercise*, *40*, 1605-1605.

12. Blackburn, J. T., Reimann, B. L., Padua, D. A., Guskiewicz, K. M. (2004). Sex comparison of extensibility, passive, and active stiffness of the knee flexors. *Clinical Biomechanics*, *19*, 36-43.

13. Brockett, C. L., Morgan, D. L., Proske, U. (2001). Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Medicine and Science in Sports and Exercise*, *33*, 783-790.

14. Brooks, J. H. M., Fuller, C. W., Kemp, S. P. T., Reddin, D. B. (2006). Incidence, risk, and prevention of hamstring muscle injuries in professional rugby union. *The American Journal of Sports Medicine*, *34*, 1297-1306.

15. Carrasco, D. I., Delp, M. D., Ray, C. A. (1999). Effect of concentric and eccentric muscle actions on muscle sympathetic nerve activity. *Journal of Applied Physiology*, *86*, 558-563.
16. Carvalho, A., Caserotti, P., Carvalho, C., Adabe, E., Sampaio, J. (2014). Effect of a short time concentric versus eccentric training program on electromyography activity and peak torque of quadriceps. *Journal of Human Kinetics*, *41*, 5-13.
17. Chapman, D., Newton, M., Sacco, P., & Nosaka, K. (2006). Greater muscle damage induced by fast versus slow velocity eccentric exercise. *International Journal of Sports Medicine*, *27*, 591-598.
18. Chen, C. H., Nosaka, K., Chen, H.L., Lin, M.J., Tseng, K.W., Chen T.C. (2011). Effects of flexibility training on eccentric exercise-induced muscle damage. *Medicine and Science in Sports and Exercise*, *43*, 491-500.
19. Cheung, K., Hume, O., Maxwell, L. (2003). Delayed onset muscle soreness: treatment strategies and performance factors. *Sport Medicine*, *33*, 145-164.
20. Clark, R., Bryant, A., Culgan, J., Hartley, B. (2005). The effects of eccentric hamstring strength training on dynamic jumping performance and isokinetic strength parameters: a pilot study on the implications for the prevention of hamstring injuries. *Physical Therapy in Sport*, *6*, 67-73.
21. Clarkson, P. M., Tremblay, I. (1988). Exercise-induced, muscle damage, repair, and adaptation in humans. *Journal of Applied Physiology*, *65*,1–6.
22. Clarkson, P. M., Nosaka, K., Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine and Science in Sports and Exercise*, *24*, 512-520.

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

24. Coutinho, E. L., Gomes, A. R., Franca, C. N., Oishi, J., Salvini, T.F. (2004). Effect of passive stretching on the immobilized soleus muscle fiber morphology. *Brazilian Journal of Medical and Biological Research, 37,* 1853-1861.

25. Cox, V. M., Williams, P. E., Wright, H., James, R. S., Gillott, K. L., Young, I. S., Goldspink, D. F. (2000). Growth induced by incremental static stretch in adult rabbit lattissimus dorsi muscle. *Experimental Physiology, 85,* 193-202.

26. Dartnall, T. J., Nordstrom, M. A., Semmler, J. G. (2011). Adaptations in biceps brachii motor unit activity after repeated bouts of eccentric exercise in elbow flexor muscles. *Journal of Neurophysiology, 105,* 1225-1235.

27. DeVries, H.A. (1966). Quantitative electromyographic investigation of the spasm theory of muscle pain. *American Journal of Physical Medicine and Rehabilitation, 45,* 119-134.

28. Dintman, G. B. (1964). Effects of various training programs on running speed. *Research Quaterly, 35,* 456-463.

29. Dixon, J. K., Keating, J. L. (2000). Variability in straight leg raise measurements: review. *Physiotherapy, 86,* 361-370.

30. Duncan, P. W. Chandler, J. M., Cavanaugh, D. K. Johnson, K. R., Buehler, A. G. (1989). Mode and speed specificity of eccentric and concentric exercise training. *The Journal of Orthopaedic and Sports Physical Therapy, 11,* 70-75.

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

32. Escamilla, R. F., Andrews, J. R. (2009). Shoulder muscle recruitment patterns and related biomechanics during upper extremity sports. *Sports Medicine, 39,* 569-590.

33. Eston, R. G., Lemmey, A. B., McHugh, P., Byrne, C., Walsh, S. E. (2000). Effect of stride length on symptoms of exercise-induced muscle damage during a repeated bout of downhill running. *The Scandinavian Journal of Medicine and Science in Sports, 10,* 199-204.

34. Eston, R. G., Rowlands, A. V., Coulton, D., McKinney, J., Gleeson, N. P. (2007). Effect of flexibility training on symptoms of exercise-induced muscle damage; a preliminary study. *Journal of Exercise Science and Fitness, 5,* 33-39.

35. Farina, D., Holobar, A., Merletti, R. Enoka, R. M. (2010). Decoding the neural drive to muscles from the surface electromyogram. *Clinical Neurophysiology, 121,* 1616-1623.

36. Farthing, J. P., & Chilibeck, P. D. (2003). The effects of eccentric and concentric training at different velocities on muscle hypertrophy. *European Journal of Applied Physiology, 89,* 578-586.

37. Faul, F., Erdfelder, E., Buchner, A., Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. *Behavior Research Methods, 41,* 1149-1160.

38. Ferreira, G. N. T., Teixeira-Salmela, L. F., Guimarães, C. Q. (2007). Gains in flexibility related to measures of muscular performance: impact of flexibility on muscular performance. *Clinical Journal of Sport Medicine, 17,* 276-281.

39. Fowles, J. R., Sale, D. G., MacDougall, J. D. (2000). Reduced strength after

passive stretch of the human plantarflexors. *Journal of Applied Physiology, 89,*1179–1188.

40. Fredsted, A., Clausen, T., Overgaard, K. (2008). Effects of step exercise on muscle damage and muscle Ca2+ content in men and women. *Journal of Strength and Conditioning Research, 22,* 1136–1146.
41. Gabriel, D. A., Kamen, G., Frost, G. (2006). Neural adaptations to resistive exercise: Mechanisms are recommendation for training practices. *Journal of Sports Medicine, 36,* 133-149.
42. Gajdosik, R. L. (1991). Passive compliance and length of clinically short hamstring muscles of healthy men. *Clinical Biomechanics, 6,* 239-244.
43. Garland, S. J., Gerilovsky, L., Enoka, R. M. (1994). Association between muscle architecture and quadriceps femoris H-reflex. *Muscle & Nerve, 17,* 581-592.
44. Godges, J. J., MacRae, H., Longdon, C., Tinberg, C., MacRae, P. (1989). The effects of two stretching procedures on hip range of motion and gait economy. *Journal of Orthopaedic and Sports Physical Therapy, 10,* 350-357.
45. Golden, C. L., Dudley, G. A. (1992). Strength after bouts of eccentric or concentric actions. *Medicine and Science in Sports and Exercise, 24,* 926-933.
46. Handel, M., Horstmann, T., Dickhuth, H. H., Gulch, R. W. (1997). Effects of contract-relax stretching training on muscle performance in athletes. *European Journal of Applied Physiology and Occupational Physiology, 76,* 400-408.
47. Heckathorne, C. W., Childress, D. S. (1981). Relationships of the surface electromyogram to the force, length, velocity, and contraction rate of the cineplastic human biceps. *American Journal of Physical Medicine, 60,* 1-19.

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

49. Hollander, D.B., Kraemer, R. R., Kilpatrick, M. W., Ramadan, Z. G., Reeves, G. V., Francois, M., et al. (2007). Maximal eccentric and concentric strength discrepancies between young men and women for dynamic resistance exercise. *Journal of Strength and Conditioning Research, 21,* 34-40.

50. Hortobágyi, T., Faludi, J., Tihanyi, J., Merkely, B. (1985). Effects of intense stretching-flexibility training on the mechanical profile of the knee extensors and on the range of motion of the hip joint. *International Journal of Sport Medicine, 6,* 317-321.

51. Hortobágyi, T., Barrier, J., Beard, D., Braspennincx, K., Koens, P., Devita, P., Dempsey, L., Lambert, J. (1996a). Greater initial adaptations to sub-maximal muscle lengthening than maximal shortening. *Journal of Applied Physiology, 81,* 1677-1682.

52. Hortobágyi, T., Hill, J. P., Houmard, J. A., Fraser, D. D., Lambert, N. J., Israel, R.G. (1996b). Adaptive responses to muscle lengthening and shortening in humans. *Journal of Applied Physiology, 80,* 765-772.

53. Hortobágyi, T., Lambert, N. J., Hill, J. P. (1997). Greater cross education following training with muscle lengthening than shortening. *Medicine and Science in Sports and Exercise, 29,* 107-112.

54. Hortobágyi, T., Houmard, J., Fraser, D., Dudek, R., Lambert, J., Tracy, J. (1998). Normal forces and myofibrillar disruption after repeated eccentric exercise.

Journal of Applied Physiology, *84*,492–498.

55. Hough, T. (1902). Ergographic studies in muscular soreness. *American Journal of Physiology*, *7*, 76–92.
56. Hubal, M. J., Rubinstein, S. R., Clarkson, P. M. (2008). Muscle function in men and women during maximal eccentric exercise. *Journal of Strength and Conditioning Research, 22*, 1332–1338.
57. Hunter, J. P., Marshall, R. N. (2002). Effects of power and flexibility training on vertical jump technique. *Medicine and Science in Sports and Exercise*, *34*, 478-486.
58. Hydahl, R. D., Hubal, M. J. (2014). Lengthening our perspective: morphological, cellular, and molecular responses to eccentric exercise. *Muscle Nerve, 49*, 155-170.
59. Inman, V. T., Ralston, H. J., Todd, F. (1981). *Human walking*. Baltimore, Md: William & Wilkins, 89-100.
60. Isner-Horobeti, M. E., Dufour, S. P., Vautravers, P., Geny, B., Coudeyre, E., Richard, R. (2013). Eccentric exercise training: modalities, applications and perspectives. *Sports Medicine, 43*, 483-512.
61. Johansson, P. H., Lindström, L., Sundelin, G., Lindström, B. (1999). The effects of preexercise stretching on muscular soreness, tenderness and force loss following heavy eccentric exercise. *Scandinavian Journal of Medicine and Science in Sports*, *9*, 219-225.
62. Kilgallon, M., Donnelly, A. E., Shafat, A. (2007). Progressive resistance training temporarily alters hamstring torque-angle relationship. *Scandinavian Journal of*

Medicine and Science in Sports, *17*, 18-24.

63. Kingma, J. J., Knikker, R., Wittink, H. M., Takken, T. (2007). Eccentric overload training in patients with chronic Achilles tendiopathy: a systematic review. *British Journal of Sports Medicine*, *41*, e3.
64. Kokkonen, J., Nelson A. G., Cornwell, A. (1998). Acute muscle stretching inhibits maximal strength performance. *Research Quarterly for Exercise and Sport*, *69*, 411–415.
65. Kokkonen, J., Nelson, A. G., Eldredge, C., Winchester, J. B. (2007). Chronic static stretching improves exercise performance. *Medicine and Science in Sports and Exercise*, *39*, 1825-1831.
66. Komi, P.V. (1984). Physiological and biomechanical correlates of muscle function: effects of muscle structure and stretch-shortening cycle on force and speed. *Exercise and Sport Sciences Reviews*, *12*, 81-121.
67. Kraemer, R., Hollander, D., Reeves, G., Francois, R., Ramadan, Z., Meeker, B., et al. (2006). Similar hormonal responses to concentric and eccentric muscle actions using relative loading. *European Journal of Applied Physiology*, *96*, 551-557.
68. Krentz, J. R., Quest, B., Farthing, J. P., Quest, D. W., Chilibeck, P. D. (2008). The effects of ibuprofen on muscle hypertrophy, strength, and soreness during resistance training. *Applied Physiology, Nutrition and Metabolism*, *33*, 460-475.
69. Krentz, J. R., Farthing, J. P. (2010). Neural and morphological changes in response to a 20-day intense eccentric training protocol. *European Journal of Applied Physiology*, *110*, 333-340.
70. Krishnan, C., Williams, G. N. (2014). Effect of knee joint angle on side-to-side

strength ratios. *Journal of Strength and Conditioning Research, 28*, 2981-2987.

71. Kujala, U. M., Orava, S., Jarvinen, M. (1997). Hamstring injuries. Current trends in treatment and prevention. *Sports Medicine, 23,* 397–404.
72. Lapier, T. K., Burton, H. W., Almon, R., Cerny, F. (1995). Alterations in intramuscular connective tissue after limb casting affect contraction-induced muscle injury. *Journal of Applied Physiology, 70,* 2498-2507.
73. LaRoche, D. P., Connolly, D. A. J. (2006). Effects of stretching on passive muscle tension and response to eccentric exercise. *American Journal of Sports Medicine, 34*, 1000-1007.
74. Leslie, A., Krentz, J., Farthing, J. (2011, March). The effects of flexibility training on muscle hypertrophy: a preliminary study. Poster session presented at the 18th annual Life and Health Sciences Research Day, Saskatoon, SK.
75. Lieber, R. L., Fridén, J. (1993). Muscle damage is not a function of muscle force but active strain. *Journal of Applied Physiology, 74*, 520-526.
76. Lima, K. M., Carneiro, S. P., Alves Dde, S., Peixinho, C. C., Oliveira, L. F. (2015). Assessment of muscle architecture of the biceps femoris and vastus lateralis by ultrasound after a chronic stretching program. *Clinical Journal of Sport Medicine, 25*, 55-60.
77. Lindstedt, S. L., Reich, T. E., Keim, P., LaStayo, P.C. (2002). Do muscles function as adaptable locomotor springs? *The Journal of Experimental Biology, 205,* 2211-2216.
78. Lynn, R., Morgan, D. L. (1994). Decline running produces more sarcomeres in rat vastus intermedius muscle fibers than does incline running. *Journal of Applied*

Physiology, *77*, 1439-1444.

79. Lynn, R., Talbot, J. A., Morgan, D. L. (1998). Difference in rat skeletal muscles after incline and decline running. *Journal of Applied Physiology*, *85I*, 98-104.
80. Mattacola, C. G., Perrin, D. H., Gansneder, B. M., Allen, J. D., & Mickey, C. A. (1997). A comparison of visual analog and graphic rating scales for assessing pain following delayed onset muscle soreness. *Journal of Sport Rehabilitation*. *6*, 38-46.
81. McGlynn, G. H., Laughlin, N. T., Rowe, B. S. (1979). Effect of electromyographic feedback and static stretching on artificially induced muscle soreness. *American Journal of Physical Medicine, 58*, 139-148.
82. McHugh, M. P., Connolly D. A., Eston, R. G., Gleim, G. W. (1999a). Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. *Sports Medicine*, *27*, 157-170.
83. McHugh, M. P., Connolly D. A., Eston, R. G., Kremenic, I. J., Gleim, G. W. (1999b). The role of passive muscle stiffness in symptoms of exercise induced muscle damage. *American Journal of Sports Medicine, 27*, 594-599.
84. McHugh, M. P., Connolly D. A., Eston, R. G., Gartman, E. J., Gleim, G. W. (2001). Electromyographic analysis of repeated bouts of eccentric exercise. *Journal of Sports Sciences*, *19*, 163-170.
85. McHugh, M. P. (2003). Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scandinavian Journal of Medicine and Science in Sports, 13*, 88-97.

86. Morgan, D. L., Allen, D. G. (1999). Early events in stretch-induced muscle damage. *Journal of Applied Physiology, 87,* 2007-2015.

87. Moritani T., Muramatsu, S., Muro, M. (1987). Activity of motor units during concentric and eccentric contractions. *American Journal Physical Medicine, 66,* 338-350.

88. Nelson, A. G., Kokkonen, J., Eldredge, C. (2005a). Strength inhibition following an acute stretch is not limited to novice stretchers. *Research Quarterly for Exercise and Sport, 76,* 500-506.

89. Nelson, A.G., Driscoll, N. M., Landin, D.K., Young, M. A., Schexnayder, I. C. (2005b). Acute effects of passive muscle stretching on sprint performance. *Journal of Sports Science, 23,* 449-454.

90. Nelson, A. G., Kokkonen, J., Arnall, D. A. (2005c). Acute muscle stretching inhibits muscle strength endurance performance. *Journal of Strength and Conditioning Research, 19,* 338-343.

91. Nelson, R. T., Bandy, W. D. (2004). Eccentric training and static stretching improve hamstring flexibility in high school males. *Journal of Athletic Training, 39,* 254-258.

92. Newham, D. J., Jones, D. A., Clarkson, P. M. (1987). Repeated high-force eccentric exercise: effects on muscle pain and damage. *Journal of Applied Physiology, 63,* 1381-1386.

93. Nosaka, K., Clarkson, P. M. (1995). Muscle damage following repeated bouts of high force eccentric exercise. *Journal of Medicine & Science in Sports & Exercise, 27,* 1263-1269.

94. Nosaka, K., Newton, M., Sacco, P. (2002). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine & Science in Sports, 12,* 337-346.

95. Nosaka, K., Sakamoto, K., Newton, M., Sacco, P. (2001). How long does the protective effect on eccentric exercise-induced muscle damage last? *Medicine and Science in Sports and Exercise, 33,* 1490-1495.

96. O'Sullivan, K., McAuliffe, S., Deburca, N. (2012). The effects of eccentric training on lower limb flexibility: a systematic review. *British Journal of Sports Medicine, 46,* 838-845.

97. Peake, J., Nosaka, K., Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans. *Exercise Immunology Review, 11,* 64-85.

98. Pincivero, D. M., Campu, R.M., Coelho, A. J. (2003). Knee flexor torque and perceived exertion: gender and reliability analysis. *Medicine and Science in Sports and Exercise, 35,* 1720-1726.

99. Potier, T. G., Alexander, C. M., Seynnes, O. R. (2009). Effects of eccentric strength training on biceps femoris muscle architecture and knee joint range of movement. *European Journal of Applied Physiology, 105,* 939-944.

100. Pousson, M., van Hoecke, J., Goubel, F. (1990). Changes in elastic characteristics of human muscle induced by eccentric exercise. *Journal of Biomechanics, 23,* 343-348.

101. Proske, U., Morgan, D. L. (2001). Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *The Journal of*

Physiology, *537*, 333-345.

102. Proske, U. Morgan, D. L., Brockett, C. L., Percival, P. (2004). Identifying athletes at risk of hamstring strains and how to protect them. *Clinical and Experimental Pharmacology and Physiology*, *31*, 546-550.

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

104. Reich, T. E., Lindstedt, S. L., Lastayo, P. C., Pierotti, D. J. (2000). Is the spring quality of muscle plastic? *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology*, *278*, R1661-R1666.

105. Sam, M., Shah, S., Fridén, J., Milner, D. J., Capetanaki, Y., Lieber, R. L. (2000). Desmin knockout muscles generate lower stress and are less vulnerable to injury compared with wild-type muscles. *American Journal of Physiology. Cell Physiology*, *279*, C1116-C1122.

106. Samuel, M. N., Holcomb, W. R., Guadagnoli, M. A., Rubley, M. D., Wallmann, H. (2008). Acute effects of static and ballistic stretching on measures of strength and power. *Journal of Strength and Conditioning Research*, *22*, 1422-1428.

107. Santonja Medina, F. M., Sainz De Baranda Andjújar, P., Rodríguez García, P. L., López Miñarro, P. A., Canteras Jordana, M. (2007). Effects of frequency of static stretching on straight-leg raise in elementary school children. *The Journal of Sports Medicine and Physical Fitness*, *47*, 304-308.

108. Seger, J. Y., Thorstensson, A. (2005). Effects of eccentric versus concentric training on thigh muscle strength and EMG. *International Journal of Sports Medicine*, *26*, 45-52.

109. Seward, H., Orchard, J., Hazard, H., Collinson, D. (1993). Football injuries in Australia at the elite level. *The Medical Journal of Australia, 159*, 298-301.

110. Sewright, K. A., Hubal, M. J., Kearns, A., Holbrook, M. T., Clarkson, P. M. (2008). Sex differences in response to maximal eccentric exercise. *Medicine and Science in Sports and Exercise*. *40*, 242–251.

111. Shrier, I. (2004). Does stretching improve performance? A systematic and critical review of the literature. *Clinical Journal of Sport Medicine, 14,* 267-273.

112. Smith, L. L., Brunetz, M. H., Chenier, T.C., McCammon, M. R., Houmard, J. A., Franklin, M. E., Israel, R. G. (1993). The effects of static and ballistic stretching on delayed-onset muscle soreness and creatine kinase. *Research Quarterly for Exercise and Sport*, *64*,103–107.

113. Smith, C. A. (1994) The warm-up procedure: to stretch of not to stretch. A brief review. *Journal of Orthopaedic and Sports Physical Therapy*, *19*, 12-17.

114. Stanton, P., Purdam, C. (1989). Hamstring injuries in sprinting: the role of eccentric exercise. *The Journal of Orthopaedic and Sports Physical Therapy*, *10*, 343-349.

115. Stauber, W. T., Miller, G. R., Grimmett, J. G., Knack, K. K. (1994). Adaptation of rat soleus muscles to 4 wk of intermittent strain. *Journal of Applied Physiology*, *77*, 58-62.

116. Timmins, R., Shield, A., Williams, M., Lorenzen, C., Opar, D. (2015). Biceps femoris long-head architecture: a reliability and retrospective injury study. *Medicine & Science in Sports & Exercise*, *47*, 905-913.

117. Tomberlin, J. P., Basford, J. R., Schwen, E. E., Orte, P. A., Scott, S. C.,

Laughman, R. K., Listrup, D. M. (1991). Comparative study of isokinetic eccentric and concentric quadriceps training. *Journal of Orthropaedic and Sports Physical Therapy, 14,* 31-36.

118. Torres, R., Francisco, P., Duarte, J. A., Cabri, J. M. H. (2013). Effect of single bout versus repeated bouts of stretching on muscle recovery following eccentric exercise. *Journal of Science and Medicine in Sport, 16,* 583-588.

119. Vogt, M., Hoppeler, H. H. (2014). Eccentric exercise mechanisms and effects when used as training regime or training adjunct. *Journal of Applied Physiology, 116,* 1446-1454.

120. Warren, G. L., Hermann, K. M., Ingalls, C. P., Masselli, M. R., Armstrong, R. B. (2000). Decreased EMG median frequency during a second bout of eccentric contractions. *Journal of Medicine & Science in Sports & Exercise, 32,* 820-829.

121. Wilson, G. J., Murphy, A. J., Pryor, J. F. (1994). Musculotendinous stiffness: its relationship to eccentric, isometric, and concentric performance. *Journal of Applied Physiology, 76,* 2714-2719.

122. Worrell, T. W., Smith, T. L., Winegardner, J. (1994). Effect of hamstring stretching on hamstring muscle performance. *Journal of Orthopaedic and Sports Physical Therapy, 20,* 154-159.

123. Worrell, T. W., Karst, G., Adamczyk, D., Moore, R. (2001). Influence of joint position on electromyographic and torque generation during maximal voluntary isometric contractions of the hamstrings and gluteus maximus muscles. *Journal of Orthopaedic & Sports Physical Therapy, 31,* 730-740.

124. Young, M. A., Cook, J. L., Purdam, C. R., Kiss, Z. S., Alfredson, H. (2005).

Eccentric decline squat protocol offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *British Journal of Sports Medicine, 39*, 102-105.

Appendices

Appendix A - Consent Form

PARTICIPANT INFORMATION AND CONSENT FORM

STUDY TITLE: Static Stretching vs. Repeated Bout Effect: Which is the better muscle "primer" for subsequent eccentric training?

PRINCIPAL INVESTIGATOR: Jonathan Farthing, Ph.D., College of Kinesiology, University of Saskatchewan.

SUB-INVESTIGATORS and/or STUDENT RESEARCHERS: Phil Chilibeck, PhD, College of Kinesiology, University of Saskatchewan; Joel Lanovaz, PhD, College of Kinesiology, University of Saskatchewan, Andrew Leslie (MSc Candidate), College of Kinesiology, University of Saskatchewan.

SPONSOR [or Funding Agency]: None

CONTACT PHONE NUMBER: Dr. Jon Farthing: 966-1068

INTRODUCTION
You are being invited to participate in a research study because we want to see whether a person's muscle gets stronger, larger and more powerful if they either first stretch for a few weeks or if they do just one heavy strength training session a few weeks before starting a consistent strength training program. We will investigate this further by examining the effects of flexibility training program or a single heavy strength training session followed by a strength-training program on your hamstring muscles.

Your participation is voluntary. It is up to you to decide whether or not you wish to take part. If you wish to participate, you will be asked to sign this form. If you do decide to take part in this study, you are still free to withdraw at any time and without giving any reasons for your decision.

If you do not wish to participate, this will not affect your employment or academic standing at the University of Saskatchewan to which you are entitled or are presently receiving. It will not affect your relationship with any of the researchers or University of Saskatchewan

Please take time to read the following information carefully. You can ask the researcher to explain any words or information that you do not clearly understand. You may ask as many questions as you need. Please feel free to discuss this with your family, friends or family physician before you decide.

WHY IS THIS STUDY BEING DONE?
The purpose of this study is to investigate the effects of either a flexibility-training program (i.e. regular stretching) or a single, heavy strength training session (weeks before regular strength training) followed by a strength training program on your hamstring muscles. Flexibility training has been shown to increase your muscle length over time, and allows you to move your joint through a greater range of motion. Recent research has shown that longer, more flexible muscles become less sore following an intense session of "eccentric" exercise. Similar to stretching, one single, intense "eccentric" training session weeks before performing another session has also been shown to reduce muscle soreness and damage. Eccentric muscle contractions are performed by trying to resist while your muscle is pulled into a lengthened or stretched position. This resisted lengthening of the muscle is what often causes the damage in your muscle and makes you feel sore. If performed regularly, you will feel less sore after training because your muscles begin to adapt and they are protected against damage. This type of training is effective for building muscle and making you stronger. For this research study, we want to investigate if flexibility training or a single, intense eccentric session completed a few weeks prior to eccentric training will aid in gaining stronger, larger, and more powerful muscles in a shorter time.

WHO CAN PARTICIPATE IN THE STUDY? (if applicable)
You are eligible to participate in this study if you are between the ages of 18 and 40, with a limited to no history of consistent resistance training (that includes eccentric hamstring curls) or flexibility training, healthy and have no reasons to refrain from maximal muscular resistance testing or flexibility.

WHAT DOES THE STUDY INVOLVE?
If you agree to be in this study the following will happen:
Muscle size and length measures, muscle strength and power measures, and flexibility measures will be completed on one day, before and after a training period. Total time for the testing will be approximately 45mins-1hr for both the flexibility group and the eccentric training only group. The single, eccentric training group will take about 1.5hrs on the first visit. You will be asked to bring athletic shorts and shirt, and athletic shoes to wear for all testing and training sessions in the laboratory.

On your first visit, you will be asked to fill out brief questionnaires about prior weight training, and flexibility training, and leg dominance. If you have done too much prior training experience (weight or flexibility) you may not be eligible to participate. Also if you're too flexible or have current injuries preventing you from training, you may not be able eligible to participate. Upon your first visit to the lab you will also be randomly assigned to one of three groups: 1) Flexibility training followed by Eccentric training; or 2) Single, eccentric strength session followed by eccentric training; or 3) Only eccentric training. The following procedures will then take place:

1. Measure the size (i.e. thickness) and length of your hamstring muscles (i.e. back of the thigh) using an ultrasound machine. A flat probe is placed on your skin with a small amount of gel, and it is harmless. We will measure muscle thickness and length using the picture on screen. A few markings will be put on your skin using a non-permanent marker to make sure the probe is in the right place.
2. Warm up on a stationary bike for five minutes.

3. Standard sit and reach test. Sitting on the ground with both legs straight out in front, you will reach forward toward of your toes, pushing a small slider tool with your hands.
4. Leg flexibility measures using a "dynamometer". Lying face up on a machine bed the experimenter will slowly help raise one leg straight up while keeping your other leg flat on the table. Your muscles will feel relaxed until you reach the end of your range of motion. The experimenter will stop when you indicate you have reached the end of your range (i.e. when minor discomfort is felt during the stretch). The dynamometer machine will measure your range of motion.
5. Hamstring strength and power tests using a dynamometer: Lying face down on the dynamometer machine bed with one leg strapped to the machine attachment, maximal muscle contractions will be performed by attempting to bend one leg at the knee joint (i.e. hamstring curl). The range of motion is set using the computer. Safety devices are positioned on the machine to prevent your leg from moving beyond the set range. Three different types of contractions will be measured: an "eccentric" contraction (muscle lengthens while you contract your hamstring muscles, and your leg will be forced to straighten even though you are trying to bend it, slow and controlled movement), a fast "concentric" contraction (muscle shortens during contraction and the knee bends when you contract the hamstring muscles; in this study it will be a fast & powerful movement) and an "isometric" contraction (knee joint does not move during contraction). Three maximal contractions for each type of contraction will be recorded, separated by one minute of rest.
6. Muscle activation. The natural electrical activity of your muscle will be measured during the strength tests by placing stickers, called electrodes, on the skin. Wires are connected to the electrodes and feed into a device that records the activity in your muscles on a computer. In order to make sure the recording signal is accurate your skin will be cleaned with alcohol, and shaved if necessary using a disposable razor.

The testing procedures described above will be completed on three occasions: on your initial visit to the lab, after 4 weeks and after 8 weeks. During the eccentric training phase, brief testing sessions (only power, strength and muscle soreness will be measured) will also be completed (all 3 groups) immediately before and after your 1st & 4th eccentric training session, with follow up testing 48hrs after these sessions. In addition, muscle size and length will be measured prior to your 7th eccentric training session for all groups.

Group 1 will complete 4 weeks of flexibility training. You will return to the university and complete flexibility training 3 times per week with a minimum of 48 hrs between sessions. Each session will begin with a 5-min warm-up on a stationary bike, followed by 15 min of stretching using a similar stretch to the partnered stretch used for testing. These stretching sessions will be group sessions done in the same motor control lab or in the fitness area at the PAC laboratory. Each stretch will be held (until you indicate that a minor discomfort is felt) using either a belt to hold your leg up or a wall for 60 sec followed by a 30 sec rest and will be repeated 10 times/leg. The training sessions will take about 30 mins to complete. These stretching sessions will be supervised and a trained professional will make sure both your form and technique are correct to decrease the risk of injuries. Both legs will be stretched to avoid imbalances between legs. During the flexibility training, group 3 (the eccentric training only) and group 2 (the single, eccentric strength session group) will not receive this treatment.

Group 2 will perform a single session of strength training a few days following these tests listed above on their first testing day in the lab. This group will perform a number of intense eccentric contractions (one training session) under supervision. The set up for the eccentric, hamstring curls will be the same as the strength and power measurements done in pre-testing. The participant will be performing 6 sets of 8 near maximal effort eccentric repetitions with 1-minute rest between sets. The training will be done on both legs to avoid any imbalances between legs. You will be briefly tested immediately before and after this single intense strength training session (only for power, strength and muscle soreness). You will then return for the same brief testing 24hrs and 48hrs after this strength session. This group will then proceed to do no other intervention for the next 4 weeks. The group 1 (flexibility group) and group 3 (the eccentric training only group) will not receive this treatment.

After the 4-week period of flexibility training is completed for the first group, all groups will participate in 4 weeks of eccentric training. Eccentric training will be done in the lab on the dynamometer machine (as for strength testing), and will be supervised. Sessions begin with a 5 min warm-up on a stationary bike. A warm-up set of 5 sub-maximal repetitions will be done prior to training to familiarize you with the procedure. Training sessions will be performed 2-3 times per week and begin with 3-4 sets of 8 repetitions, with one minute of rest between sets. This training will be progressive and more sets will be added each week (maximum of 6 sets by week 4). The eccentric training will be near maximal effort, using a load that is about 80% of your maximal effort recorded during the isometric contractions. Both legs will be trained to avoid strength imbalances. Each strength training session will take about 30 mins to complete.

WHAT ARE THE BENEFITS OF PARTICIPATING IN THIS STUDY?
You will get an assessment of your hamstrings strength, size, flexibility, power, and muscle activity while participating in the study. Depending on your assigned group you may experience increases in hamstring flexibility, strength, power, and muscle size following the training periods. However every individual is different and these benefits are not guaranteed.

ARE THERE POSSIBLE RISKS AND DISCOMFORTS?
Due to the maximal nature of the strength testing it could result in discomfort as well as muscle fatigue. After the strength testing is complete you may experience stiff or sore muscles for the next 2 or 3 days. There is a slight risk of muscle injury due to the maximal nature of the tests; however this risk will be minimized by a proper warm up (5 mins on stationary bike). Eccentric training can result in muscle soreness that persists up to one week after the start of training. After the first week of training, soreness usually goes away. In rare instances (less than 1% of participants) soreness will persist for a longer time, and in this case first aid will be administered and you will be referred to a physician if necessary.

There is a small risk of injury with flexibility tests and training, but this will be minimized by a proper warm up. Since the end range of motion for the flexibility testing and training sessions is determined by your own level of discomfort, the risk of injury is very low.

The adhesive on the electrodes might cause some discomfort to your skin, however this is a rare occurrence. If any discomfort is felt than first aid will be administered by the Certified Exercise Physiologist who will be supervising the testing sessions.

WHAT IF NEW INFORMATION BECOMES AVAILABLE THAT MAY AFFECT MY DECISION TO PARTICIPATE?
During the course of this study, if new information that may affect your willingness to continue to participant should become available it will be provided to you by the researchers.

WHAT HAPPENS IF I DECIDE TO WITHDRAW?
Your participation in this research is entirely voluntary. You may withdraw from this study at any time. If you decide to enter the study and to withdraw at any time in the future, there will be no penalty or loss of benefits to which you are otherwise entitled. Your academic standing and/or employment will not be affected.

If you choose to enter the study and then decide to withdraw later, all data collected about you during your enrolment will be retained for analysis.

WILL I BE INFORMED OF THE RESULTS OF THE STUDY?
The results of the study will be available from Dr. Jon Farthing at the completion of the study. These results will be included as part of a Master's Degree thesis and presentation. Data will be presented in aggregate form. Your name will not be included on any of the data and your identity will remain confidential. You can access the results of this study by contacting Dr. Farthing, Andrew Leslie, by reading the thesis in electronic format through the ETD (at http://library.usask.ca/etd/) and by attending the presentation of this work during the thesis defense – to be scheduled by the College of Kinesiology.

WHAT WILL THE STUDY COST ME?
You will not be charged for any research-related procedures. You will not be paid for participating in this study. You will not receive any compensation, or financial benefits for being in this study, or as a result of data obtained from research conducted under this study.

WHAT HAPPENS IF SOMETHING GOES WRONG?

In the case of a medical emergency related to the study, you should seek immediate care and, as soon as possible, and notify a doctor. Inform the medical staff you are participating in a clinical study. Necessary medical treatment will be made available at no cost to you. If any discomfort is felt during any of the testing or training procedures conducted during the study, first aid will be administered by the Certified Exercise Physiologist who will be supervising. By signing this document, you do not waive any of your legal rights.

WILL MY TAKING PART IN THIS STUDY BE KEPT CONFIDENTIAL?
Your confidentiality will be respected. No information that discloses your identity will be released or published without your specific consent to the disclosure. The testing procedures will take place in an enclosed space in the Physical Activity Complex. Your name will not be attached to any information, nor mentioned in any study report, nor be made available to anyone except the research team. It is the intention of the research team to present the findings to faculty and related workshops, but your identity will not be revealed.

WHO DO I CONTACT IF I HAVE QUESTIONS ABOUT THE STUDY?

If you have any questions or desire further information about this study before or during participation, you can contact Dr. Jon Farthing at 306-966-1068 or Andrew Leslie at 306-380-9469.

If you have any concerns about your rights as a research participant and/or your experiences while participating in this study, contact the Chair of the University of Saskatchewan, Biomedical Research Ethics Board at (306) 966-4053. This study has been reviewed and approved on ethical grounds by the University of Saskatchewan, Biomedical Research Ethics Board.

CONSENT TO PARTICIPATE

Study Title: Static Stretching vs. Repeated Bout Effect: Which is the better muscle "primer" for subsequent eccentric training?

- o I have read (or someone has read to me) the information in this consent form.
- o I understand the purpose and procedures and the possible risks and benefits of the study.
- o I was given sufficient time to think about it.
- o I had the opportunity to ask questions and have received satisfactory answers.
- o I understand that I am free to withdraw from this study at any time for any reason and the decision to stop taking part will not affect my future relationships.
- o I give permission to the use and disclosure of my de-identified information collected for the research purposes described in this form.
- o I understand that by signing this document I do not waive any of my legal rights.
- o I will be given a signed copy of this consent form.

I agree to participate in this study:

Printed name of participant: Signature Date

Printed name of person obtaining consent: Signature Date

Appendix B – Data Collection Sheet

General Information

Subject No._____ Age: _____years Gender: M or F
Name: _____ Dominant Leg: R or L Leg Tested: R or L
Contact #:_____ Footedness score: _____
Height (cm): _____ Weight (kg): _____ Group: F S C

Ultrasound Data

EMG Placement

50% of total distance between ischial tuberosity and lateral epicondyle of tibia: _____cm

Muscle Thickness – Bicep Femoris Lh (cm)

Pre-Priming	Post-Priming	Post-Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Fascicle Length – Bicep Femoris Lh (cm)

Pre-Priming	Post-Priming	Post-Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Flexibility Testing Data

Sit & Reach Test (cm)

Pre-Priming	Post-Priming	Post- Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____

Passive ROM Test (degrees)

Pre-Priming	Pre-Priming	Pre-Priming
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____

Norm Settings for Tests

Passive ROM Test – Supine Straight Leg Hip Flexion

Chair Rotation: _____ Monorail: _____
Dyna Angle: _____ Leg Attachment: _____
Dyna Height: _____ Slide: _____

All Strength & Power Test – Hamstring Curl Prone

Chair Rotation: _____ Monorail: _____
Dyna Angle: _____ Leg Attachment: _____
Dyna Height: _____ Slide: _____

Strength & Power Testing Data

Peak Force - Isometric Knee-Flexion (Nm)

Pre-Priming	Post-Priming	Post-Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Velocity Dependent Contractions (peak velocity in deg/s)

Pre-Priming	Pre-Priming	Pre-Priming
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Optimal Angle (degrees)

Pre-Priming	Post-Priming	Post-Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Peak Torque – Eccentric Knee-Flexion (Nm)

Pre-Priming	Post-Priming	Post-Training
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Muscle Damage Tracking Data (S Group Only after initial damaging bout)

Peak Force - Isometric Knee-Flexion (Nm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Velocity Dependent Contractions (deg/s)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

VAS – Pain (mm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____

Muscle Damage Tracking Data (Post 1st Training Bout)

Peak Force - Isometric Knee-Flexion (Nm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Velocity Dependent Contractions (deg/s)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

VAS – Pain (mm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____

Muscle Damage Tracking Data (Post 4th Training Bout)

Peak Force - Isometric Knee-Flexion (Nm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

Velocity Dependent Contractions (deg/s)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____
2. _____	2. _____	2. _____
3. _____	3. _____	3. _____
4. _____	4. _____	4. _____

VAS – Pain (mm)

0hrs	24hrs	48hrs
1. _____	1. _____	1. _____

Appendix C – Waterloo Footedness Questionnaire

Waterloo Footedness Questionnaire---Revised

Instructions: Answer each of the following questions as best you can. If you *always* use one foot to perform the described activity, circle Ra or La (for **right always or left always**). If you **usually** use one foot circle Ru or Lu, as appropriate. If you use **both** feet **equally often, circle Eq.**

Please do not simply circle one answer for all questions, but imagine yourself performing each activity in turn, and then mark the appropriate answer. If necessary, stop and pantomime the activity.

1. Which foot would you use to kick a stationary ball at a target straight in front of you?
   La Lu Eq Ru Ra

2. If you had to stand on one foot, which foot would it be?
   La Lu Eq Ru Ra

3. Which foot would you use to smooth sand at the beach?
   La Lu Eq Ru Ra

4. If you had to step up onto a chair, which foot would you place on the chair first?
   La Lu Eq Ru Ra

5. Which foot would you use to stomp on a fast-moving bug?
   La Lu Eq Ru Ra

6. If you were to balance on one foot on a railway track, which foot would you use?
   La Lu Eq Ru Ra

7. If you wanted to pick up a marble with your toes, which foot would you use?
   La Lu Eq Ru Ra

8. If you had to hop on one foot, which foot would you use?
   La Lu Eq Ru Ra

9. Which foot would you use to help push a shovel into the ground?
   La Lu Eq Ru Ra

10. During relaxed standing, people initially put most of their weight on one foot, leaving the other leg slightly bent. Which foot do you put most of your weight on first?
    La Lu Eq Ru Ra

11. Is there any reason (i.e. injury) why you have changed your foot preference for any of the above activities? YES NO (circle one)

12. Have you ever been given special training or encouragement to use a particular foot for certain activities? YES NO (circle one)

13. If you have answered YES for either question 11 or 12, please explain:

Appendix D - Resistance Training and Injury Questionnaire

Resistance Training Experience and Previous Injury

1. If one month of resistance training is considered 3 times per week for 4 weeks, how much resistance training (in months) have you done?

a. In the previous year? _____ months

b. In your lifetime? _____ months

2. If you had previous resistance training experience, did this resistance training include any leg training? Y or N

If yes, did it include hamstring exercises for example curls or deadlifts? Y or N

If yes, was eccentric training (forceful muscle lengthening) part of your hamstring exercises? Y or N

3. Have you ever experienced an injury to the hamstring, hip or any part of the leg? If so, what was the duration of this condition?