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## ACCEPTANCE

This dissertation, EFFECT OF ECCENTRIC CONTRACTION-INDUCED INJURY ON INDIVIDUAL QUADRICEPS MUSCLES: IMPACT ON MUSCLE ACTIVATION, JOINT TORQUE AND MOTOR CONTROL, by CHRISTOPHER RAWDON, was prepared under the direction of the candidate's Dissertation Advisory Committee. It is accepted by the committee members in partial fulfillment of the requirements for the degree, Doctor of Philosophy, in the College of Education & Human Development, Georgia State University.

The Dissertation Advisory Committee and the student's Department Chairperson, as representatives of the faculty, certify that this dissertation has met all standards of excellence and scholarship as determined by the faculty.

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## **Published Abstracts**

<u>SEACSM Annual Meeting 2022-</u>**Rawdon**, C.L., Ingalls, C., Yang, F. (2022). COMPARISON OF THE BIODEX DYNAMOMETER AND KINEMATIC ANALYSIS FOR MEASURING INDIVIDUAL QUADRICEPS MUSCLE TORQUE.

<u>ACSM Annual Meeting 2021</u> – **Rawdon**, **C.L**., Brandenberger, K.J. (2021). Using Mechanomyography To Detect Muscle Atrophy Following Knee Ligament Injury: A Case Study. *Medicine and Science in Sports and Exercise*, 53(8S), 102.

<u>Military Health System Research Symposium 2020-</u> Brandenberger, K.J., Armstrong E., **Rawdon** C.L., Banda, J.M., Lonowski J., Cooper L., Warren, G.L. (2020). An Involuntary Intermittent Fatigue Test Measured by Mechanomyography Can Detect Functional Changes in Skeletal Muscle (conference canceled)

<u>ACSM Annual Meeting 2020</u> – Ingalls, C., Adan M., **Rawdon**, **C.L**.(2020). Effects of Exerciseinduced Muscle Injury on Quadriceps Muscle EMG during Locomotion. *Medicine and Science in Sports and Exercise*, 52(7S), 496. (conference canceled)

<u>ACSM Annual Meeting 2019</u> - \***Rawdon**, **C.L**., Ingalls, C. (2019). Low-Dose Rapamycin Facilitates Recovery from Exercise-Induced Muscle Injury. *Medicine and Science in Sports and Exercise*, 51(6S), 901-902.

<u>ACSM Annual Meeting 2018</u> - \***Rawdon**, C.L., Ingalls, C. (2018). Age Associated Muscle Strength Loss During A Single Bout of Eccentric Contractions in Mice. *Medicine and Science in Sports and Exercise*, 50(5S), 34-35.

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# EFFECT OF ECCENTRIC CONTRACTION-INDUCED INJURY ON INDIVIDUAL QUADRICEPS MUSCLES: IMPACT ON MUSCLE ACTIVATION, JOINT TORQUE AND MOTOR CONTROL

BY CHRISTOPHER RAWDON

### UNDER THE DIRECTION OF DR. CHRISTOPHER INGALLS

#### ABSTRACT

Research has shown that exercise-induced muscle injury can cause 25-50% reductions in maximal joint torque. However, it is unknown whether individual muscles of a synergist group are injured to the same extent following injury. We hypothesized that the extent of injury among synergistic muscles is not uniform, and the primary cause of the weakness stems from the failure of muscle and not the ability of the nervous system to activate the muscle. We also presumed that muscle injury would alter balance (postural sway) and quadricep muscle activation patterns (electromyography [EMG]) during locomotion. 15 healthy sedentary or recreationally active male subjects between 18 and 35 years old completed the study. Subjects performed either downhill running (DHR) on a treadmill for 60 min to induce injury (n=8) or level treadmill walking for 30 min as control (n=7). Before and after (immediately and 2-days) exercise, we measured 1) maximal voluntary contraction (MVC) torque of quadricep muscles (QMs), 2) torque produced by vastus medialis (VM), rectus femoris (RF) and vastus lateralis (VL) via electrical stimulation (20 and 80 Hz), 3) soreness of individual QMs, 4) QMs EMG root mean square (RMS) during running and MVCs, and 5) standing postural sway. MVC 90° torque was

significantly reduced immediately (25.3%) and 2-days (14.0%) after DHR, whereas torque was unchanged after level walking. After DHR (immediately and 2-days), MVC RMS across all three quadriceps muscles was significantly reduced by 16.8% immediately following injury. There was a 13.2% decrease in stimulated torque (20 & 80 Hz) collapsed across all muscles for the injury group immediately post and a decrease of 9.1% at 48H following. At 48 hours, the VL experienced greater soreness than RF and VM. Running RMS of the knee extensor muscles increased immediately after DHR. Postural sway increased immediately after DHR and decreased in the control group at 2-days. In conclusion, DHR caused the differential injury of the QMs, and reduced activation (i.e., RMS) of the quadriceps and force depression (i.e., 20 & 80 Hz torque) account for the decreases in MVC torque after DHR. In addition, muscle injury from DHR disrupted standing balance and normal muscle activation patterns during running.

Index words: Injury, Eccentric Contractions, Synergist, Skeletal Muscle, Exercise

## EFFECT OF ECCENTRIC CONTRACTION-INDUCED INJURY ON INDIVIDUAL

## QUADRICEPS MUSCLES: IMPACT ON MUSCLE ACTIVATION,

## JOINT TORQUE AND MOTOR CONTROL

BY

## CHRISTOPHER RAWDON

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## PRESENTED

## IN PARTIAL FULFILLMENT OF THE REQUIREMENTS

FOR THE DEGREE

## DOCTOR OF PHILOSOPHY

IN

## KINESIOLOGY

IN

## THE COLLEGE OF EDUCATION AND HUMAN DEVELOPMENT

## GEORGIA STATE UNIVERSITY

## ATLANTA, GA

#### **DEDICATION**

I dedicate this to the love of my life, Katie, and our son Nolan. Your love has motivated me to complete this journey and I will forever dedicate all that I do to the both of you.

To my mother, thank you for raising me, providing me love and instilling my best qualities including my relentless pursuit of achieving.

To my brother, thank you for being my life-long companion and a constant source of joy.

To my father, thank you for the constant support.

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#### CHAPTER ONE

#### THE PROBLEM

#### Introduction

Musculoskeletal injury is a risk associated with increased physical activity and exercise training. Musculoskeletal injuries can be classified into three grades based on the severity of the injury. Grade I is characterized as injury with no major architectural distortion of the tissue. Partial tears of tissue are observed in grade II injuries, and complete tears of the tissue are associated with grade III injuries (Lee & Healy, 2004; Chan et al., 2012). Overuse of musculotendinous structures can result in degeneration of the tendon, which can ultimately lead to a secondary injury such as complete rupture (Kjær, 2004). According to the Bureau of Labor Statistics, musculoskeletal disorders including sprains and strains from lifting, accounted for nearly a third of the total cases of all workers in 2015. Sprains, strains, and tears in skeletal muscle tissue were the leading occupational injury or illness for missed workdays (37% of 1.15 million cases) totaling roughly 421,610 days away from work in 2015. Strategies to reduce the incidences of partial or complete tears of skeletal muscle would have a profound economic impact on the workplace, increase the likelihood that individuals develop lifelong physical activity routines and prevent athletes from missing time away from their sport. Furthermore, understanding the impact of repetitive loading on the musculoskeletal system would aid in the prevention of secondary traumatic injuries such as tendinopathies, sprains, strains, and stress fractures that may result from overuse.

Exercise typically involves skeletal muscles performing three types of contractions: 1) concentric contractions normally accelerate limbs, 2) isometric contractions generally stabilize

joints, and 3) eccentric contractions typically decelerate limbs. Myofibers experiencing eccentric contractions produce higher forces than both static (isometric) and shortening (concentric) contractions (Hessel et al., 2017; Hody et al., 2019). When exercise is unaccustomed, the repetitive loading can lead to a grade I injury of skeletal muscle often referred to as "exerciseinduced injury." It is generally accepted that exercise-induced muscle injury is caused by highforce eccentric contractions that occur when the central nervous system allows external torque to exceed the torque produced by the skeletal muscles, causing sarcomeres and myofibers to lengthen while activated. This type of injury is characterized by muscle weakness, delayed onset muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation, swelling, and reduced range of motion in the days and weeks that follow (Howell et al., 1993; Clarkson et al., 1999; Warren et al., 2001; Warren et al., 2002). Importantly, research has shown that exercise-induced injury can cause an immediate 40-50% reduction of maximal strength in injured skeletal muscle groups that requires weeks to fully recover (Warren et al., 2001; Warren et al., 2002). In addition, exercise-induced injury alters neuromuscular recruitment and activation in both injured and uninjured muscles (Warren et al., 1999; Warren et al., 2000; Prasartwuth et al., 2006; Brandenberger et al., 2021). Although some studies report a decrease in muscle activation after eccentric contractions (Prasartwuth et al., 2006; Brandenberger et al., 2021), other studies do not find evidence of a failure in the nervous system to activate skeletal muscle (Warren et al., 2000; Warren 2001; Hubal et al., 2007). However, changes in the median frequency of the power spectrum of the electromyography (EMG) signal after eccentric contractions have been interpreted as a change in muscle activation pattern within the muscle (motor unit and fiber type change) (Warren et al., 2000; Warren et al., 2001). It is likely that

changes in muscle activation patterns and strength contribute to the decreases in the energy efficiency of contractions and movement after injury (Warren et al., 1996; Bauman et al., 2014).

Research investigating exercise-induced injury in humans typically assess skeletal muscle damage or soreness in one of the multiple synergistic muscles acting on a given joint or via blood markers of muscle injury. Strength deficits associated with exercise-induced muscle injury are normally assessed by measuring changes in joint torque which reflect the functional integrity of a given set of synergistic muscles. However, little is known about the degree of injury across synergistic muscles that promote the same joint movement. Given differences in muscle architecture (e.g., pennation angle, fascicle and muscle length), previous usage and recruitment patterns among synergistic muscles, it is possible that the extent of injury among synergistic muscles is not uniform. The notion of exercise-induced differential injury of synergists muscles is supported by nonuniform changes in indirect markers of muscle injury (e.g., magnetic resonance imaging [MRI] and tensiomyography) amongst the individual muscles (Prior et al., 2001; Black & McCully, 2008; Maeo et al., 2018, Beato et al., 2019).

#### Purpose

The goal of our study is to measure the degree of strength loss across the different knee extensor muscles (i.e., vastus medialis, vastus lateralis, and rectus femoris) following downhill running, which is an accepted human model of eccentric contraction-induced injury. The results from this experiment will give us insights into the question of whether the individual muscles of the quadriceps group experience the same magnitude of injury after undergoing repetitive eccentric contractions. In addition, we hope to understand whether these functional differences

are caused by impaired muscle activation and/or muscle torque generation due to a failure within the skeletal muscle. This will be the first study which uses low-frequency and high-frequency electrical stimulation to measure strength changes across a synergist muscle group after injury. This data will assist in determining whether failure to activate the skeletal muscle (i.e., EMG) or failure to activate force-bearing structures of the muscle (i.e., 20 Hz/80 Hz torque ratio) contributes to volitional loss of maximal knee extensor strength and differential muscle injury. We will also explore the effects this may have on muscle activation and balance during upright posture and movement. Understanding the extent of functional deficits of individual quadriceps muscles after injury should allow for better training or rehabilitation strategies that minimize the risk associated with developing secondary soft-tissue injuries associated with the knee.

#### **Research Questions**

- 1. Does differential injury (i.e., strength deficits and soreness) exist amongst the individual muscles of the knee extensor group following eccentric contractions?
- 2. What are the mechanisms of maximal voluntary contraction (MVC) strength loss of the knee extensor muscles after eccentric contractions (i.e., failure to activate the skeletal muscle, failure to activate force-bearing structures within the skeletal muscle, and/or failure of the force-bearing structures of the muscle)?
- 3. How does eccentric contraction-induced muscle injury, and possibly differential injury impact knee extensor muscle activation during locomotion?
- 4. How does eccentric contraction-induced muscle injury, and possibly differential injury impact balance during upright standing?

#### Hypotheses

- 1. Post-injury strength deficits and soreness will be significantly different between at least two knee extensor muscles following exercise-induced injury.
- The primary etiology of knee extensor strength deficits will reside in the skeletal muscle and not the nervous system, with a failure to activate force-bearing structures (20 Hz-to-80 Hz stimulation torque ratio) contributing to a failure of the force-bearing structures (80 Hz stimulation torque) in explaining volitional strength loss.
- Eccentric contraction-induced muscle injury will alter knee extensor muscle activation patterns during treadmill walking and running, as measured by EMG RMS and median frequency.
- 4. Eccentric contraction-induced muscle injury will impair balance, as indicated by the prolonged trajectory of the center of pressure (COP) during all three experimental conditions during quiet standing.

#### Limitations

Limitations for this study include determining activated muscle fibers, musculoskeletal architecture variability, and running stride. There is not a way to directly stimulate the vastus intermedius with stimulating electrodes since it lies under the rectus femoris. Consequently, the post-injury results will omit changes that occur in the vastus intermedius muscle. We cannot determine the total amount of skeletal muscle fibers that will be activated during stimulation. Being that this is the first attempt to quantify torque from the individual muscles of the quadriceps group in humans, there is no reference on how to maximize the number of fibers that are stimulated. However, we have chosen anatomical references for electrode placement that we

believe will effectively activate the most fibers of each tested knee extensor muscle. The variability of individual torque will be the measure used to determine significant differences between synergist skeletal muscles. Analysis of individual stimulated muscle torque was determined to be underpowered at the current sample size.

#### Delimitations

Delimitations include age, gender and physical activity level. Our proposed study focuses on healthy males between the ages of 18-35. Therefore, the results of our study may not reflect events that occur in females or in an older population. The reasoning for selecting a male population was determined based on anatomical and strength differences between males and females and differences that can occur in maximal strength during the ovulation cycle. The age range was chosen to determine neuromuscular changes that will occur in non-trained but healthy individuals without age-induced sarcopenia or bone loss which could alter strength and motor control.

#### CHAPTER TWO

#### **REVIEW OF THE LITERATURE**

#### Introduction

The effects of heterogenous injury on the activation and torque generation of synergist muscles have not been determined. Therefore, the purpose of this review is to investigate potential differences in susceptibility to injury across a synergist group after eccentric contractions and present findings to support the hypothesis of heterogeneous injury across a synergist muscle group. The focus of this review will be centered on the knee extensor muscles composed of the vastus medialis (VM), vastus intermedius (VI), vastus lateralis (VL), and rectus femoris (RF), but will include information from studies investigating other synergist groups. This review will present current findings regarding the neuromuscular control of joint movement and locomotion, susceptibility to eccentric contraction induced injury and the effects that injury have on the neuromuscular system in order to determine if there is evidence to support this hypothesis.

#### **Neuromuscular Control of Movement**

#### The Somatic Nervous System

Locomotion and volitional movements are initiated and maintained via the somatic nervous system, which includes a vast network of both central and peripheral nerves. Afferent (sensory) nerves relay peripheral somatosensory information to the central nervous system (CNS). Interneurons relay both sensory and motor signals between sections of the brain and spinal cord. Upper motor neurons project from the cerebral cortex and the brainstem to activate either interneurons of the CNS or lower motor neurons (Levine et al., 2012). Lower motor neurons originating from the brainstem and spinal cord directly innervate skeletal muscle fibers at the

neuromuscular junction (Purves, 2018). Lower motor neurons are classified as alpha or gamma neurons that innervate extrafusal (i.e., myofibers) and intrafusal (i.e., muscle spindles) fibers, respectively. The voluntary movement will require the activation of groups of skeletal muscle fibers each innervated by an  $\alpha$ -motor neuron (i.e., motor units) (Floeter et al., 2010). Skeletal muscle motor units generate the force necessary to maintain posture and to create voluntary movements of our skeleton. A single muscle can contain hundreds or even thousands of motor units that make up the muscle's entire motor pool. The central nervous system will recruit specific motor units throughout the movement of joints and can be influenced by an array of external and internal stimuli. Figure 1 illustrates a simplified model of neuromuscular regulation during voluntary trunk and limb movement. Regulation of movements requires signaling from sensory, motor and interneurons to coordinate activation of skeletal muscle fibers.

#### Motor Processing in the Brain

Many sections of the brain are involved with the initiation of voluntary movement via skeletal muscle activation. The direct pathway is the circuitry of the brain that initiates voluntary movement. Activation of this pathway starts in the motor cortex which synapses with neurons of the striatum that suppresses inhibitory neurons of the globus pallidus and in turn increases thalamocortical signaling (Alexander et al., 1990; Galvan et al., 2006; Freeze et al., 2013). The upregulation of excitatory signaling between the thalamus and primary motor cortex leads to increased activation of upper and then  $\alpha$ -motor neurons to activate skeletal muscle fibers. The indirect pathway is the circuitry of the brain that suppresses unwanted movements from interfering with desired movements (Freeze et al., 2013). Inhibitory neurons stemming

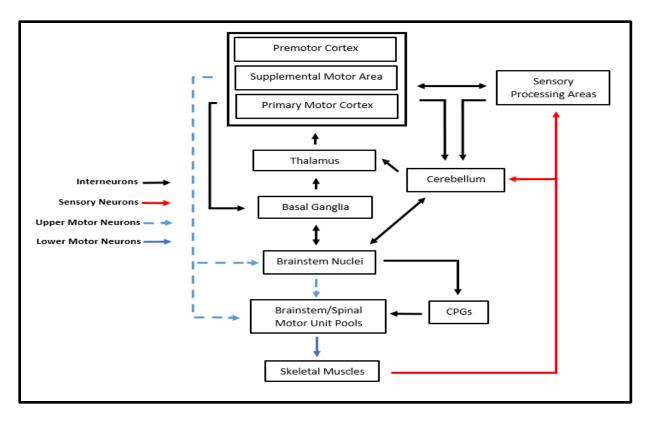


Figure 2.1 Simplified model of neuromuscular regulation during voluntary trunk and limb movement. Not all connections involved in motor control are shown. Higher areas of the brain initiate drive for movement and develop motor pattern. Upper motor neurons will synapse with lower brain centers and lower motor neurons to initiate skeletal muscle contraction. Peripheral sensory information is relayed to the nervous system influencing motor pathways.

from the substantia nigra and subthalamic nucleus inhibit the external globus pallidus to suppress activity of the thalamus which results in inhibition of downstream neuromuscular activity (Freeze et al., 2013). These areas of the brain are in constant communication to carry out motor tasks.

The motor cortex of the cerebrum includes the premotor cortex, primary motor cortex and supplementary motor area. The motor cortex receives signals from the thalamus to mediate a motor plan (Purves, 2018). The supplemental motor area is active prior to volitional movement and is thought to assist in the preparation of voluntary movement (Protopapa et al., 2019). Premotor cortex activity will help to plan, direct, and sequence the activity and strength of the skeletal muscles involved in a desired voluntary movement (Purves, 2018). This information is

relayed to the primary motor cortex. Some original understanding of the primary cortex "map" began through the research of Dr. Wilder Penfield and Dr. Edwin Boldery's in 1937. Direct stimulation to regions of the primary motor cortex elicits movements of skeletal muscles allowing researchers to see where voluntary movement commands originate. Pyramidal cells or upper motor neurons that originate from the premotor and primary motor cortex will activate corticospinal (i.e., trunk and limbs) or corticobulbar (i.e., facial) pathways. Upper motor neurons in the corticospinal pathway will innervate  $\alpha$ -motor neurons activating skeletal muscles fibers on the opposite side of the body. The motor cortex is responsible for the mapping and recruitment of motor units to execute voluntary movements of the body.

The basal ganglia is comprised of multiple sections of the brain including the striatum, globus pallidus, subthalamic nucleus, and the substantia nigra (Alexander et al., 1990; Galvan et al., 2006). These sections of the brain are involved with the integration and processing of sensory information and are part of the circuitry between the cerebral cortex and thalamus to carry out a motor plan (Galvan et al., 2006). Upon initiation of a movement, the motor cortex sends excitatory signals to inhibitory neurons of the striatum that results in the suppression of globus pallidus activity (Swenson, 2006). This reduces the inhibitory output the globus pallidus sends to the thalamus, in turn, increasing thalamocortical communication (Swenson, 2006). Neurons of the substantia nigra can suppress the indirect pathway or also stimulate the direct pathway leading to decreased inhibitory output of the globus pallidus internal and promoting activation of motor neuron pools (Purves, 2018). The communication between the basal ganglia, thalamus and the motor cortex shows part of the complex signaling that coordinates skeletal muscle contraction and movement.

In addition to the basal ganglia and cerebrum, the cerebellum helps to process motor output and influences coordination, balance and movement. The cerebellum receives inputs from the brain stem, motor cortex and sensory receptors of the musculoskeletal, vestibular, and ocular systems to help fine tune motor planning (Paulin, 1993; Morton & Bastian, 2004; Purves 2018). The cerebellum has been noted for its role of "smoothing-out" movements notably by influencing motor neuron activity. Some efferent neurons of the cerebellum synapse with neurons in the thalamus, red nuclei, and vestibular nuclei which can directly influence motor cortex signaling (Morton & Bastian, 2004; Purves, 2018). These afferent and efferent pathways allow for the cerebellum to receive information about the motor plan and provide feedback to the motor cortex based on sensory inputs. This area of the brain is heavily associated with balance and posture in humans. During locomotion, cerebellum activity will help to maintain the center of mass over a constant moving base of support (Morton & Bastian, 2004). The cerebellum helps to coordinate hip, knee, ankles and foot placement as well as foot trajectory during a walking motion (Morton & Bastian, 2004). While subjects with a cerebellar injury can adapt to perturbations while in movement, recruitment patterns have shown to be altered (Rand et al., 1998). In those with cerebellar injury, timing and activation patterns were distinct from controls during perturbated locomotion as demonstrated by varied electromyography (EMG) measurements (Rand et al., 1998). Balance and posture in humans can be assessed by sway histograms. Sway histograms are a postural assessment used for tracking a subject's oscillation around their center of mass (i.e., postural sway) while standing on force plates (Blaszczyk et al., 2003). Standing quiet posture in humans is maintained mostly by ankle torque (Bottaro et al., 2008). Center of mass, center of pressure, and oscillation can be recorded while subjects stand on force plates (Bottaro et al., 2008). Depending on the location and severity of a cerebellar injury,

individuals can develop postural instability as shown through altered sway position histograms when compared to controls (Dichgans & Mauritz, 1983; Morton & Bastian, 2004). The effects of exercise-induced injury on postural sway and cerebellar communication have not been fully explored. By investigating muscle activation and sway following exercise-induced injury, we can better understand how the nervous system responds in order to try and maintain balance.

#### Central Pattern Generators

Stereotypical flexion and extension at limb joints during locomotion are thought to be regulated by central pattern generators (CPG) or "neural oscillators" located in the spinal cord and brainstem (Grillner, 1975; MacKay-Lyons et al., 2002; Marder et al., 2001; Dietz, 2003). Central pattern generators are capable of outputting a rhythmic movement to the limbs without sensory information but will often require neuromodulators of the descending pathways to become activated (Marder et al., 2001). The effects of sensory input on CPGs activation are incompletely understood but it is generally accepted that CPGs activity is impacted by other areas of the brain in response to audio, visual or proprioceptive senses (Marder et al., 2001; Dietz, 2003; Guertin, 2013). Animal studies have shown that reciprocally inhibiting neurons help to generate rhythms that occur between opposing muscle groups during locomotion (Marder et al., 2001). Researchers also hypothesize that interlimb coordination of muscle activation is partially regulated at the spine in humans, similar to what is found in quadruped animals (Dietz, 1986; Gorassani et al., 1994; Dietz 2002). This conclusion is drawn from experiments where perturbations or split-belt running lead to disruptions during specific phases of gait in one leg that result in a change in the recruitment pattern (i.e., EMG) as well as the duration of certain gait phases in the other lower limb (Dietz, 1986; Dietz et al., 1994; Prokop et al., 1995). It is theorized that individual pattern generators are coupled via interneuron networks in order to

coordinate the movement of different joints both within the same limb and across the other limbs of the body (Marder et al., 2001; MacKay-Lyons et al., 2002).

#### Peripheral Feedback

Peripheral somatosensory receptors will relay sensory information such as fiber stiffness, tension, length and joint positioning to the central nervous system. The processing of these sensory inputs is a major component of mapping out movement pathways in the brain (Dietz, 1992; Andersen et al., 2002). Proprioceptors and mechanoreceptors of the musculoskeletal system relay spatial information that helps plan the desired speed and force of contraction necessary for a desired movement (Benarroch, 2006). The brain also uses feedback from the peripheral nervous system to accurately repeat previously performed movements (Benarroch, 2006). The posterior parietal cortex is another association area that routes sensory information and provides an interface between association and motor areas of the brain. Both the prefrontal cortex and posterior parietal cortex help to plan movement before sending messages to other motor processing regions of the brain. Sensory inputs are used by the cerebral cortex to map out the body's spatial recognition and properly recruit skeletal muscles for the execution of movement.

#### Motor Unit Recruitment and Activation

Throughout a movement, the nervous system will alter levels of motor unit recruitment based on the characteristics of the skeletal muscles involved as well as the joint torque needed to complete the movement. The nervous system coordinates the activity of synergist muscles to carry out a movement of a joint. In addition, activation of opposing muscle group is required for

the stabilization of the joint and to complete the other phases of movement. Different skeletal muscles will contribute to different segments of movement at a given joint. Skeletal muscles can cross multiple joints, which influences activation during a given phase of locomotion (Laqcuaniti et al., 2012). Many researchers theorize that the central nervous system activates a pool of motor units across multiple muscles based on the timing or phase of a specific movement as shown in EMG recordings of the trunk and leg muscles during locomotion (Laqcuaniti et al., 2012). A combination of four to five basic modular patterns coordinate walking across all the different leg muscles involved in human locomotion (Patla, 1985; Olree & Vaughan, 1995; Ivanenko et al., 2004 et al., 2005 et al., 2008; Cappellini et al., 2006; McGowan et al., 2010). Some skeletal muscles of the limb are activated during one of these phases, but others are activated during multiple phases. It has been determined that there is little change in the phases of activation even when there are changes in speed, loading or unloading, moving backwards or even while running (Ivanenko et al., 2004; Cappellini et al., 2006; Laquanti et al., 2012). However, the amplitude of activation as demonstrated by EMG does change with variations in speed, direction, or body weight loading and unloading (Ivanenko et al., 2004; Laquanti et al., 2012). Increasing the speed of locomotion also results in earlier peak activation and a decreased duration of the stance phase (Ivanenko et al., 2004; Cappellini et al., 2006). The central nervous system will coordinate smooth movements of joints during locomotion by regulating the recruitment of motor units across muscle groups.

The force generated by a given skeletal muscle is dependent upon the number of motor units recruited and the rate coding of an  $\alpha$ -motor neuron (Fuglevand et al., 1993; Enoka & Duchateau, 2017). As force requirements increase, the number of motor units recruited also increases. The number and type of skeletal muscle fibers recruited during a movement will

depend on the intensity of that activity. A skeletal muscle will contain motor units that differ in the threshold of activation. In general, low-threshold motor units are comprised of slow-twitch muscle fibers (Type I), whereas fast-twitch oxidative fibers (Type IIa) are associated with slightly higher threshold motor units and fast-twitch glycolytic fibers (Type IIx) have the highest threshold of motor units. Motor units associated with Type II fibers contain α-motor neurons with greater soma volumes and higher thresholds of activation than motor units associated with Type I fibers (Henneman et al., 1965b; Mendell, 2005). If the external load during a movement is great, high-threshold motor units become increasingly activated in addition to the low threshold motor units to generate the additional force necessary to complete the movement. Motor units are recruited based on their size termed the Henneman's size principle. Generally, lower threshold motor units containing fewer myofibers with small physiological cross-sectional area are recruited first, followed by larger, higher threshold motor units with greater myofibers if more force is required to generate the movement (Henneman et al., 1965a; Henneman et al., 1965b). Increasing the number of motor units recruited during a movement is one mechanism to increase the force developed by a skeletal muscle.

The nervous system also increases the force produced by a skeletal muscle fiber through rate coding. Rate coding is the frequency that  $\alpha$ -motor neurons are discharging action potentials (Enoka & Duchateau, 2017). A single firing of a motor neuron will produce a muscle twitch where the muscle will produce tension and then relax. When  $\alpha$ -motor neurons emit action potentials at a high frequency, there is an overlap and summation of twitch force producing a sustained activation of the muscle known as a tetanic contraction. Increases in action potential frequency by  $\alpha$ -motor neurons during tetanic contractions lead to a longer duration of SR Ca<sup>2+</sup> release, which more fully activates the sarcomeres resulting in a summation of twitch force that

maximizes the tension produced by skeletal muscle fibers (Altringham et al., 1982; Stephenson et al., 1982). Both the number and rate of impulses fired by an  $\alpha$ -motor neuron have been shown to impact the tension developed by a skeletal muscle fiber (Hennig & Lømo, 1987). Rate coding impacts forces produced by skeletal muscle fibers and are responsible for fused contractions that create smooth and coordinated movements of our musculature (Kernell & Sjo, 1975; Kanosue et al., 1979; Huijing, 1998).

The magnitude of recruitment and rate coding will vary during different types of contractions and is dependent on the motor unit type and size, fascicle length, and previous usage (Huijing, 1996; Dartnall et al., 2009; Semmler, 2014). The characteristics of rate coding and development of tension have been shown to be significantly different when comparing lowthreshold slow (Type I) and high-threshold fast (Type II) twitch motor units. In 1965, Buller and Lewis confirmed that slow motor units require a lesser frequency of firing to produce a tetanic contraction than compared to fast-twitch motor units. Kernell and Sjo (1975) demonstrated that faster motor units have a higher minimum-firing rate than slow-contracting motor units. Other experiments demonstrated that for every 1 Hz increase above half-maximum force, there was a greater increase in tension for slow-twitch fibers than fast-twitch fibers (Kernell et al., 1983). Torque and EMG experiments in human TA muscle have shown that fascicle length during contraction can also influence recruitment. Motor unit recruitment and discharge rates were greater in human tibialis anterior muscle during a submaximal isometric contraction at shorter fascicle lengths (10° dorsiflexion) than compared to longer lengths (10° plantarflexion) (Pasquet et al., 2005). It has also been observed that motor neurons have a greater discharge rate during concentric contractions than during eccentric contractions (Pasquet et al., 2006). The type of contraction has been shown to influence motor unit recruitment thresholds in humans, with

concentric and eccentric contractions having lower thresholds compared to isometric contractions but firing frequency was found to be greater during an isometric contraction (Tax et al., 1989; Theeuwen et al., 1994). The velocity of a contraction can also alter motor unit recruitment (Desmedt & Godaux, 1977; Tillin et al., 2018). Increases in velocity during an isokinetic movement are accompanied by increases in motor unit recruitment (Desmedt & Godaux, 1977; Tillin et al., 2018). Rate coding and recruitment of motor units during movement will not only vary based on the characteristics of the motor unit itself, but also based on proprioceptive feedback such as changes in muscle length, speed of the contractions, and the phase of locomotion. (Lacquanti et al., 2012).

#### Electromyography (EMG)

In order to measure the pattern and level of activation of a skeletal muscle, researchers have used surface electromyography. Du Bois-Reymond first recorded EMG signals in 1849. Since then, the method has been advanced to record the electrical signals created by the movement of ions during skeletal muscle contraction (Fridlund & Cacioppo, 1986; Kamen & Caldwell, 1996). Surface EMGs can record the action potentials of activated motor units and record the EMG wave while the muscle contracts (Moore, 1966; Merletti & Farina, 2016). Many researchers have also validated the use of EMG to predict skeletal muscle forces, but the relationship is not exactly linear (Hof & Van den Berg, 1981; Solomonow et al., 1990; Alkner et al., 2000). The root mean square (RMS) and mean power frequency (MPF) of the EMG varies with the number and frequency of action potentials being generated by motor units within a skeletal muscle (Christie et al., 2009; Li et al., 2014). However, EMG signal can also vary based on factors independent of motor unit activation and recruitment, including but not limited to muscle fiber conduction velocity, the distance between the electrode and the muscle, muscle

fiber length, and muscle fiber orientation (Christie et al., 2009; Li et al., 2014). It has been hypothesized that the physiological differences in skeletal fiber type and size can ultimately influence EMG median frequency and amplitude (Lissen et al., 1991; Kupa et al., 1995). However, it has been debated whether the spectral properties of EMG can properly differentiate muscle activity patterns of different motor units because conduction velocity can be impacted by muscle fiber diameter and not just type (Farina, 2008). This is one of a few reasons why relying on just EMG to determine levels of activation across different skeletal muscles has drawbacks. Increasing intensities of exercise result in decreased reliability of EMG measurements that could lead to variable measurements during a maximal voluntary contraction (Yang & Winter, 1983; Dankaerts et al., 2004; Mathur et al., 2005). Additional strategies such as twitch interpolation have been implemented to try to determine maximal voluntary activation levels by comparing force produced during a maximal voluntary contraction with the addition of an imposed electrical stimulation (Shield et al., 2004). Nonetheless, despite some shortcomings, EMG is the most commonly accepted method to investigate the level of skeletal muscle activation under specific conditions.

#### Skeletal Muscle Force Generation

When a skeletal muscle group is recruited, the linear force produced by the myofibers is translated to rotational force defined as torque. The force produced by activated skeletal muscle fibers will depend on several factors. Fibers with a greater cross-sectional area will produce greater tension than those that are smaller (Lieber & Fridén, 2001). The tension produced by each fiber is also dependent upon myofiber length often depicted as the length-tension relationship (Gordon et al., 1966). A fiber undergoing an eccentric contraction will also produce

greater tension than compared to a concentric or isometric contraction. The process of activation of force generating structures is known as "Excitation-Contraction Coupling" (E-C coupling) (Sandow, 1952). The "Sliding Filament Theory" describes force generation that occurs through the forming of cross-bridges between the contractile proteins myosin and actin (Huxley & Hansen, 1954). The cross-bridges formed through this process will contribute to the force production necessary to carry out movement by skeletal muscles.

#### Excitation-Contraction (E-C) Coupling

The E-C coupling process begins when an action potential from the  $\alpha$ -motor neuron reaches the neuromuscular junction causing acetylcholine (ACh) to be released from the end terminal of the α-motor neuron (Calderón et al., 2014). ACh binds to ACh receptors (AChR) that are located on the sarcolemma of the skeletal muscle fiber. The binding of ACh to AChR will change the receptor's conformation, leading to an influx of sodium (Na<sup>+</sup>) ions into the cell that causes a depolarization at the motor end plate (i.e., "end plate potential" (EPP) (Landau, 1978). When EPPs cause a skeletal muscle cell membrane potential to reach a threshold, neighboring voltage-gated Na<sup>+</sup> channels will open, further depolarizing the membrane and triggering an action potential. When the action potential reaches the T-tubules of the cell, it will activate Ltype voltage-gated calcium (Ca<sup>2+</sup>) channels (i.e., Dihydropyridine Receptors [DHPRs]) located on the sarcolemma. The action potential reaches the t-tubules and leads to an allosteric interaction between the DHPR and ryanodine receptors (RyR1) that will then open, resulting in Ca<sup>2+</sup> release from the sarcoplasmic reticulum (SR) (Calderón et al., 2014). Some of the Ca<sup>2+</sup> released will bind troponin that forms a complex with tropomyosin and actin on the thin filament of the sarcomere. The binding of Ca<sup>2+</sup> to troponin will shift the troponin/tropomyosin complex revealing myosin-binding sites on actin (Calderón et al., 2014). Excitation of a myofiber by an α-

motor neuron leads to SR Ca<sup>2+</sup>-release and formation of cross-bridges that increase the tension produced by an active myofiber.

#### The Sliding Filament Theory

The sliding filament theory developed in the 1950s proposed a molecular mechanism for skeletal muscle contraction (Huxley & Hansen, 1954). In this model, when  $Ca^{2+}$  is bound to troponin the myosin heads will hydrolyze ATP into ADP and an inorganic phosphate releasing energy (Huxley, 1969). The energized myosin forms a cross-bridge with an actin filament. If the internal tension produced by a fiber exceeds the external tension, the myosin heads will pull actin filaments towards the M-line of the sarcomere in an action termed the "power stroke" (Muretta et al., 2015). Although the exact process remains unclear, the release of inorganic phosphate from the myosin head is thought to trigger the power stroke. Binding of another ATP to the myosin head after the release of the ADP dissociates the myosin head from actin. If Ca<sup>2+</sup> remains on troponin and ATP is present, the force produced during myosin-actin cross-bridge cycling on each half of the sarcomere pulls the thin filaments over the thick filament acting to bring the Zlines closer during a concentric contraction. The relaxation of the muscle fiber will occur when sarco/endoplasmic reticulum  $Ca^{2+}$  ATPase (SERCA) pumps intracellular calcium  $[Ca^{2+}]_I$  back into the SR (Murray et al., 1998). The decreased intracellular  $Ca^{2+}$  levels will lead to tropomyosin covering myosin binding site on actin filaments and a decrease in cross-bridge formation resulting in the relaxation of the myofiber. The E-C coupling process and sliding filament theory outline the molecular events that are thought to result in force production and contraction of myofibers and skeletal muscles.

#### Joint Torque

#### Agonist vs. Antagonistic Muscle Groups

Joint movement is controlled by opposing muscle groups, commonly called agonist and antagonist groups, which are recruited to varying degrees during phases of movement. Joint movement and the rate of joint movement is dependent upon the forces produced by skeletal muscles that cross that joint. During joint movement, the agonist muscle group will be recruited to a higher degree than the antagonist group. However, both agonist and antagonist groups are activated to some degree during all phases of joint movement (Simmons & Richardson, 1988; Gottlieb et al., 1992). Although antagonist muscles oppose the joint movement produced by the agonist muscle group, antagonist muscle co-activation is important for joint stability and for deceleration of a limb (Simmons & Richardson, 1988; Gottlieb et al., 1992). During cyclical or rhythmic movements such as walking and running recruitment of agonist and antagonist muscle groups is thought to be regulated by the central pattern generators (Marder et al., 2001; MacKay-Lyons et al., 2002). CPGs are proposed to not only coordinate muscle activation in a single limb but also coordinate muscle activation bilaterally during locomotion (Guertin, 2013). Joint movement and locomotion are accomplished by recruitment and activation of both agonist and antagonistic skeletal muscle groups.

#### Contribution of Synergist Muscles to Joint Torque

The skeletal muscles of a synergist muscle group will be activated together to create joint movement in the same direction. For example, the vastus muscles and the rectus femoris will each produce force to contribute to the extension of the knee joint. Many synergist muscle groups will have a common insertion. In the case of the knee extensors the insertion is the tibial tuberosity, but these muscles are also connected via aponeurosis and fascial structure (Waligora

et al., 2009). It has been shown that many VM fibers insert to the aponeurosis of the VI and are activated by the same medial division of the femoral nerve to counter laterally acting forces on the patella which is critical for stability of medial patellofemoral joint (Grob et al., 2018). In addition to joining synergists anatomically, it is apparent that these connective structures have an impact on the transmission of force to joint movement. Huijing and Baan (2003) found that in addition to the tendon, rat EDL muscle transmits force through the extracellular matrix of muscle. The force transmission of skeletal muscles of the same group can be impacted by both intermuscular and extramuscular factors. Intermuscular transmission occurs between two neighboring muscles via the continuous connective tissue at their muscle belly interface (Maas et al., 2010). Extramuscular transmission occurs between the epimysium of a muscle and an adjacent non-muscular structure including connective tissue around the tendons or part of neurovascular tract as well as fascia lining synergist group through a superficial layer of connective tissue (e.g., subcutaneous) (Maas et al., 2010). Although the mechanics of synergist activation have been investigated there is still a limited understanding of how skeletal muscles within the same group individually influence torque and the movement of joints throughout an entire range of motion.

Current studies observing humans have used EMG to predict individual contributions of synergists during submaximal and maximal isometric contractions (Hubley-Kozey & Smits, 1998; Place et al., 2006; Saito & Akima, 2013) as well as during dynamic movement (Amarantini et al., 2010). Integrated myography (iEMG) is defined as the area under the curve of a rectified EMG signal and is used to quantify electrical activity from activated motor units. This is used to determine the ability of the nervous system to activate certain muscles or muscle groups (Sleivert et al., 1994). iEMG data has shown that the individual muscles of the knee

extensor group act in conjunction and that none of the individual muscles are principally responsible for fully extending the knee (Lieb & Perry, 1968; Speakman & Weisberg, 1977; Grob et al., 2018). Some of this research demonstrates that the recruitment of synergists can vary based on the activity as well as the individual (Hug et al., 2015a; Crouzier et al., 2019). When producing submaximal isometric knee extension torque (i.e.,  $\leq 50\%$  of MVC), the number of participants using greater activation (i.e., EMG) of the lateral head of the quadriceps (VL) was almost equal to those using greater activation of the medial head (VM) (Hug et al., 2015a). There is high variability across subjects when measuring normalized EMG amplitudes of individual skeletal muscles in both the knee extensor and plantar flexor groups during submaximal isometric contractions (Crouzier et al., 2019). The bias in activation to a particular skeletal muscle during a single-joint movement was correlated with the activation observed during locomotor tasks (Crouzier et al., 2019). This research points to the differences in muscle recruitment across individuals that may occur during many various types of movements. Since each individual muscle force contributes to joint loading, these differences across people may have implications for forces placed on the knee during movement (Sasaki & Neptune, 2010; Manal et al., 2013). It is unclear if individual architecture and varied movement patterns across subjects will alter relative force contributions in a synergist group during movement or during fatiguing or injurious contractions.

Maximal isokinetic and isometric torque are variables used to assess skeletal muscle strength and function. In humans, joint torque is typically measured using dynamometers. Many researchers have used both voluntary and electrically stimulated contractions when assessing skeletal muscle strength. Voluntary contractions will measure the joint torque produced by an entire synergist muscle group. There are no studies in which multiple muscle force

measurements from individual muscles of an agonist group were measured simultaneously during normal human movement. Shear wave electrography, which measures muscle stiffness, has also been used to compare synergist muscles (Hug et al., 2015b; Frietas et al., 2019) but force and stiffness are not linearly related (Herzog, 2017). The contribution of forces from individual skeletal muscles during locomotion have been previously investigated in animal models. Walmsley et al. (1978) first observed that the soleus muscle contributed more force to walking and slow trotting of cats compared to the medial gastrocnemius muscle which is much larger in size. This is an interesting considering that the cat soleus produces a quarter of the maximum isometric force of the medial gastrocnemius, highlighting the differences in motor processing between submaximal movement patterns and maximal isometric recruitment (Herzog, 2017). The soleus muscle also produced the same peak force during both walking and running while the force produced gastrocnemius tripled during running (Walmsley et al., 1978). Similar findings have been found by other laboratories and are displayed in Fig. 2 (Herzog, 2017). Standing requires significant contributions from the soleus muscle while the medial gastrocnemius contributes very little. In the activity of paw-shaking there is almost no contribution of the soleus and relies almost exclusively on the force generated by the gastrocnemius. At slow walking speeds, the contribution of the soleus is more than the gastrocnemius. As the velocity of walking or running increases, the gastrocnemius produces more force while the soleus produces relatively the same amount. When jumping, the medial gastrocnemius produces higher forces and soleus produces less force than during locomotion. Together, this data shows that the recruitment, activation, and force generated by specific muscles of the same group will vary based on the type of movement and intensity. More research must be done to further investigate how different joint movements are regulated both during submaximal and maximal voluntary contractions.

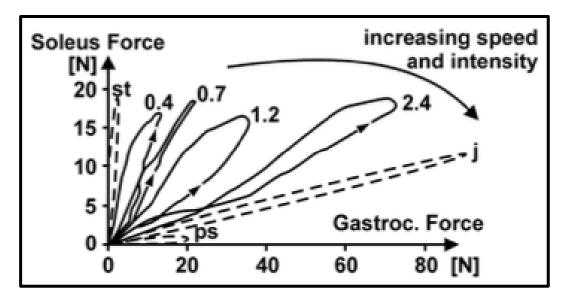


Fig. 2.2 Soleus vs. medial gastrocnemius forces obtained by direct measurement in the cat during a variety of postural and movement tasks. Soleus and gastrocnemius muscle forces are plotted for walking speeds of 0.4, 0.7 and 1.2 m/s and a running speed of 2.4 m/s. Forces also estimated using data from cats while standing still (st), pawshake (ps), and jumping (j) (Herzog, 2017).

There have been recent efforts dedicated to testing the current assumptions regarding the mechanics of synergist muscles. First, it has been assumed that torque-angle relationships of are similar across the individual muscles of a group (de Brito Fontana et al., 2018; Han et al., 2019). A second assumption is that the summation of maximal torque during individual muscle contractions in a synergist group equals the maximal torque when all are activated together (de Brito Fontana et al., 2018; Han et al., 2019). When testing these assumptions by maximally stimulating the knee extensors of rabbits, researchers found that relative contributions of individual quadriceps muscles to the total knee joint torque are not constant across joint angles (de Brito Fontana et al., 2018; Han et al., 2019). At many different angles, normalized forces are significantly different between the individual muscles of the knee extensor group (de Brito Fontana et al., 2018). The individual torque contribution of the VL was found to be over five

times the contribution of the VM and over double the contribution of RF despite similar physiological cross-sectional areas between those two muscles (Han et al., 2019). It was observed that normalized torque-angle curves of the individual muscles did not match the entire knee extensor group (de Brito Fontana et al., 2018). Peak torques for the individual muscles and the entire knee extensor groups occur at various angles (shown in Fig. 3). It was also found that the sum of the maximum torque capacity for the isolated stimulation of VL, VM and RF was approximately 10-20% higher than the maximum torque capacity for simultaneous stimulation of all muscles of the agonist group (de Brito Fontana et al., 2018; Han et al., 2019). The contribution towards movement by muscles within the same group is not just the summation of individual forces produced by each (Herzog, 2017; de Brito Fontana et al., 2018). An explanation for the loss of force when these muscles are stimulated together may be due to a change in the moment arm, a change in the amount of contractile element shortening associated with series elastic element elongations between the two conditions or a loss of longitudinal force due to the lateral compression that occurs when agonistic muscles contract simultaneously (de Brito Fontana et al., 2018). Surprisingly, the relative force produced by the individual muscles did not match the physiological cross-sectional area of the knee extensor group (Han et al., 2019). Researchers must develop methods that allow for simple, non-invasive, reliable, and accurate measurement of individual muscle forces in humans (Herzog, 2017). Having a reliable test to measure individual skeletal muscle forces could expand research in the fields of biomechanics, sports performance, and injury by potentially identifying differential strength deficits in synergist muscle groups. In addition, the effects of altered synergist mechanics on the development of secondary traumatic injuries are not clear. It is important for future research to expand current

understanding on how motor units within synergist muscles are recruited and how this translates to the production of force during various movements (Herzog, 2017).

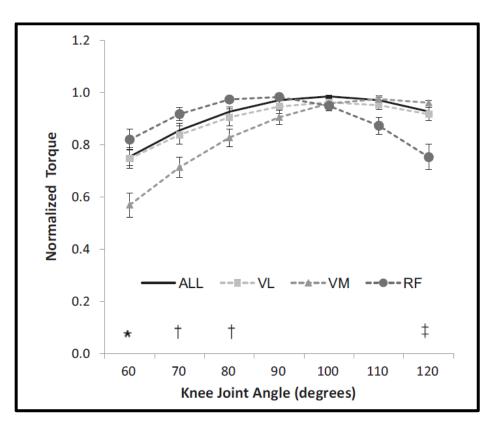


Fig. 3 Torque-angle relationships of the individual agonist muscles (VL, VM and RF) and of the entire agonist group (ALL) from rabbits. Muscle torque generating potential was measured by supramaximal stimulation of the corresponding femoral nerve branches simultaneously (ALL) and in isolation (VL, VM, RF). Increasing knee angles indicate knee flexion and increasing muscle lengths (0 = full extension). Symbols: \*, indicates lower values for VM compared to RF, VL and ALL (0.004 ); †, lower values for VM compared to RF (p = 0.003), and ‡, lower values for RF compared to VM, VL and ALL (<math>0.012 ). (de Brito Fontana et al., 2018)

## **Exercise-Induced Muscle Injury**

Characteristics and Time Course of Muscle Injury

Movement created by synergist muscle activation is critical to maintaining skeletal

muscle mass and function, as well as preventing inactivity related disease. However,

unaccustomed exercise is capable of injuring skeletal muscle and reducing functional capacity

for prolonged periods of time. Grade I exercise-induced injury is the most common type of injury

to skeletal muscle. This injury is a result of eccentric contractions that occur when internal torque

produced by the skeletal muscle or muscle group is less than external torque causing the activated myofibers to lengthen. Eccentric contraction-induced muscle injury is characterized by delayed onset muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation, swelling, and reduced range of motion (Howell et al., 1993; Clarkson & Sayers, 1999; Warren et al., 2001). The loss in maximal muscle strength, usually measured during isometric contractions is the primary functional characteristic of injured skeletal muscle fibers (McCully & Faulkner, 1985; Warren et al., 1992). Although this type of injury is marked by minor overt muscle cellular damage and focal cellular degeneration, both human and animal studies have indicated that maximal fiber and muscle force can be reduced by 40-50% immediately after unaccustomed eccentric contractions that can take 5-6 weeks to fully recover (Howell et al., 1993; Warren et al., 1993a; Lowe et al., 1995; Ingalls et al., 1998a; Ingalls et al., 1998b; Warren et al., 2001).

# Inflammatory Response and Delayed Onset Muscle Soreness (DOMS)

In response to injury, many cells of the inflammatory immune response are mobilized and activated to help repair damaged skeletal muscle tissue. Following eccentric exercise there is an immediate increase of neutrophils and monocytes in skeletal muscle tissue (Fielding et al., 1993; Malm et al., 2000). Experiments have shown that macrophage count will continue to increase in the days following injury (Malm et al., 2000; Stupka et al., 2001; Lowe et al). Some of these inflammatory cells release either pro or anti-inflammatory signaling proteins including cytokines, prostaglandin, and histamines. The pro-inflammatory cytokine IL-Iβ is elevated within an hour of eccentric exercise (Cannon et al., 1989; Fielding et al., 1993). An effect of IL-Iβ is that it contributes to the pain response in our bodies (Ferreira et al., 1988; Ren & Torres,

2009) which could partially explain some of the delayed offset muscle soreness (DOMS) that occurs after injury. Soreness will generally peak between 24-72 hours after exercise induced injury and has been commonly used as an indirect marker to detect muscular damage (Warren et al., 1999; Clarkson & Hubal, 2002; Lee et al., 2002; Peake et al., 2005). Prostaglandins, synthesized from lipid autacoids, are released from injured tissues and are also known to be part of the redness, swelling and pain response (Funk, 2001; Prisk & Huard, 2003). DOMS, swelling and stiffness are all secondary characteristics of eccentric contraction-induced muscle injury (Armstrong, 1984; Cleak & Eston, 1992). Not only is the immune response responsible for an increase in soreness and swelling following injury, but it also contributes to the loss of muscle proteins.

# Muscle Damage, and Protein Degradation and Synthesis

Myofiber disruption is seen immediately following eccentric contractions and histologic lesions will increase in size over the following 48 hours (Armstrong et al., 1983; Newham et al., 1983; McCully & Faulkner, 1985). Immunohistochemistry has shown that the damage that occurs in injured myofibers following eccentric contractions is segmental (Fridén & Lieber, 1998). Hypercontracted areas of fibers will undergo focal necrosis following eccentric injury (Fridén & Lieber, 1998; Lauritzen. 2009). Morgan and Talbot (2002) suggested that the segmental injury is due to nonuniform stretching of sarcomeres during a series of eccentric contractions. In skeletal muscle, recovery from injury is associated with an upregulation of degenerative processes (i.e., calpain, autophagy, ubiquitin-proteasome) to remove damaged proteins and organelles (Stupka et al., 2001; Kanzaki et al., 2016; Shang et al., 2019). In rabbits, rapid loss of the cytoskeletal protein desmin has been observed shortly after the onset of an injurious eccentric contraction protocol (Lieber et al., 1994). In mice, the protein degradation

rate increases significantly 24 hours post-injury and by two days plateaus at a rate 60% greater than normal (Lowe et al., 1995; Ingalls et al., 1998a). The elevated rate is maintained for at least three more days post-injury (Lowe et al., 1995; Ingalls et al., 1998a). There is a significant loss of contractile protein beginning at 24 hours following injury and by five days actin and myosin heavy chain contents are reduced by 20% (Lowe et al., 1995; Ingalls et al., 1998a; Ingalls et al., 1998b). In vivo mouse injury models have demonstrated that strength begins to recover three to five days post injury but the strength deficit from 2 weeks on can be accounted for by loss of contractile protein content (Lowe et al., 1995; Ingalls et al., 1998a; Warren et al., 2002). An upregulation in protein synthesis in the days following injury is necessary for the recovery skeletal muscle function and strength (Baumann et al., 2016). Rates of muscle protein synthesis are depressed within the first 6 hours after injury but significantly increase above baseline levels 3 to 5 days after injury (Lowe et al., 1995). Satellite cell proliferation is crucial for repairing damaged fibers and is also necessary for the recovery of force following injury (Rathbone et al., 2003). Removal and replacement of damaged proteins following eccentric contractions are part of the response which contributes to the recovery of structure and function of skeletal muscle fibers.

### Effects of Exercised-Induced Injury on Motor Control

Research has demonstrated that activation patterns are altered in both injured and uninjured muscles following eccentrically biased exercise. Human studies have shown an increase in motor unit recruitment while performing submaximal contractions following injury (Dartnall et al., 2008/2009). An increase in elbow flexor EMG at submaximal intensities following eccentric contractions has been found in multiple studies (Prasartwuth et al., 2005;

Semmler et al., 2007; Dartnall et al., 2008/2009; Dundon et al., 2008). These changes may be due to alterations in motor unit recruitment threshold and firing rate. Recruitment threshold or the force at which motor units are recruited, can be altered depending on the details of a movement performed and may be altered after eccentric contractions (Desmedt & Godaux, 1977; Pasquet et al., 2006; Dartnall et al., 2009). Motor unit recruitment thresholds decrease after injury (Dartnall et al., 2009) and discharge rates of single motor units are significantly higher after eccentric exercise (Dartnall et al., 2008). Motor unit synchronization, defined by crosscorrelation of motor unit pairs during low-force submaximal contractions, was 30% greater immediately following eccentric exercise and sustained for 24 hours (Dartnall et al., 2008). A 57% increase in motor unit synchronization from baseline has been observed in the elbow flexor muscles 7 days following eccentric contraction injury while other markers including strength, soreness and relaxed elbow joint angles had recovered by this time (Dartnall et al., 2011). There is also evidence that motor unit recruitment patterns are altered in uninjured muscles with multiple studies showing increases antagonist muscle co-activation after eccentric injury (Leger & Milner, 2001a/b; Semmler et al., 2007; Vila-Chã et al., 2014). Other studies have even demonstrated altered activation in uninjured muscles following eccentric contractions of a different limb. Voluntary activation (i.e., EMG) of the elbow flexors was reduced at 24- and 48hours post-injury after a downhill running protocol designed to injure the knee extensor group (Brandenberger et al., 2021). In addition, maximal voluntary forces of elbow flexors were significantly reduced immediately following and 24 hours following the downhill running protocol (Brandenberger et al., 2021). Based off these findings, Brandenberger et al. (2021) presumed that pain and inflammation caused by injury could potentially promote CNS dysfunction leading to altered recruitment patterns. It is thought that increases in motor unit

recruitment during submaximal contractions are a mechanism to compensation for the reduced intrinsic tension produced by injured fibers of that muscle (Dartnall et al., 2009). Altered motor unit recruitment patterns have been suggested to partially contribute to less damage following eccentric contractions (Chen, 2003; McHugh, 2003; Howatson et al., 2007). With fatigue, the nervous system tends to increase muscular contraction to maintain joint stability (Cashaback & Cluff, 2015). Motor unit threshold, firing rate and recruitment are all impacted following injury to maintain motor control during movement.

Changes in motor recruitment may be due to impairments in proprioception after eccentric injury. Moreover, increased inflammation due to eccentric exercise can decrease activity of Group IV afferent nerves altering muscle sensory motor control (Marqueste et al., 2004). After eccentric contraction-induced injury was induced in the elbow flexors of one arm, subjects consistently under-shot a target force produced by their unexercised contralateral arm (Saxton et al., 1995; Brockett et al., 1997; Miles 1997). The subjects perceived that they were producing more force than they actually were. These results show how altered spatial inputs from injured muscles can potentially impact proprioception and recruitment patterns of other muscles throughout the body. When trying to match joint angles of injured and non-injured arms, subjects tended to be in a greater extended position (Brockett et al., 1997) or a greater flexed position (Saxton et al., 1995) for their injured arm. Despite the differences in these studies, it appears that eccentric contractions can alter brain processing of spatial recognition, which may have abnormal effects on motor unit recruitment throughout the body. These effects have been shown to be prolonged following injury.

Mechanisms of Skeletal Muscle Strength Loss and Recovery Associated with Eccentric Contraction-Induced Injury

Although animal studies clearly demonstrate that the etiology of the eccentric contraction-induced force deficit resides in the skeletal muscle itself, human studies also demonstrate that the decrease in volitional maximal muscle strength may also stem from a failure to activate skeletal muscle (Prasartwuth et al., 2006; Brandenberger et al., 2021). Currently, each mechanism's exact contribution to volitional strength deficits following eccentric contractions have not been distinguished. However, much research has been dedicated to the mechanisms of strength loss throughout the neuromuscular system.

#### Mechanisms of Strength Loss Associated with Skeletal Muscle

The loss of strength following exercise-induced injury can be separated into distinct phases. In mouse models of muscle injury, immediate and early (i.e., out to 5 days) muscle strength deficits are primarily attributed to excitation-contraction coupling failure of muscle fibers ("E-C uncoupling") while prolonged muscle weakness for up to 5 weeks stems primarily from loss of myofibrillar proteins (Lowe et al., 1995; Ingalls et al., 1998a; Warren et al., 2001; Warren et al., 2002). Force loss following injury at the cellular level can be contributed to three categories: (1) damage to force-generating and/or force-transmitting structures (2) a failure to activate intact force-generating structures (3) a frank loss of force-generating or forcetransmitting structures (Warren et al., 2001; Warren et al., 2002). Eccentric contractions lead to force deficits for prolonged periods in skeletal muscle fibers.

## E-C Uncoupling

Most of the force deficit that occurs within the immediate hours and days following eccentric contraction induced injury is attributed to reduced SR Ca<sup>2+</sup> release in myofibers known as "E-C uncoupling" (Warren et al., 1993a; Balnave 1995; Lowe et al., 1995; Ingalls et al., 1998a; Warren et al., 2001; Warren et al., 2002). Intracellular Ca<sup>2+</sup> levels are an indicator of cross-bridge formation and the resultant production of force in skeletal muscle fibers (Huxley & Simmons, 1971; Hibberd & Trentham, 1986; Zot & Potter, 1987; Stein et al., 1988). As intracellular  $Ca^{2+}$  levels increase so will the force produced by the fiber until  $Ca^{2+}$  saturates all troponin binding sites and allows for maximal cross-bridge formation. Multiple studies have shown there is a significant decrease in tetanic  $[Ca^{2+}]_i$  in injured skeletal muscle fibers within just one hour following eccentric contractions (Balnave et al., 1995; Ingalls et al., 1998a; Kamandulis et al., 2017). It has been determined that E–C coupling failure could account for 57– 75% of the isometric strength deficit in the first five days after injury in an *in vivo* mouse injury model (Ingalls et al., 1998a). Many researchers have tried to identify the site of uncoupling in skeletal muscle motor units. A combination of multiple factors could potentially account for the uncoupling of the excitation-contraction process. Previous studies have been able to identify that neuromuscular junction function, action potential-conducting capacity along the plasmalemma, and intrinsic sarcoplasmic reticulum function are not significantly altered 24 hours after injury (Warren et al., 1993a; Ingalls et al., 1998a; Warren et al., 1999). It is presumed based on this data that disruptions to these cellular structures are not the primary cause of E-C coupling failure and consequential force deficits following injury (Warren et al., 2002). This has led to the hypothesis that alterations in the allosteric communication between DHPR and RyR could potentially be the point of uncoupling in the excitation-contraction mechanism following injury. Following injury,

changes into other auxiliary proteins, including FKBP12, calmodulin, calsequestrin, and junctophilin, may also alter DHPR or RyR function contributing to E-C uncoupling (Favero, 1999; Ito et al., 2001; Komazaki et al., 2002; Hirata et al., 2006; Corona et al., 2010; Murphy et al., 2013; Baumann et al., 2014). Other altered cellular mechanisms may also contribute to declines in tetanic  $[Ca^{2+}]_i$  following injury. For example, it has been found that SR  $Ca^{2+}$  uptake is significantly decreased 3-5 days following injury which directly impacts  $Ca^{2+}$  flux and hence force produced by injured fibers (Ingalls et al., 1998a). It is likely that many different factors could contribute to decreases in tension produced by injured fibers in the days following injury.

Although E-C uncoupling has not been directly demonstrated in human skeletal muscle after eccentric contractions, disproportionate reductions in electrically-induced submaximal torque compared with electrically-induced tetanic torque have been traditionally used as indirect evidence supporting its contribution to strength deficits (Edwards et al. 1977; Jones et al., 1982; Ingalls et al 2004; Kamandulis et al., 2017). For example, after a series of jump drops to induce eccentric contraction injury in human subjects, maximal voluntary contractile torque of the knee extensors was decreased by approximately 30% at 1 and 24 hours after the exercise. Moreover, electrically induced submaximal torque of the knee extensors was reduced approximately 50%, whereas electrically induced maximal torque was only reduced by about 20% at 1 and 24 hours after the exercise which is suggested of E-C uncoupling. Whether E-C uncoupling occurs to the same degree among all the knee extensor muscles after eccentric contractions remain unknown.

### Damage and Loss of Force Bearing Structures

It has been observed that skeletal muscle fibers produce roughly half of the maximal  $Ca^{2+}$  activated force one hour following contraction-induced injury (Balnave et al., 1995; Kamandulis et al., 2017), implying that there are other factors contributing the loss of strength following

injury in addition to E-C uncoupling. Some of the deficit in the days and weeks following injury can be contributed to decreases in contractile protein content as well as damage to forcetransmitting structures (Warren et al., 2001; Warren et al., 2002). However, force loss following injury does not match the time-course for histopathology data (Warren et al., 2001; Warren et al., 2002). Research has found that the greatest strength loss occurs immediately following the injury (Clarkson et al., 1992; Lowe et al., 1995; Ingalls et al., 1998a). Yet findings from histopathology show that significant damage to the force-generating and force-bearing structures does not peak until two to four days following the initial injury (Armstrong et al., 1983; McCully & Faulkner, 1985; Lowe et al., 1995). In addition, loss of contractile proteins will begin roughly one day following injury and 20% of actin and myosin heavy chain contents are reduced by 5 days (Warren et al., 2002). The results from many experiments have led to the assumption that E-C uncoupling is the primary factor behind force deficits for up to 3-5 days following eccentric contractions and the loss of myofibrillar proteins accounting for suppressed strength beyond two weeks (Lowe et al., 1995; Ingalls et al., 1998a; Ingalls et al., 1998b). Changes to the forcebearing and force-transmitting structures post-injury partially explain some of the strength loss immediately following injury while the loss of contractile proteins explains the loss of strength beyond 14 days.

## Mechanism of Strength Loss Associated with the Nervous System

Eccentric contraction-induced muscle injury alters the activation of both injured and uninjured skeletal muscles. While certain studies report decreases in muscle activation following eccentric contractions (Prasartwuth et al., 2006; Brandenberger et al., 2021) many studies associate strength deficits with peripheral factors showing no changes in EMG amplitude or median frequency (MF) during voluntary maximal isometric contractions following exerciseinduced injury (Warren et al., 1999; Hamlin & Quigley, 2001; Prasartwuth et al., 2005; Hubal et al., 2007; Semmler et al., 2007; Meszaros et al., 2010). No differences in median frequency during 40 Hz isometric contractions were seen following eccentric contractions in mice (Warren et al., 1999). However, the median frequency of human tibialis anterior muscles decreased by 34% over the course of fifty maximum voluntary eccentric contractions (Warren et al., 2000). It should be noted that it is difficult to determine whether lack of motivation caused by fatigue would cause changes in motor unit recruitment during a volitional maximum effort (Gandevia, 2001). Despite most evidence showing there is not a failure in the central nervous system to activate skeletal muscle fibers following eccentric contractions, there are studies that show alterations in motor unit recruitment. Warren et al. (2000) saw increased dependence on lower threshold motor units during the performance of maximal voluntary contractions after previous exposure to eccentric contractions. Evidence for this was seen when median frequency of the tibialis anterior muscle was lower during a second bout of eccentric contractions without a concomitant decrease in RMS (Warren et al., 2000). Median frequency was significantly lower during a second bout of eccentric contractions that was one week following the first (Warren et al., 2000). This mechanism may potentially account for the adaptations seen in the "repeated bout effect" where following recurring bouts of eccentric contractions there is less injury to the skeletal muscle (Nosaka & Clarkson, 1995; Warren et al., 2000). Motor unit recruitment is significantly affected throughout the nervous system following eccentric contraction injury.

## Susceptibility to Exercise-Induced Muscle Injury

#### Mechanical Factors

Eccentric contractions cause an immediate uncoupling of the excitation contraction process in vivo which is not seen following an equal number of concentric contractions performed at the same angular velocity and displacement (Warren et al., 1999). Peak torque decreased roughly 43% over a bout of 150 eccentric contractions while there was no significant difference in peak torque between the first and 150<sup>th</sup> concentric contraction (Warren et al., 1999). Although several mechanical variables (i.e., strain, starting length, and velocity) are known to contribute to strength deficits associated with eccentric contraction-induced muscle injury, the primary mechanical variable that causes strength loss is the peak force of the contraction. Warren et al. (1993b; 1993c) concluded that the magnitude of injury was related to muscle stress in rat soleus muscle when other mechanical variables were controlled. These studies extended the observations of Katz and McCully by demonstrating that peak eccentric force, independent of lengthening velocity and length changes at any point, is associated with initiating the injury process (Katz, 1939; McCully & Faulkner, 1986). However, Lieber and Fridén observed that the magnitude of injury in rabbit tibialis anterior muscles was closely related to the magnitude of the muscle strain since maximum tetanic tension was similar in two different strain-timing patterns (1993). The high forces and strain produced during eccentric contractions cause damage to skeletal muscle fibers but other factors such as the number of contractions, previous myofiber contractile activity, fiber type and muscle architecture also can contribute to skeletal muscle susceptibility to injury.

## Number of Contractions

The severity of exercise-induced muscle injury increases with the number of eccentric contractions. As the number of forced lengthening contractions increases, muscle damage and strength deficits also progressively increase (Hesselink et al., 1996). In single fibers, thirty stimulated eccentric contractions resulted in significant decreases in maximal Ca<sup>2+</sup>-activated force while a protocol with only ten contractions did not show statistically significant differences from baseline values (Balnave et al., 1995). Balnave and Allen (1995) demonstrated that under physiological conditions it takes between thirty and several hundred eccentric contractions to see single flexor brevis fiber force declines of up to 30% of control values. It should be noted that just 5 high force eccentric contractions are enough stress to cause significant damage and strength deficits in rat soleus fibers (Warren et al., 1993b). Increasing the number of eccentric contractions will result in higher prevalence of injury in skeletal muscle fibers.

### Previous Contractile History: The Repeated Bout Effect

Many markers of muscle damage (i.e., muscle soreness and creatine kinase release into circulation, joint range of motion) are minimized after a repeated bout eccentric contraction in both animals and humans i.e., "repeated bout effect" (Nosaka & Clarkson, 1995; Hortobágyi et al., 1998; McHugh et al., 1999). Although persistent immediate strength losses can be seen for multiple bouts, recovery of strength loss is accelerated with the repeated bout effect (Nosaka & Clarkson, 1995; Warren et al., 2000; Ingalls et al 2004). Warren and coworkers (1994) determined that the history of mechanical use of skeletal muscle is a primary reason why some skeletal muscles are more susceptible to injury than other muscles. For example, mouse EDL muscle is a non-weight-bearing fast-twitch muscle whereas the soleus muscle is a first soleus for the soleu

contractions, whereas the soleus only displays 7.6% strength loss after the exact same eccentric contraction protocol (Warren et al 1994). However, when mechanical stress experienced by the soleus muscle was reduced via hindlimb-suspension for two weeks, the soleus muscle exhibited maximal isometric force deficits similar to that of the EDL muscle and was nearly 4-times greater than weight bearing mice after eccentric contraction-induced injury (Warren et al., 1994). Although fast-twitch muscle does exhibit greater intrinsic susceptibility to exercise-induced muscle injury than slow-twitch muscle, the primary explanation of differences in the magnitude of the injury between the two fiber types is mechanical use and not fiber-type per se. In general, slow-twitch myofibers are associated with low-threshold slow motor units that are used for all physical activity, whereas fast-twitch myofibers are associated with higher threshold motor units that are used less frequently than slow-twitch myofibers (Henneman 1965a; Altenburg et al., 2007).

Potential mechanisms behind the protective effect of previous contractile activity could reside in the skeletal muscle itself or be associated with changes in the nervous system control of the skeletal muscle (Clarkson & Sayers, 1992; Warren et al., 2000). It has been estimated that the addition of sarcomeres in series accounts for at least 85% of the protection from future bouts of eccentric contractions (Morgan & Talbot, 2002). Researchers have also suggested that the change in optimum fiber angle, due to the addition of sarcomeres in a fiber following a period of eccentric exercise training, is the major mechanism by which the muscle is protected from damage from future bouts of eccentric contractions (Sacco & Jones, 1992; Morgan & Talbot, 2002). It is also suggested that the removal of structures within susceptible fibers or complete removal of these fibers could also partially explain this adaptation (Armstrong et al., 1983; Foley

et al., 1999; Ingalls et al 2004). The repeated bout effect is associated with reduced markers of muscle damage and faster recovery from eccentric-contraction induced injury.

#### Muscle Fiber Type

Many researchers have argued that fiber type alters susceptibility to injury. Animal experiments have shown that fast-twitch skeletal muscle fibers are predominately injured following a bout of maximal eccentric contractions (Fridén et al., 1983; Lieber & & Fridén, 1988; Warren et al., 1994; Vijayan et al., 2001). Low-threshold motor units typically innervate Type I fibers and are used for everyday movement and locomotion (Henneman et al., 1965a). It is suggested that since slow-twitch fibers of these animals are more exposed to active lengthening contractions day to day, the addition of sarcomeres in these fibers alters the length-tension characteristics making them less susceptible to injury than type II fibers (Brockett et al., 2002; Morgan & Talbot, 2002). Since these fibers are used more often, Type I fibers will be more resistant to injury compared to Type II fibers innervated by high threshold motor units that may never be recruited during normal activity for a sedentary individual. However, exercise intensity would also theoretically impact the distribution of injured fibers. Since Type I fibers are predominantly recruited at lower intensities (Henneman et al., 1965a; Beltman et al., 2004), this would potentially make them more susceptible to injury than Type II fibers following repetitive low-force eccentric contractions. Type II fibers are increasingly recruited as exercise intensity increases (Henneman et al., 1965a; Beltman et al., 2004) thus increasing susceptibility as external loads increase during physical activity. Although fiber type has been proposed as a factor that influences susceptibility to injury, previous usage and exercise intensity would also account for differences in injury across fibers.

# Musculoskeletal Architecture

Susceptibility to injury across individual muscles of a group may be influenced by musculoskeletal architecture. Longer fibers with additional sarcomeres would experience less sarcomere strain at a given joint angle change than compared to shorter fibers with the same fascicle angle. Pennation angle can also impact fiber and aponeurosis strain (Shin et al., 2009). Altered fascicle lengths and angles can significantly impact forces produced by skeletal muscle (Lieber & & Fridén, 2000) and could potentially impact the stress and strain intrinsically during eccentric contractions. Given differences in architecture, the individual muscles of the knee extensor group will have different mechanics throughout the joint's entire range of motion (ROM). During locomotion, varying recruitment and mechanics would lead to differences in myofiber stress and strain which are the primary causes of exercise-induced injury. The relationship between architecture and susceptibility to injury also has implications for individuals with musculoskeletal conditions such as knee valgus (knock-knees) or knee varus (bow legged) where there are differences in muscle fiber length and pennation angle. When observing the gastrocnemius fibers in both populations, Namavarian et al. (2017) observed that those with knee varus had medial gastrocnemius fibers that were shorter and had less cross-sectional area than the lateral gastrocnemius while those with valgus had longer and bigger fibers in their medial gastrocnemius. Alterations in musculoskeletal alignment whether through genetic disposition, inactivity or other lifestyle behaviors would place chronic stress and strain that could modify muscle architecture properties of synergist muscles (Timmins et al., 2016) and impact susceptibility to injury. The effects of musculoskeletal conditions and limb alignment on susceptibility to injury has not been fully explored. Changes in muscle architecture can ultimately impact the forces produced by muscles of a synergist group and could potentially influence susceptibility to injury in individual muscles.

# **Question of Differential Injury**

## Evidence of Differential Injury in a Synergist Muscle Group

Injury research in humans often focuses on a synergist muscle group (e.g., knee extensors) but it is currently unknown if the level of injury across the independent muscles is the same. No studies to date have measured differences in force deficits of individual muscles after eccentric exercise in humans. Current knowledge on differential injury among synergistic muscles has been based on measurements including MRI, tensiomyography and EMG each of which have shortcomings when interpreting the presence of functional skeletal muscle injury. Tensiomyography is a method used to measure muscle contractile properties including stiffness and relative muscle contraction velocity. This method has high retest reliability for measuring contraction time and radial displacement of muscle belly (Piqueras-Sanchiz et al., 2019; Beato et al., 2019). Although it may not be able to measure the force produced by the individual muscles of the same group, it can be used to measure specific contractile differences between muscles after skeletal muscle injury. Beato et al. (2019) found that depending on the eccentric exercise activity (i.e., crosscut step with pulley, flywheel, flywheel squat) there are significant differences in contraction velocity, muscle reactivity (time delay) and maximal radial displacement for some of the individual muscles of the quadriceps but not for others. This is evidence that following eccentric contractions there can be significant differences in the contractile properties between the vastus medialis, vastus lateralis and rectus femoris.

Transverse relaxation time (T2)-weighted MRI is a method that can measure the water content of muscle tissue given as a T2 value (Black & McCully, 2008; Maeo et al., 2018). By using this method researchers can quantify the edema or swelling that takes place after skeletal muscle injury. As stated earlier in this review, swelling will peak 24-48 hours following injury

and is correlated with the loss of strength following injury but cannot substitute as an indicator of functional changes to skeletal muscle following eccentric exercise. Black & McCully, 2008 (2008) investigated differences in the T2 signal in the individual muscles of the knee extensor group both after voluntary and stimulated eccentric exercise. T2 signal intensity increased in all four individual quadriceps muscles (VM/VL/VI/RF) after both electrically stimulated and voluntary eccentric exercise. However, the VM muscle demonstrated the largest increase in T2 signal, which was significantly different then the T2 signal of the VI muscle after the original eccentric bout (Black & McCully, 2008). A greater change in T2 was also seen in the RF muscle compared with VL (Black & McCully, 2008). The results of this study were similar to Prior et al., in which a greater change in T2 signal was shown in the RF muscle compared with the vasti muscles (Prior et al., 2001). There was not a significant increase in T2 signal after the second voluntary bout of eccentric exercise for all knee extensor muscles (Black & McCully, 2008). Maeo et al. also investigated changes in T2 intensity after three different methods of injury application including downhill weighted walking, squat, knee extension. All three methods caused a significant damage to at least one muscle of the quadriceps group (Maeo et al., 2018). After the bout of knee extensions there was significance change in T2 for the distal, middle and proximal portion of the RF muscle at 48 and 72 hours as well as for the middle portion of the VI muscle at 48 hours in addition to proximal/medial VM muscle at 24,48 and 72 hours (Maeo et al., 2018). After the squat protocol there was significance change in T2 for the middle portion of the VM muscle at 24 and 48 hours (Maeo et al., 2018). Following weighted downhill walking there were significant increases in T2 for the proximal RF muscle at 72 hours and middle VM muscle at 48 hours (Maeo et al., 2018). Results from this MRI data show that heterogeneous damage may exist across a group and demonstrate that the modality of exercise may also lead to

variations of injury across synergists. However, these studies only partially address differences that may occur across muscles when undergoing eccentric contractions and do not explore the functional loss of strength for each muscle after injury.

Multiple characteristics among muscles of the same group may be able to explain potential differences in injury. There are significant differences in physiological cross-sectional area (PCSA), fiber length and pennation angle across muscles of the same group (Lieber & Fridén, 2000). As state earlier, it is theorized since architecture is different between muscles, fiber forces and strain patterns during eccentric contractions will also vary amongst these muscles (Lieber & Fridén, 2000). Differing torque-angle relationships across muscles may ultimately influence the likelihood of injury depending on the movement (de Brito Fontana et al., 2018; Han et al., 2019). In addition, there are differences in the level of recruitment for each of these muscles throughout the knee joint range of motion (Pincivero et al., 2004). The susceptibility to injury for a single skeletal muscle may be influenced by differences based on fiber type, previous usage and exercise as well as whether the muscle crosses one joint (monoarticular) or multiple joints. Patterns of differential injury may also differ between persons based on some of these factors. In addition, the modality of movement taking place can alter the activation of muscle fibers across a muscle or muscle group making them more susceptible to injury (Maeo et al., 2018). It is important that future research investigating injury utilize methods that directly measure individual muscle forces to compare if force deficits are similar across a synergist skeletal muscle group.

## Differential Muscle Injury Hypothesis

In the field of exercise-induced injury, many questions must be addressed pertaining to synergist muscle groups. The research referenced in this review suggests that there could be heterogeneous injury across muscles of a synergist group due to differences in architecture, torque/angle relationships, levels of activation through movement and many other characteristics. These differences across muscles likely mean that strain and stress during eccentric contractions will vary across the fibers of the same group and thus result in different degrees of injury within muscles of a group. If this hypothesis is correct, there would be significant differences in strength deficits and soreness between the individual muscles of a synergist group. Would these strength deficits be attributed to the failure of the nervous system to activate skeletal muscle or due to E-C coupling failure within injured myofibers? There is also the question of how the body would adjust its motor plan and how that may affect limb stability while standing and during locomotion? It is also unclear whether the muscle weakness and/or altered muscle fiber recruitment associated with grade I injuries contributes to the development of secondary tissue injuries. Although changes in gait mechanics (i.e., stride length and frequency) during locomotion are seen following eccentric contraction-induced muscle injury (Paschalis et al., 2007), it is unknown how the continuation of daily physical activity while having a grade I injury can affect the susceptibility for a secondary musculoskeletal injury. Moreover, muscle weakness and altered tendon strain patterns are known risk factors associated with the development of overuse musculoskeletal injuries (e.g., tendinopathies) (Reeves et al., 2005; Dillon et al., 2008; Verrelst et al., 2014; Scott et al., 2014). We suggest a full investigation into the potential for differential injury across synergist skeletal muscle groups and potential adaptations that may occur in response to that injury. Current evidence suggests that synergistic muscles experience

heterogeneous changes in swelling and contractile properties after injury. It remains unknown whether there are differential strength deficits across the synergistic muscles and whether that impacts motor unit recruitment or activation during both submaximal and maximal contractions.

### CHAPTER THREE

### **METHODS**

## Subjects

After providing informed consent, potential subjects were screened using a health assessment form to ensure that they were free of contraindications to exercise and did not have a history of traumatic lower body injuries such as ligament tears. Subjects recruited for our study were sedentary or recreationally active males between 18 to 35 years of age. Sedentary is operationally defined as spending most of the day in activities requiring minimal energy expenditure or sitting/lying and failing to achieve the American College of Sports Medicine's (ACSM) recommended weekly amount of physical activity (i.e., at least 150 minutes/day of moderate physical activities and/or 75 minutes of vigorous physical activity) at any point in the past six months. Recreationally active was defined as those who participate in light and/or moderate intensity exercise ≤2 days per week with exercise sessions lasting 30 minutes or less. Subjects who were recreationally active but performed resistance or plyometric exercise involving the lower body, and/or those who participated in downhill running were excluded from the study. Subjects who are required to have medical clearance for exercise after completing the ACSM exercise preparticipation screening were excluded from the study.

#### Experimental Design

On Day 0 of data collection, the subject reported for the initial screening and provided informed consent prior to completing any screening information. The subject then filled out the health history questionnaire and was screened for suitability to continue the study. Researchers were not blinded to the study however all experimental procedures were standardized for all subjects and

performed by the same researcher. Cleared subjects then had height, weight and anthropometric measurements of the legs conducted first. The subject was randomly placed into either the downhill run protocol (INJ) or control exercise protocol (CON). The subject was then familiarized with the muscle soreness, knee pain, and limb circumference measurements. Then the subject had reflective markers and EMG electrodes placed on their lower limbs before undergoing the postural sway assessment. Following this assessment, the subject was instructed to walk at their "normal walking pace" between two markers that are placed 10 meters apart. Using the average time of three walking trials, walking velocity was calculated to be used during locomotion assessments and the control group experimental protocol. A minimum of at least 2.0 mph for walking speed was set. Subjects then jogged on the treadmill for roughly 5 minutes while wearing a HR monitor. The jogging speed corresponding to roughly 70% of the subject's age-predicted maximum heart rate (HRmax) at a steady state was used for locomotion assessments and the experimental protocol of the injury group. The subject was then familiarized with the maximal voluntary contractile (MVC) and individual muscle strength assessments which were recorded on a Biodex dynamometer (Biodex Medical Systems, Inc., Shirley, NY). All settings for the Biodex dynamometer were logged and used for all subsequent testing. Following the familiarization protocol, the subject was scheduled for two remaining data collection sessions.

On Day 1, the subject returned to the lab and was measured for baseline (Pre) muscle soreness, knee pain and limb circumference. Then EMG electrodes were applied over the vastus medialis, rectus femoris, vastus lateralis, bicep femoris, tibialis anterior and soleus muscles and secured by athletic wrap. EMG data were collected to estimate levels of muscle activation during all

assessments. To prepare the skin for electrode placement, the subject was provided with a razor to remove hair from the areas where electrodes were to be placed. To increase conductance, sandpaper was rubbed on the skin of the application sites to help remove dead skin cells and then wiped with a rubbing alcohol pad. To ensure consistency of electrode placement throughout the study, the researchers outlined the electrode placement using indelible marker. EMGs electrodes were secured by athletic wrap to secure their placement on the subject. The subject then completed their pre-injury balance assessment. Then the subject completed the pre-injury locomotion assessments where EMG was recorded during three trials of walking and running on the treadmill. EMG electrodes were removed from the tibialis anterior, gastrocnemius medialis, biceps femoris and the quadriceps group on the dominant leg following the locomotion assessment. The subject was then assessed for maximal voluntary knee extensor strength on the Biodex. The researcher removed the EMG electrodes on the non-dominant quadriceps muscles before testing the subject's individual muscle strength via electrical stimulation on the nondominant leg. Once the baseline (Pre) testing was complete, the subject then performed the 60minute downhill running injury protocol or the 30-minute flat walking control exercise protocol based on their group assignment. After a 5 to 10-minute break following the completion of the experimental protocol, subjects were measured for post-injury (Post) muscle soreness, knee pain and limb circumference. The subject had all EMGs reapplied then underwent the post-injury postural sway and locomotion assessments. Post-injury strength assessments concluded the data collection for Day 1. The subject then was instructed to return to the lab in 48 hours for Day 2 of data collection. On Day 2, the subject was measured for post-injury muscle soreness, knee pain and limb circumference. EMG electrodes were reapplied before completing the participant's posture assessment plus walking and running assessments. The subject was then placed on the

Biodex for the 48 hours post-injury MVC strength and individual knee extensor torque

assessments. This concluded all necessary testing and data collection for the subject in this study.

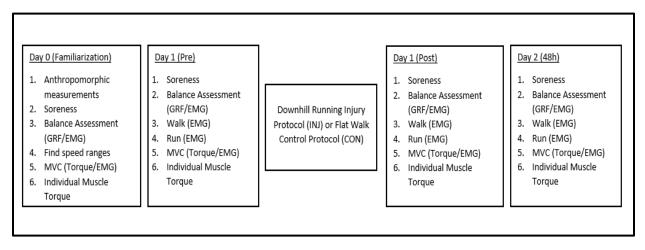


Figure 3.1 Timeline of experimental design.

Muscle Soreness and Knee Pain

Total quadricep muscle soreness and anterior knee pain was evaluated by having the subjects step onto and off a 12 inch box for 3 separate repetitions on each leg. Soreness and pain are subjective measures, so the subjects indicated their level of soreness/pain by placing a tick mark on a scale from 1-100 with 0 indicating the absence of soreness/pain and 100 indicating the greatest amount of soreness/pain. Muscle soreness and knee pain were measured three times per time point and the average was reported.

Perceived soreness of individual knee extensor muscles was measured using a myometer using procedures similar to Newham et al., 1983. The force transducer of the myometer (Manual Muscle Tester, Lafayette Instrument) was applied four times at equal distances across the vastus medialis, rectus femoris and vastus lateralis while the subject was in a supine position. The myometer was set to alarm at 45N of force when applied to each site. The subject is then asked to rate the pain from a 1 to 100 mm visual analog scale with 0 indicating the absence of soreness and 100 indicating the greatest amount of soreness. The average of the four sites served as an overall soreness score for a given knee extensor muscle.

#### Thigh Circumference

Thigh circumference was assessed at the greatest girth of the thigh of both legs with an anthropometric tape. Subjects were asked to stand in a fully relaxed anatomical position before being instructed to put all their weight on the opposite leg while measurements were taken. Measurement sites were marked with indelible marker to ensure consistent measurements and the averages are reported.

#### Balance Assessment with Posturography

Balance during quiet stance was quantified by measuring spontaneous sway as the participant stood on two side-by-side force plates. A trial of 30 seconds was tested for each of the three following conditions: eyes-open (EO), eyes-closed (EC), and eyes-open while standing on a block of compliant foam (FP) (10-cm thick, Aeromat Fitness Product, CA). The eyes-open condition will evaluate the subjects' balance with the three main sensory systems (i.e., vestibular, visual, somatosensory) that are used for standing balance. The eyes-closed condition evaluated the dependence on the visual sensory system and the foam pad condition evaluated the dependence on the somatosensory system while maintaining a standing posture. Subjects were told to remain as still as possible under all conditions with feet shoulder width apart and arms resting at the sides in a comfortable standing position and for eyes-open conditions, to look straight ahead at a marker located roughly 12 feet away. The bilateral ground reaction forces

were measured by the respective force plate. The center of pressure (COP) trace during each trial was determined by the filtered ground reaction force. The COP 95% trajectory area and total length was calculated. Proprioception quotient (PQ) and Romberg's quotient (RQ) was calculated using the COP area and trajectory length based on the methods previously described by Yang and Liu (2020) to determine the relative importance of vision and proprioception in maintaining standing balance during the study.

### Muscle EMG

Bilateral quadriceps muscle (VL; RF; VM), biceps femoris (BF), tibialis anterior (TA) and soleus (SOL) EMG data was recorded for three separate ten-second periods during locomotion trials. Additionally, non-dominant quadriceps EMG data was recorded for approximately ten seconds during voluntary maximal isometric strength tests. Muscle EMG was recorded using surface electrodes placed on the skin using double-sided adhesive tape and wrapping to keep electrodes in place. One EMG electrode was placed on the mid-belly of each quadricep, hamstring (BF), and calf muscles (TA; SOL). The EMG root mean square (RMS) and median frequency (MF) were measured from the raw data and used to determine the level of muscle activation during 3 seconds of maximal isometric contractions on the Biodex as well as the 3 separate 10-second trials of walking and running on the treadmill.

#### Locomotion Assessment

Participants began by walking for three minutes on the treadmill at the determined speed from Day 0 testing. This initial three minutes of walking was the participant's warm-up. The participant then continued to walk at the same self-selected speed for an additional three minutes. Leg muscle EMG data was recorded during the last ten seconds of the final three minutes. The

walking speed remained constant throughout the testing period. Immediately after the walking trials, subjects then began to run on the treadmill at the speed that corresponds to 70% of their age-predicted maximum heart rate determined from Day 0 testing. Subjects warmed-up at the speed for 3-minutes followed by 3-minutes of data collection. Running speeds for each participant remained constant throughout the testing period. Leg muscle EMG data was recorded during the last 10 seconds of the final three minutes.

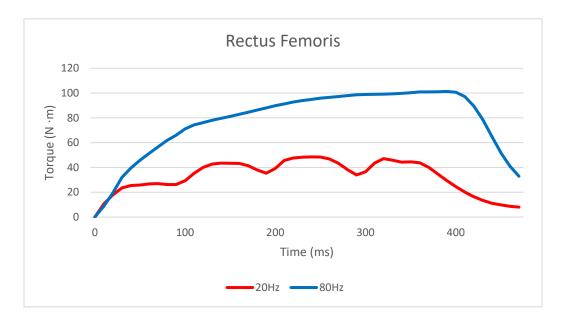
#### Maximal Voluntary Contraction Torque Assessment (Biodex)

Maximal voluntary contractile muscular strength of the non-dominant quadriceps muscles was evaluated using the Biodex dynamometer. Subjects were strapped into the Biodex using a seatbelt and chest harness. A padded strap connected to the dynamometer lever arm was wrapped directly proximal to the subject's ankle on their non-dominant leg. Subjects performed three maximal voluntary isometric contractions at 0°/sec with the leg at 20°, 45° and 90° of knee flexion. Each contraction lasted for three seconds and one minute of recovery was given after each contraction. Maximal voluntary torque is defined as the average of the greatest torque value achieved from the three contractions at each joint angle. EMG data collection from the non-dominant quadriceps muscles began five seconds prior to the contraction and concluded three seconds following.

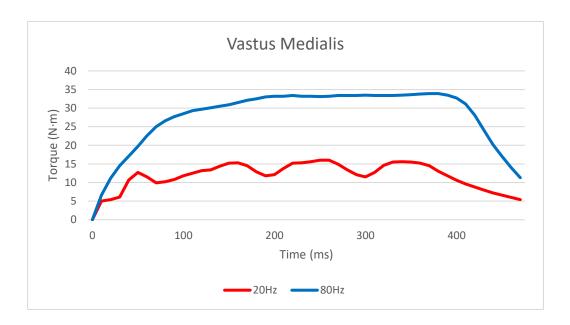
### Direct Electrical Stimulation of Quadriceps Muscle

Electrically induced torque produced by three superficial knee extensor muscles (vastus lateralis, vastus medialis, rectus femoris) on the non-dominant leg was evaluated using the Biodex dynamometer. Directly stimulated muscle tetanus is a widely used method in order to determine changes in muscle activation and force development after various types of injury and fatigue in

humans (Brown et al., 1996; Mackey et al., 2016; Kamandulis et al., 2017/2019). The use of the Biodex for the collection of individual muscle torque was determined by a reliability analysis conducted in our Pilot study (APPENDIX A). The hair on the leg was shaved, and to increase conductance the skin was rubbed with sandpaper and alcohol prep pads. Pairs of stimulating electrodes separated by 3 cm were placed on the skin over one of the quadriceps muscles at a time. Muscles were stimulated by the voltage and current settings of a Digitimer (model DS7AH) electrical stimulator, while the stimulation frequency (20 Hz and 80 Hz) and duration (0.4 s) was set by the "Sync Train" output of a Grass S48 electrical stimulator that is connected to the trigger input of the Digitimer stimulator. Permanent markers were used to trace the location of stimulating electrodes for multi-day analyses. The subjects were seated and strapped to the Biodex chair and asked to completely relax between contractions to relieve muscle tension. A padded strap connected to the dynamometer lever arm was wrapped directly proximal to the subject's ankle on their non-dominant leg. The subjects also wore headphones and listened to the content of their choice during the assessment as not to anticipate the stimulations based on auditory indications. The output current from the Digitimer was set to 200mA and 200mV. After the first series of 3 low-frequency tetanic contractions at 20 Hz, each muscle was stimulated at a frequency 80 Hz for 2 repetitions. The order of quadriceps muscle activation was randomized for each subject. All stimulations were separated by 60 seconds of rest.



b)



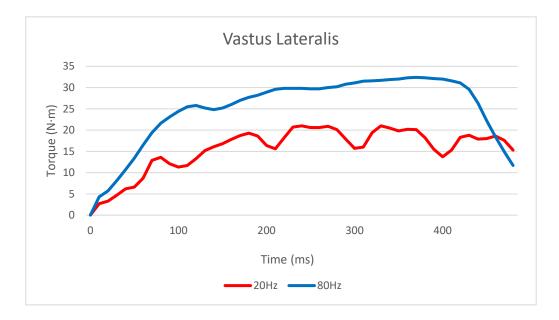


Figure 3.2 a) Torque produced by the rectus femoris during low-frequency stimulation (20 Hz) and high-frequency stimulation (80 Hz). b) Torque produced by Vastus Medialis during low and high frequency stimulation. c) Torque produced by Vastus Medialis during low and high frequency stimulation. Data collected on a Biodex dynamometer during pilot study. Data shown was collected from a single subject during the pilot study (Appendix A).

## **Downhill Running Protocol**

c)

Eight subjects performed the downhill running protocol (INJ) on Day 1. The downhill running protocol is designed to induce injury through a series of repeated eccentric contractions. Subjects completed a total of 60 minutes of downhill running on a -14% at a HR range of 70-85% of their age-predicted HR max. Subjects were offered a break every 10-15 minutes, each break lasting a maximum 5 minutes.

# **Control Exercise Protocol**

Seven subjects performed the control walking protocol (CON) on Day 1. Reduced times of level treadmill walking were chosen to serve as an exercise control that would likely minimize the risk of recreational participants from experiencing muscle injury from running for 60 minutes.

Subjects completed 30 minutes of level walking (0%) at a speed that was calculated based on their normal walking pace for 10 meters but no lower than 2.0 mph. Subjects were offered a break every 10-15 minutes, each break lasting a maximum 5 minutes.

### Statistics

Independent Samples T-Tests were used to compare group characteristics. Un-pooled variances and a correction to the degrees of freedom was implemented to determine the p-value when Levene's test for Equality of Variances was violated. A ratio of a muscle's capacity of producing torque following injury (T2) to the torque produced prior to injury (T1) was used to evaluate individual muscle force before and following the experimental protocol. This formula is calculated as: r = T2 / T1. From this ratio we can thus determine variability between the three muscles. With this variability metric, we can capture the heterogeneity of the impact of the injury protocol on the torque production capacity amongst the muscles of the quadriceps group. Group (INJ vs. CON) by time (Post/Pre ratio, 48H/Pre ratio) by muscle (VL, VM, RF) by frequency (20 vs 80 Hz) analysis of variance (ANOVA) with repeated measures on time, was used to evaluate changes in individual muscle torque. The same analysis with the Immediate Post/Pre and 48H/Pre torque ratios were also run. Separate group (INJ vs. CON) by time (Post/Pre ratio, 48H/Pre ratio) by muscle (VL, VM, RF) by leg (Dominant vs Non-dominant) ANOVA with repeated measures on time, was used to evaluate changes in individual muscle soreness. Muscle activation (EMG RMS and MF) during the locomotion trials was evaluated using a group (INJ vs. CON) by time (Pre, Post, 48H) by muscle (VL, VM, RF) by leg (Dominant vs Nondominant) ANOVA with repeated measures on time. Muscle activation (EMG RMS and MF) during the MVC trials was evaluated using a group (INJ vs. CON) by time (Pre, Post, 48H) by

muscle (VL, VM, RF) by angle (20°, 45°, 90°). Separate group (INJ vs. CON) by time (Pre, Post, 48h) by leg (Dominant vs Non-dominant) ANOVA with repeated measures was used to evaluate changes in total muscle soreness, knee pain, and thigh circumference. Balance was evaluated by using a group (INJ vs. CON) by time (Pre, Post, 48H) by condition (EO vs. EC vs. FP; RQ vs. PQ) ANOVA with repeated measures. Shapiro-Wilk tests revealed some conditions of MVC, individual stimulation and muscle activation had failed (p < 0.05) normality assumptions. However further analysis indicated little to moderate skewness (<0.7) and minimal kurtosis (< 1.3) for all data. It has been previously determined that analysis of variance and subsequent testing is robust to violations of normality assumptions (Montgomery, 2017; Schmidt & Finan, 2018; Tsagris & Pandis, 2021). Therefore, given the nature of the primary research question requires a statistical design (i.e., at least a group by muscle by time ANOVA with repeated measures on time) that does not allow for non-parametric testing, we report the statistical results of the parametric tests. In the event of significant statistical interactions, simple main effects analyses and Bonferonni's post hoc tests were performed when indicated. Simple linear regression was used to determine the degree of association between variables. An α-level was set at 0.05. Data was processed using Excel (Microsoft Office 2021) and MATLAB (Matlab R2021b, MathWorks, Natick, MA, USA) with statistical analysis conducted using SPSS (IBM) version 27 and Excel. Values for the results are reported as mean  $\pm$  SD for subject descriptive data and mean  $\pm$  SE for all other analyses. If a subject did not have useable baseline data for an assessment, then they were not included in that analysis. Missing data from Post and 48H was estimated by using the relative change for the group at that time and multiplying it by the subject's baseline value.

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### CHAPTER FOUR

#### RESULTS

# **Subjects**

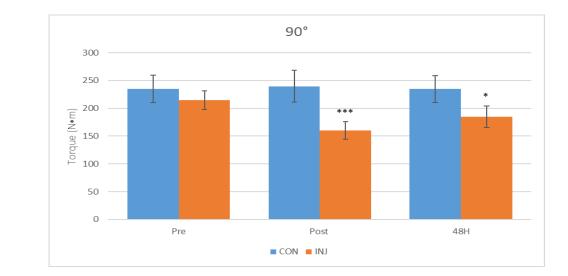
A total of 15 subjects completed the research protocol (Injury = 8, Control = 7) with one subject in the injury group and one subject in the control group completing the pre and post-assessments but could not attend the 48-hour follow-up assessments. For these subjects, 48H mean scores are estimated by multiplying the subject's baseline (Pre) value with the group's mean percentage change from baseline at 48H. One control subject's stimulated torque data was not included due to uncontrollable movement during the baseline assessment. The average age of the subjects was  $24.7 \pm 5.7$  years. The injury group was younger (p = 0.01) than the control group (CON =  $28.4 \pm$ 4.5 y/o, range = 22.0-33.0; INJ =  $21.4 \pm 4.5$  y/o, range = 18.0-30.0). Although subject age was correlated with baseline MVC torque ( $r \le 0.550$ ;  $p \ge 0.038$ ), age was not correlated to baseline 20 and 80 Hz muscle torque (r  $\le$  0.239; p  $\ge$  0.410) nor torque deficits for the injured group (r<sup>2</sup>  $\le$ 0.112;  $p \ge 0.418$ ). The average BMI of the subjects was  $26.9 \pm 5.0 \text{ kg/m}^2$  with no significant difference in the BMI between the two groups (p = 0.451, CON =  $28.4 \pm 3.6$  kg/m<sup>2</sup>, INJ =  $25.5 \pm$ 5.9 kg/m<sup>2</sup>). The average Q-angle for the subjects was  $12.3 \pm 1.7^{\circ}$  on the non-dominant (ND) leg and  $12.2 \pm 2.0^{\circ}$  on the dominant (D) leg. There was no significant difference in Q-angle for either leg between the two groups ( $p \ge 0.41$ , CON ND =  $11.7 \pm 0.9^{\circ}$ , CON D =  $12.3 \pm 1.7^{\circ}$ , INJ ND =  $12.3 \pm 2.2^{\circ}$ , INJ D =  $12.7 \pm 2.3^{\circ}$ ). Mean HR during 60 minutes of downhill running was 157.8 bpm or 79.4% of the age-predicted HR max of the injury group's mean age.

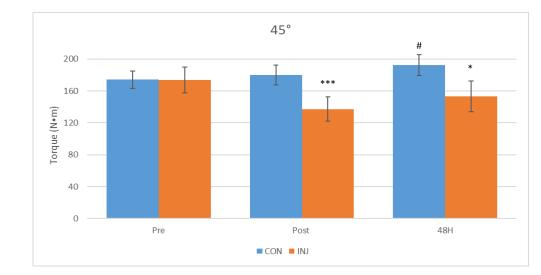
	CON	Range	INJ	Range	p-value					
Age (y/o)	28.4 (4.5)	22.0-33.0	21.4 (4.5)	18.0-30.0	p = 0.010					
Height (cm)	178.2 (6.6)	168.4-185.5	178.5 (5.5)	173.5-184.5	p = 0.926					
Weight (kg)	90.2 (13.1)	73.0-108.4	81.4 (19.2)	63.2-108.9	p = 0.324					
BMI (kg/m <sup>2</sup> )	28.4 (3.6)	23.9-32.7	25.5 (5.9)	17.4-32.2	p = 0.281					
ND Q-Angle (deg)	11.7 (0.9)	10.5-13.0	12.3 (2.2)	9.0-15.0	p = 0.411					
D Q-Angle (deg)	12.3 (1.7)	9.0-14.0	12.7 (2.3)	9.0-15.0	p = 0.786					
Values are means (SD	Values are means (SD). CON, control group. INJ, injury group. ND, non-dominant leg. D, dominant									
leg										

Table 4.1 Subject Characteristics

## **MVC of Knee Extensor Muscles**

Changes in knee extensor strength are shown in Figure 4.1. The Angle by Time by Group ANOVA of MVC torque determined a significant three-way interaction (p = 0.30). Baseline knee extensor strength was not different between the control and injury groups at 90° (p = 0.531), 45° (p = 0.979) and 20° (p = 0.905). The only change in Control MVCs occurred at 48H with 45° torque being significantly greater (p = 0.044) than baseline. All other control torque values were unchanged ( $p \ge 0.411$ ). The injury group experienced strength deficits ( $p \le 0.002$ ) at 90° (-25.3%), 45° (-21.0%) and 20° (-22.8%) immediately following their downhill run. Knee extensor strength for the injury group remained significantly lower ( $p \le 0.015$ ) than baseline values 48 hours later at 90° (-14.0%), 45° (-11.8%) and 20° (-14.2%). Immediate Post and 48H MVC torques were not different ( $p \ge 0.148$ ) for the injury group.





C)

B)

A)

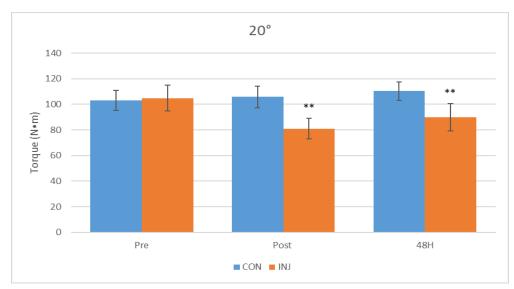


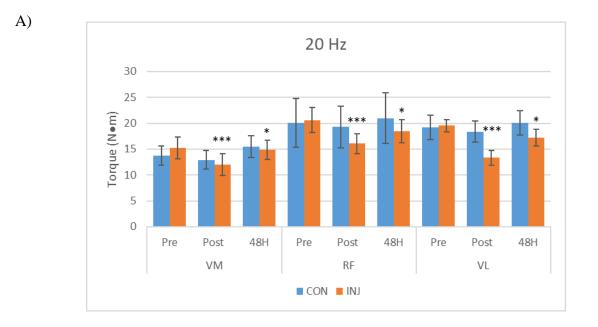
Figure 4.1 Maximal voluntary knee extensor torque on the non-dominant leg before (Pre), immediately following (Post), and 48 hours (48H) following either 30 minutes walking on the treadmill (CON) or 60 minutes of downhill running (INJ). Values are mean  $\pm$  SEM. \*, Significantly lower than Pre (p  $\leq$  0.05). \*\*, Significantly lower than Pre (p  $\leq$  0.01). \*\*\*, Significantly lower than Pre (p  $\leq$  0.05).

#### Individual Knee Extensor Muscle Torque

The mean low-frequency (20 Hz) stimulation torque and normalized ratio for the individual knee extensor muscles at all time points are displayed in Figure 4.2. The mean high-frequency (80 Hz) stimulation torque and normalized ratio for the individual knee extensor muscles at all time points are displayed in Figure 4.3. The Group by Muscle by Frequency by Time ANOVA of the stimulated (i.e., 20 and 80 Hz) muscle torque indicated a significant Group by Time interaction. Baseline torque (combined 20 and 80 Hz) collapsed across all muscles was not different (p = 0.811) between groups. There were no differences ( $p \ge 0.415$ ) in stimulated torque (combined 20) and 80 Hz) collapsed across all muscles for the control group at any time point. There was a 13.2% decrease (p < 0.001) in stimulated torque (combined 20 and 80 Hz) collapsed across all muscles for the injury group immediately post and a decrease (p = 0.018) of 9.1% at 48H following the downhill run. No muscle interactions with group or time were found ( $p \ge 0.268$ ). There was a significant (p = 0.01) Muscle by Frequency interaction that revealed stimulated 80 Hz torque was greater (p < 0.001) than 20Hz torque in all three muscles. The VL produced greater (p = 0.041) torque (i.e., uninjured and injured) than the VM at 80 Hz and trended towards greater (p = 0.07) 20 Hz torque. There was a trend for the RF producing greater (p = 0.082) torques at 80 Hz than the VM.

When normalized to pre-exercise values, the Group by Muscle by Frequency by Time ANOVA of the stimulated torque (20 and 80 Hz) did not produce any significant 3 or 4-way interactions

( $p \ge 0.095$ , Observed Power  $\le 0.467$ ). There was a significant main effect for group (p = 0.005) with the injury group having lower normalized stimulated torque (average of 20 and 80 Hz) across muscles and timepoints (CON =  $1.01 \pm 0.014$ ; INJ =  $0.87 \pm 0.014$ ). There was a significant Frequency by Time interaction which revealed 20 Hz normalized stimulated torque was lower (p < 0.001) immediately post exercise than at 48H following exercise collapsed across muscles and groups (Post =  $0.837 \pm 0.026$ ;  $48H = 0.980 \pm 0.025$ ). Immediate post exercise normalized ratios were lower (p = 0.002) at 20 Hz compared to 80 Hz (20 Hz =  $0.837 \pm 0.026$ ;  $80 \text{ Hz} = 0.952 \pm 0.015$ ) but were not found to be different (p = 0.184) at 48H (20 Hz =  $0.980 \pm 0.025$ ; 80 Hz =  $0.950 \pm 0.015$ ). No statistical difference across muscles were found ( $p \ge 0.059$ ; Observed Power  $\le 0.556$ ).



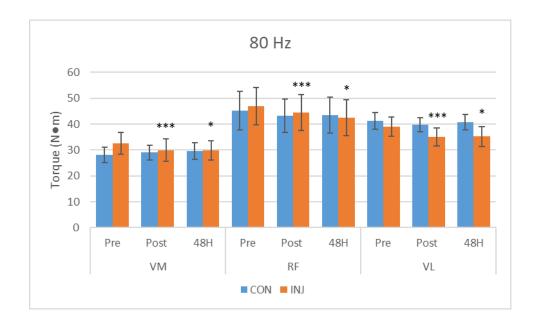
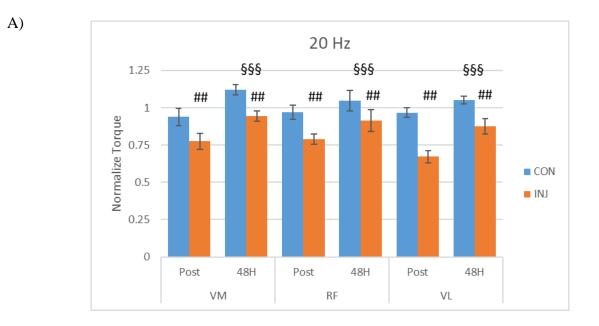


Figure 4.2 A) 20 Hz and B) 80 Hz mean data for individual muscle torque for the control and injury group across the three knee extensor muscles (VM, RF, VL). Values are mean  $\pm$  SEM. \*, Significantly less than Pre (p  $\leq$  0.05). \*\*\*, Significantly less than Pre (p  $\leq$  0.001).



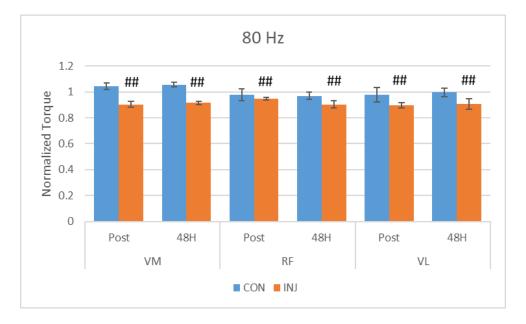


Figure 4.3 A) 20 Hz and B) 80 Hz normalized to Pre ratio for individual muscle torque for the control and injury group across the three knee extensor muscles (VM, RF, VL). Values are mean  $\pm$  SEM. ##, Significantly less than Control (p  $\leq$  0.01). §§§, Both groups significantly greater than Immediate Post/Pre (p  $\leq$  0.001).

### 20/80 Hz Torque Ratio

B)

A summary of the 20 Hz/80 Hz torque ratio is displayed in Figure 4.4. There were no significant group differences (p = 0.533) in the baseline 20 Hz/80 Hz torque ratio. There were no statistical differences ( $p \ge 0.112$ , Observed Power = 0.553) in the 20/80 Hz ratio amongst the muscles for either group. Compared to pre-exercise, there were no changes ( $p \ge 0.424$ ) in the 20/80 Hz torque ratio immediately after and 48 hours following walking on the treadmill. However, the 20/80 Hz torque ratio was greater (p = 0.017) at 48H than immediately post in the control group. Immediately after the downhill run, the 20/80 Hz torque ratio decreased (p = 0.001) by 19.6% across all muscles. Although there was no muscle interaction ( $p \ge 0.112$ ), the decrease in 20/80 Hz torque ratio for the VL (25.7%) tended to be greater than decreases in the VM (15.0%) and RF (17.2%) immediately following the downhill run. At 48H, 20 Hz/80 Hz torque ratio returned to baseline values for the injury group (p = 1.0).

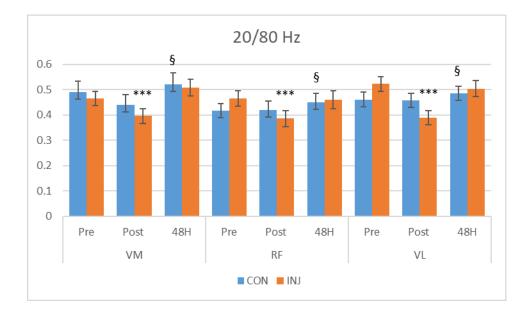


Figure 4.4 20/80 Hz torque ratio for both groups at baseline (Pre), immediately following (Post), and 48 hours (48H) following either 30 minutes walking on the treadmill (CON) or 60 minutes of downhill running (INJ). \*\*\*, Significantly less than Pre and 48H ( $p \le 0.001$ ). §, Significantly greater than Post ( $p \le 0.05$ ).

### **Quadriceps Muscle Soreness and Knee Pain**

Knee pain data is shown in Table 4.1. There was no significant difference in knee pain between groups at any time point in the study ( $p \ge 0.382$ ). Global knee extensor muscle soreness is displayed in Figure 4.5. Global knee extensor soreness was not different (p = 0.565) between both groups before the experimental protocols. There was no difference in global soreness between legs (p = 0.532). There was no difference (p = 1.0) in global quadriceps muscle soreness for the control group at any time point in the study. Muscle soreness increased (p = 0.046) immediately following downhill running (INJ Pre =  $0.5 \pm 0.22$  mm, INJ Post =  $8.7 \pm 3.47$  mm) and increased (p < 0.001) further at 48H (INJ 48H =  $14.9 \pm 1.66$  mm).

Table 4.2 Knee Pain (mm) collapsed across both legs								
	Pre	Post	48H					
CON	0.5 (0.4)	2.0 (0.9)	1.2 (0.8)					
INJ	0.4 (0.2)	4.9 (3.5)	3.8 (1.7)					
Values are means (SEM). CON, control group INJ, injury								
group								

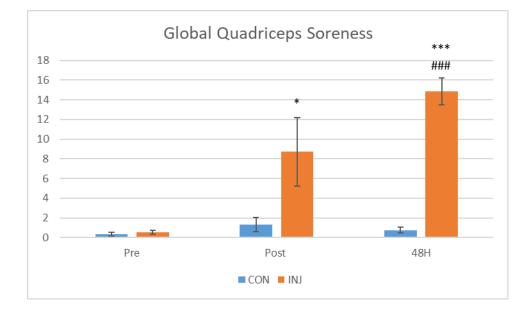


Figure 4.5 Global quadriceps muscle soreness values collapsed across legs. Values are mean  $\pm$  SEM. CON, control group. INJ, injury group. \*, Significantly greater than Pre (p  $\leq$  0.05). \*\*\*, Significantly greater than Pre (p  $\leq$  0.001). ###, Significantly greater than Control (p  $\leq$  0.001)

Figure 4.6 displays the mean soreness scores for individual knee extensor muscles. There was no significant change ( $p \ge 0.350$ ) in soreness for the control group for any muscle at any time point. Pre and immediately post soreness scores were similar ( $p \ge 0.086$ ) between the two groups. Immediately following 60 minutes of downhill running, VM soreness increased (p = 0.039, INJ VM Pre =  $0.2 \pm 0.07$  mm, INJ VM Post =  $2.4 \pm 0.86$  mm). However, soreness in the RF and VL were not different ( $p \ge 0.206$ ) from baseline immediately following the downhill run. At 48H, the injury group had significantly higher ( $p \le 0.004$ ) soreness scores in all muscles compared to

baseline values (VM: Pre =  $0.2 \pm 0.07$  mm,  $48H = 5.8 \pm 1.4$  mm; RF: Pre =  $0.3 \pm 0.1$  mm,  $48H = 5.9 \pm 1.0$  mm; VL: Pre =  $0.3 \pm 0.1$  mm,  $48H = 9.9 \pm 1.2$  mm). The injury group experienced greater (p  $\leq 0.004$ ) soreness in the VL than both the VM and the RF at 48H with no statistical difference (p = 1.0) between the VM and RF. Quadriceps muscle soreness was not different between legs at any time point (p  $\geq 0.124$ ).

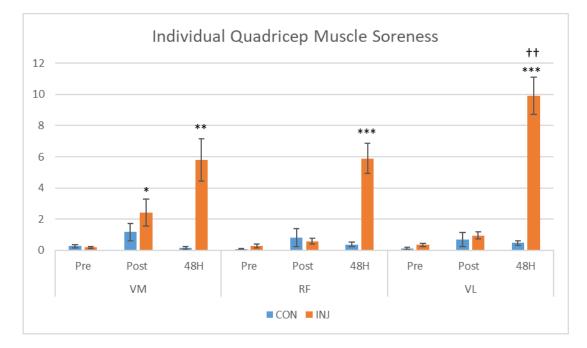


Figure 4.6 Individual quadriceps muscle soreness values collapsed across legs. Values are mean  $\pm$  SEM. \*, Significantly greater than Pre (p  $\leq$  0.05). \*\*, Significantly greater than Pre (p  $\leq$  0.01). \*\*\*, Significantly greater than Pre (p  $\leq$  0.001). †† Significantly greater than VM and RF (p  $\leq$  0.01).

## **Knee Extensor Activation During MVC**

Table 4.2 displays the mean EMG RMS and MF for the non-dominant knee extensors during MVCs at the three measured joint angles. Before the experimental protocols there were no differences ( $p \ge 0.432$ ) in EMG RMS and MF between groups for MVCs at all three angles. In the control group there was no difference (p = 1.0) in EMG RMS amongst the three time points, for all three knee joint angles. Control MVC MF decreased (p = 0.045) at 48H when collapsed across all three angles (CON Pre =  $120.7 \pm 5.8$  Hz, CON 48H =  $113.2 \pm 4.3$  Hz). In the injury

group, there were no differences in EMG RMS at any time point for MVCs at 20° and 45° ( $p \ge 0.735$ ). However, at 90° EMG RMS decreased ( $p \le 0.038$ ) by 16.8% at post and remained under baseline levels at 48H for the injury group (Pre =  $120.5 \pm 20.0 \mu$ V, Post =  $100.3 \pm 18.3 \mu$ V, 48H =  $100.3 \pm 14.1 \mu$ V). There were no differences ( $p \ge 0.246$ ) in EMG RMS amongst muscles at any time point. There was no difference ( $p \ge 0.425$ ) in MVC MF at any time point for the injury group.

# **Correlation of MVC Torque Loss**

Linear regression analyses of post-exercise (i.e., Post and 48H) MVC 90° torque are shown in Figure 4.7. The greatest correlating factor with post-exercise MVC 90° torque was the change in EMG RMS at 90° (r = 0.663, p < 0.001). Changes in 20 Hz (r = 0.480, p = 0.013) and 80 Hz torque ( $r^2 = 0.447$ , p = 0.021) were both significant factors for explaining variations in postexercise MVC 90° torque. 20/80 Hz torque ratio was not a statistically significant factor for explaining changes in post-exercise MVC 90° torque (r = 0.332, p = 0.095).

90°)								
			RMS (µV)		MF (Hz)			
		Pre	Post	48H	Pre	Post	48H	
CON	VM	125.0 (25.1)	111.8 (17.2)	113.1 (17.3)	112.1 (6.3)	112.9 (6.2)	101.0 (6.1)*	
	RF	79.3 (10.7)	85.6 (11.2)	89.4 (12.9)	86.2 (5.7)	86.0 (6.2)	82.6 (4.6)*	
	VL	80.3 (20.7)	85.0 (17.4)	82.8 (18.4)	124.9 (10.6)	122.5 (9.3)	110.2 (12.2)*	
INJ	VM	196.5 (44.1)	156.8 (41.6)*	156.6 (27.0)*	89.4 (4.1)	101.6 (6.8)	93.6 (5.2)	
	RF	91.5 (20.0)	78.2 (21.8)*	80.4 (17.7)*	101.3 (7.0)	93.4 (2.9)	101.5 (7.9)	
	VL	73.4 (11.9)	65.8 (14.3)*	64.0 (11.8)*	135.2 (6.7)	133.6 (3.2)	138.3 (4.3)	

Table 4.3 Root mean square and Median Frequency of non-dominant knee extensors during MVCs at 90°, 45° and 20°

45°)

			RMS			MF	
		Pre	Post	48H	Pre	Post	48H
CON	VM	95.3 (17.6)	94.4 (14.5)	100.1 (11.7)	112.2 (6.6)	113.0 (7.8)	107.5 (7.0)*
	RF	64.4 (11.8)	65.2 (11.5)	66.5 (11.8)	107.0 (6.6)	101.2 (7.7)	100.8 (5.2)*
	VL	70.6 (18.7)	74.1 (14.8)	71.5 (18.7)	149.9 (11.4)	136.7 (13.8)	143.7 (10.4)*
INJ	VM	128.0 (26.9)	145.8 (47.5)	131.0 (28.5)	93.4 (4.8)	99.4 (5.9)	99.8 (4.9)
	RF	62.6 (14.5)	64.3 (16.9)	71.1 (19.8)	124.2 (9.1)	109.3 (3.2)	116.4 (5.8)
	VL	59.1 (13.1)	80.3 (26.8)	62.6 (16.9)	162.8 (8.6)	157.4 (5.4)	175.7 (4.3)

20°)

			RMS			MF	
		Pre	Post	48H	Pre	Post	48H
CON	VM	93.8 (16.0)	85.8 (9.6)	97.2 (11.1)	118.0 (7.9)	122.8 (8.1)	113.5 (6.2)*
	RF	62.0 (10.3)	61.5 (9.5)	60.9 (6.8)	113.6 (8.2)	110.4 (8.1)	107.9 (6.9)*
	VL	66.4 (13.1)	75.3 (11.4)	65.8 (12.3)	162.0 (12.1)	148.5 (11.5)	151.7 (10.4)*
INJ	VM	131.3 (24.2)	132.1 (33.5)	130.1 (25.8)	104.4 (5.1)	107.3 (5.7)	107.4 (3.4)
	RF	64.7 (13.5)	59.2 (11.3)	75.6 (20.4)	134.1 (8.8)	116.6 (2.7)	122.0 (3.6)
	VL	69.3 (15.1)	82.7 (22.7)	66.2 (15.5)	176.1 (9.9)	169.9 (4.6)	181.9 (3.7)
			ON, Control group.	. INJ, Injury group		-	

VM, Vastus Medialis. RF, Rectus Femoris. VL, Vastus Lateralis. \*, Significantly less than Pre ( $p \le 0.05$ )



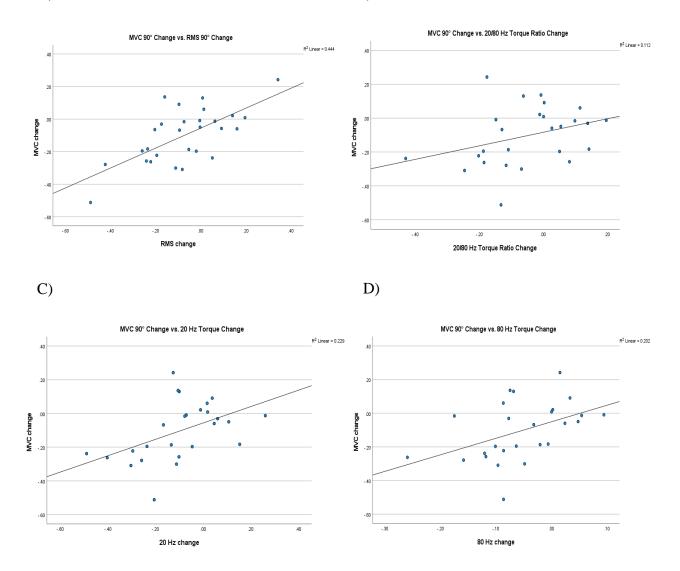


Figure 4.7 Linear regression analysis comparing MVC 90° torque change to A) EMG RMS MVC 90° change B) 20/80 Hz torque ratio change C) 20 Hz torque change and D) 80 Hz torque change. Coefficients of determination (r<sup>2</sup>) are indicated.

# Leg Muscle Activation During Locomotion

A summary of lower limb muscle activation during treadmill walking is displayed in Table 4.3. EMG RMS of the lower limb muscles did not change ( $p \ge 0.294$ ) during walking for the control group at any time point during the study. The injury group exhibited significantly greater ( $p \le$ 0.002) walking RMS of the TA immediately following the downhill running compared to both baseline and 48H (INJ Pre =  $20.7 \pm 1.0 \mu$ V, INJ Post =  $26.2 \pm 1.8 \mu$ V, INJ 48H =  $22.6 \pm 1.2 \mu$ V). Walking RMS for the injury group tended to increase (p  $\ge 0.294$ ) in the VM (17.9%), RF (35.8%), and VL (32.2%) immediately post downhill running.

EMG MF of the lower limb muscles did not change ( $p \ge 0.255$ ) during walking for the control group at any time point during the study. Walking EMG MF in the TA for the injury group was lower ( $p \le 0.002$ ) immediately following 60 minutes of downhill running compared to baseline and 48H values (INJ Pre = 161.4 ± 3.6 Hz, INJ Post = 147.6 ± 4.1 Hz, INJ 48H = 160.8 ± 3.6 Hz). Walking EMG MF remained unchanged ( $p \ge 0.139$ ) in the VM (0.6% decrease), RF (9.8% decrease), and VL (3.9% increase) immediately following the downhill run. At 48H, the EMG MF remained unchanged ( $p \ge 0.139$ ) in the VM (16.0% increase), RF (3.3% decrease) and VL (4.2% increase).

			RMS (µV)			MF (Hz)	
		Pre	Post	48H	Pre	Post	48H
CON	VM	7.2 (0.8)	6.9 (0.7)	6.2 (0.6)	114.6 (4.6)	114.6 (5.0)	115.6 (5.9)
	RF	3.9 (0.3)	3.6 (0.3)	3.1 (0.2)	124.4 (4.1)	118.4 (4.2)	115.6 (4.9)
	VL	8.2 (0.6)	8.6 (0.8)	8.1 (0.5)	144.0 (6.1)	141.0 (7.7)	145.1 (6.5)
	BF	7.4 (0.9)	8.3 (1.1)	7.2 (0.8)	154.4 (8.6)	163.9 (9.1)	165.9 (8.5)
	TA	26.9 (2.4)	26.5 (2.2)	26.1 (2.4)	142.3 (3.4)	143.3 (2.8)	137.9 (4.3)
	SOL	19.4 (1.5)	19.0 (1.3)	19.6 (1.5)	158.3 (4.1)	158.4 (3.5)	154.8 (3.1)
INJ	VM	8.1 (0.8)	9.6 (1.4)	9.4 (1.2)	103.4 (4.6)	102.8 (5.3)	119.9 (5.3)
	RF	3.9 (0.4)	5.2 (1.1)	4.8 (0.8)	120.9 (3.7)	109.1 (2.5)	116.9 (2.8)
	VL	8.0 (0.7)	10.6 (1.8)	8.4 (0.8)	160.3 (4.8)	166.5 (4.4)	167.0 (3.9)
	BF	8.4 (0.9)	8.5 (1.0)	8.6 (0.9)	161.6 (11.9)	157.0 (10.4)	161.9 (13.1)
	TA	20.7 (1.0)	26.2 (1.8)***	22.6 (1.2)	161.4 (3.6)	147.6 (4.1)***	160.8 (3.6)
	SOL	18.9 (1.5)	18.4 (1.5)	17.9 (1.4)	166.1 (2.6)	169.4 (2.5)	165.0 (2.3)

Table 4.4 Walking EMG root mean square and median frequency collapsed across both legs

Values are in mean ( $\pm$  SEM). CON, Control group. INJ, Injury group. RMS, Root Mean Square. MF, Median Frequency. VM, Vastus Medialis. RF, Rectus Femoris. VL, Vastus Lateralis. \*\*\*, Significantly greater than Pre ( $p \le 0.001$ )

A summary of lower limb muscle activation during treadmill running is shown in Table 4.4. There were no significant changes ( $p \ge 0.078$ ) in EMG RMS of the lower limb muscles for the control group at any time point during treadmill running. EMG RMS of the knee extensors was greater ( $p \le 0.027$ ) immediately after the downhill run than at baseline and 48H. There were no differences ( $p \ge 0.445$ ) in EMG RMS amongst the muscles. There was no statistically significant difference ( $p \ge 0.062$ ) in EMG RMS between legs. No other leg muscles experienced a statistically significant change in running EMG RMS for the injury group ( $p \ge 0.078$ ).

There was not any significant change ( $p \ge 0.144$ ) in EMG MF of the lower limb muscles for the control group at any time point during treadmill running. The EMG MF of the TA for the injury group was lower ( $p \le 0.017$ ) immediately following the downhill run compared to baseline and at 48H (INJ Pre =  $157.8 \pm 3.8$  Hz, INJ Post =  $148.6 \pm 3.8$ , INJ  $48H = 158.3 \pm 3.6$ ). There were no significant differences ( $p \ge 0.144$ ) in the running EMG MF amongst the three knee extensors immediately following the downhill run.

		U					U
			RMS (µV)			MF (Hz)	
		Pre	Post	48H	Pre	Post	48H
CON	VM	29.6 (3.7)	29.5 (4.5)	29.6 (3.6)	101.1 (5.6)	104.7 (5.7)	101.9 (4.9)
	RF	10.9 (0.7)	10.2 (0.8)	9.1 (0.6)	89.4 (2.3)	90.9 (2.7)	88.2 (2.4)
	VL	22.6 (1.9)	21.2 (1.8)	20.5 (1.6)	119.3 (6.0)	122.5 (6.7)	124.6 (6.2)
	BF	17.4 (1.4)	17.3 (1.7)	14.1 (0.6)	119.7 (4.7)	121.7 (5.3)	123.0 (4.9)
	TA	50.6 (6.1)	49.9 (5.9)	50.3 (6.3)	134.2 (3.6)	137.9 (2.7)	132.5 (3.8)
	SOL	32.6 (1.9)	30.9 (1.8)	30.6 (2.1)	133.1 (3.2)	135.9 (3.1)	134.8 (3.0)
INJ	VM	32.0 (4.0)	37.3 (4.8)*	31.3 (4.6)	93.6 (3.1)	91.2 (3.3)	97.9 (3.7)
	RF	12.6 (2.3)	16.4 (2.3)*	14.9 (3.9)	99.4 (4.5)	97.0 (3.2)	96.3 (2.7)
	VL	21.2 (2.3)	24.2 (2.7)*	21.5 (2.3)	141.8 (4.5)	149.2 (4.4)	154.5 (3.4)
	BF	15.6 (1.8)	16.6 (2.2)	17.1 (2.3)	116.9 (4.3)	119.6 (5.2)	114.8 (4.0)
	TA	45.5 (4.1)	49.6 (4.2)	47.9 (5.2)	157.8 (3.8)	148.6 (3.8) <sup>\$</sup>	158.3 (3.6)

Table 4.5 Running EMG root mean square and median frequency collapsed across both legs

### Balance

Changes in COP sway area and length for all three conditions are displayed in Table 4.5. COP sway area tended ( $p \ge 0.190$ ) to increase (90.8%) immediately following the downhill run. COP path length collapsed across all three conditions decreased (p = 0.017) at 48H for the control group (CON Pre = 9639 ± 379 mm, CON 48H = 8517 ± 168 mm) while it increased (p = 0.024) immediately after the injury group performed the downhill run (INJ Pre = 9902 ± 569 mm, INJ Post = 10113 ± 575 mm) but returned to baseline levels at 48H for the injury group (p = 1.0, INJ 48H = 9985 ± 586 mm). Changes in RQ and PQ are displayed in Table 4.6. Although there were no group differences ( $p \ge 0.208$ ) for PQ or RQ, PQ (Area) tended to increase by 40.2% immediately following the downhill run.

Table 4.6 COP Area and Length for EO, EC and FP conditions							
Area (mm <sup>2)</sup>		Pre	Post	48H			
CON	EO	66.1 (9.6)	84.3 (18)	81.00 (19.0)			
	EC	136.8 (42.3)	105.5 (21.6)	181.9 (57.5)			
	FP	270.5 (54.0)	227.9 (73.9)	129.4 (35.1)			
INJ	EO	100.4 (22.4)	147.6 (42.7)	86.9 (20.4)			
	EC	118.7 (23.4)	243.3 (91.4)	163.5 (29.6)			
	FP	196.7 (32.9)	411.8 (104.6)	214.3 (40.8)			
Length (mm)							
CON	EO	9646 (644)	9773 (751)	8808 (256)*			
	EC	10161 (601)	10187 (640)	8604 (307)*			
	FP	10127 (602)	9768 (726)	8462 (302)*			
INJ	EO	9899 (967)	10167 (987) <sup>\$</sup>	10102 (1025)			
	EC	10024 (1030)	10233 (1018) <sup>\$</sup>	10044 (1074)			
	FP	9783 (956)	9938 (979) <sup>\$</sup>	9810 (940)			

Table 4.6 COP Area and Length for EO, EC and FP conditions

Values are mean ( $\pm$  SEM). CON, Control group. INJ, Injury group. EO, Eyes-open. EC, Eyes-closed. FP, Foam Pad.\*, Significantly less than Pre ( $p \le 0.05$ ). \$, Significantly greater than Pre ( $p \le 0.05$ )

Table 4.7 Romberg's quotient and Proprioception quotient based on COP area and length

			Area			Length	
		Pre	Post	48H	Pre	Post	48H
CON	RQ	1.76 (0.22)	1.34 (0.29)	2.11 (0.6)	1.00 (0.01)	1.00 (0.01)	0.96 (0.02)
	PQ	4.00 (0.66)	2.96 (1.32)	1.54 (0.30)	1.01 (0.01)	0.95 (0.03)	0.94 (0.03)
INJ	RQ	1.82 (0.62)	1.80 (0.48)	2.22 (0.41)	1.01 (0.01)	1.01 (0.01)	0.99 (0.01)
	PQ	2.54 (0.68)	3.57 (0.67)	3.06 (0.71)	0.99 (0.01)	0.98 (0.00)	0.98 (0.01)
Values are mean (± SEM). CON, Control group. INJ, Injury group. RQ, Romberg's Quotient. PQ, Proprioception Quotient.							

### Thigh Circumference and Knee Joint Range of Motion

Table 4.7 lists thigh circumference and knee range of motion for control and injury groups across the three time points on both legs. There were no statistical differences ( $p \ge 0.154$ ) in thigh circumference between groups at any time. There were no statistical differences ( $p \ge 0.760$ ) between knee joint range of motion between the groups. Knee ROM at 48H was greater than baseline values for all subjects (p = 0.009, Pre = 135.71 ± 1.1°, 48H = 137.98 ± 1.2°).

		Limb Circumference (cm)			Knee ROM (°)			
		Pre	Post	48H	Pre	Post	48H	
CON	ND	59.5 (1.6)	59.5 (1.7)	59.5 (1.8)	136.6 (1.8)	137.5 (2.4)	139.1 (1.4)	
	D	59.2 (1.5)	59.8 (1.4)	59.4 (1.7)	135.3 (1.2)	135.9 (2.0)	136.9 (1.8)	
INJ	ND	56.8 (2.8)	58.3 (3.3)	58.7 (3.1)	137.6 (3.5)	137.4 (3.9)	138.7 (4.2)	
	D	57.7 (3.2)				133.1 (3.0)		
Values are means (SE). CON, control group INJ, injury group. ND, non-Dominant. D, dominant,								

Table 4.8 Thigh Circumference and Total Knee Range of Motion for left and right legs

## CHAPTER FIVE

#### DISCUSSION

# Degree of Quadricep Muscle Injury Caused by Downhill Running

Subjects who ran for 60 minutes downhill experienced significant exercise-induced injury to their quadriceps muscle group as evidenced by immediate and prolonged strength deficits and delayed onset muscle soreness. Skeletal muscle injury was evident by the significant decline ( $p \le 1$ 0.015) of MVC torque immediately post and 48 hours after downhill running, which has been proposed to provide the best measure of exercise-induced muscle injury in humans (Warren et al., 1999). In contrast to the control group, those in the injury group experienced an immediate 25.3% decrease in maximal voluntary strength of their non-dominant knee extensor group at 90°, a 20.7% decrease at 45°, and a 23.4% decrease at 20°. These deficits are comparable to other studies (15-27%) that use downhill running as a model of exercise-induced injury to the quadriceps group (Eston et al., 2000; Rowlands et al., 2001; Malm et al., 2004; Baumann et al., 2014). MVC torque remained 11.8-14.3% below (p < 0.015) baseline values 48H following the run. The injury group had greater ( $p \le 0.046$ ) quadricep muscle soreness immediately post and at 48H. This supports the findings of other research that shows skeletal muscle soreness is significantly elevated at 24-72 hours following eccentric exercise (Balnave & Thompson, 1993; Whitehead et al., 1998; Clarkson & Hubal, 2002; Lee et al., 2002; Green et al., 2010; Brandenberger et al., 2021). Stimulated muscle torque (20 and 80 Hz) was significantly lower (p  $\leq$  0.018) immediately and 48H following downhill running indicating a decline in intrinsic muscle contractile function following injury. Injured muscles need several days for complete functional recovery (Clarkson & Hubal, 2002) unlike fatigue in which force impairments are restored within a matter of minutes to hours (Cady et al., 1989; Gibson et al., 1993).

## **Evidence of Differential Injury**

We hypothesized that post-injury strength deficits and soreness would be significantly different between at least two knee extensor muscles following exercise-induced injury. Following downhill running there were immediate deficits of 22.4% for the VM, 21.1% for the RF, and the greatest strength deficit at 20 Hz occurring in the VL which was reduced by 32.9%. The greater deficit in the VL was not statistically ( $p \ge 0.095$ ) greater than the other muscles. However, a limitation is that our study was underpowered (Observed Power = 0.467) for electrically stimulated muscle torque and therefore we do not have the confidence to say that there were not meaningful differences in force deficits amongst the knee extensor muscles following DHR. An indication of differential injury was revealed by significant differences in the individual soreness between the knee extensor muscles of the injury group at 48 hours. The VL of the injury group was sorer than both the RF and the VM at 48H with no statistical differences between the RF and VM.

Previous studies have used transverse relaxation time ( $T_2$ )-weighted MRI to identify the magnitude of injury across the quadriceps group. Both Black & McCully (2008) and Prior et al. (2001) found greater increases in the  $T_2$  in the RF compared to the VL suggesting a higher degree of injury after eccentric-only quadricep exercise. However, it has been found that different exercises could cause varying levels  $T_2$  across the quadriceps muscles (Maeo et al., 2018). Single-joint eccentric contractions of the knee extensors caused  $T_2$  increases in the VM, VI and RF but not the VL while eccentric squat and downhill walking most affected the VM (Maeo et al., 2018). Twenty-four hours following 100 smith squats at ~70% body mass there was an increase in  $T_2$  from baseline for the VM, VI and VL but not the RF (Fulford et al., 2014).

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T<sub>2</sub> is a measurement of water content in skeletal muscle (Black & McCully, 2008; Maeo et al., 2018). This is an indirect measurement of muscle damage and does not provide a functional capacity to assess the injury. Other studies have also found differences in soreness across the individual muscles of the quadriceps following eccentric exercise. Cleary et al. (2006) found that the vasti muscles were significantly sore in the days following 45 minutes of downhill running while the RF did not exhibit any change in soreness. Following 300 unilateral max-effort eccentric actions of the quadriceps femoris, greater soreness was exhibited in the VM compared to the VL and RF (Paulsen et al., 2010). These studies collectively support our findings that the degree of injury is varied across the quadricep muscle group following eccentric contractions. It is evident that the modality and intensity of exercise may impact the degree of differential injury between a synergist group. Collectively the research shows that some markers of injury vary across a synergist group following eccentric exercise.

Several factors may explain differential injury of the quadricep muscles. First, many studies that have shown that fast-twitch skeletal muscle fibers are predominately injured following a bout of maximal eccentric contractions (Fridén et al., 1983; Lieber & Fridén, 1988; Warren et al., 1994; Vijayan et al., 2001). Following 15 stimulated eccentric contractions, force deficits in the fast-twitch extensor digitorum longus (EDL) were eight times greater than the slow-twitch soleus of normal weight-bearing mice (Warren et al., 1994). However, fiber type distribution does not exhibit a dominance across the quadricep muscles, with slow Type I fiber compostion of VM, RF and VL young males ranging from 40 to 60% (Johnson et al., 1973; Simoneau & Bouchard, 1989; Glenmark et al., 1992, Travnik et al., 1995). Moreover, it is generally accepted that fast Type II fibers are not preferentially recruited during low-to-moderate intensity downhill running.

Therefore, fiber-type cannot explain the differential injury across the quadriceps muscles seen in the current study. Second, Warren and coworkers (1994) determined that previous usage patterns of skeletal muscle more so than fiber-type influences susceptibility to injury. They found that mouse skeletal muscles that are used frequently for weight-bearing activity (i.e., soleus) are less susceptible to eccentric contraction-induced injury than muscles that are not use for weightbearing activity (i.e., extensor digitorum longus). When mechanical stress experienced by the soleus muscle was reduced via hindlimb-suspension, the soleus muscle exhibited maximal isometric force deficits similar to that of the EDL muscle and was nearly 4-times greater than weight bearing mice after eccentric contraction-induced injury (Warren et al., 1994). Differences in the previous usage also likely do not explain differential soreness across the quadriceps seen in this study. Subjects were sedentary or recreationally active individuals who do not resistance or plyometric train and everyday activities (e.g., standing, walking) likely would not result in differential use of the individual quadriceps. Third, differences in stress and strain across the quadriceps muscles during downhill running can stem from differences in muscle architecture (Lieber & Fridén, 2000; Shin et al., 2009). Moreover, peak stress is known to be a primary variable associated with eccentric contraction-induced muscle injury (Warren et al 1993; Lieber & Fridén et al., 1993). The VL and RF exhibit a bipennate fascicle arrangement and lesser pennation angles than the VM (Chiu & Daehlin, 2021). Chiu & Daehlin also estimated that the VL produces greater longitudinal force between the midpoints of the distal and proximal aspects of the muscle than the RF (Chiu & Daehlin, 2021). Therefore, it is possible that the VL muscle experienced the highest level of force amongst the measured quadricep muscles during downhill running due to a combination of architecture and mechanics, resulting in greater exerciseinduced injury.

### **Causes of MVC Torque Loss**

MVC torque at 90° decreased by 25.3% immediately following downhill running for the injury group and remained 14% below baseline at 48H. We hypothesized that the primary etiology of knee extensor strength deficits would reside in the skeletal muscle and not the nervous system, with a failure to activate force-bearing structures (20 Hz-to-80 Hz stimulation torque ratio) contributing to a failure of the force-bearing structures (80 Hz stimulation torque). Direct failure of the knee extensor muscles to generate force after injury was evidenced by significant decreases in 20 and 80 Hz stimulation torque compared to the control group immediately and 48H following exercise. Moreover, there was a significant decrease in 20/80 Hz torque ratio immediately following the downhill run. However, the change in the 20/80 Hz ratio was not significantly associated ( $r^2 = 0.11$ , p = 0.095) with changes in MVC torque. Although changes in MVC torque were significantly associated with changes in 20 Hz ( $r^2 = 0.23$ ) and 80 Hz ( $r^2 =$ 0.20) stimulation torque, intrinsic knee extensor muscle failure did not explain the greatest variation in MVC torque which was contrary to our hypothesis. Changes in EMG RMS during MVC 90° was the most correlated ( $r^2 = 0.45$ ) with changes in MVC torque 90° torque. EMG RMS at 90° MVC torque decreased by 16.8% immediately post-injury and remained at the same level 48H later. Many studies have shown immediate decreases in muscle activation while performing MVCs following eccentric contraction-induced injury (Warren et al., 2000; Prasartwuth et al., 2005; Gauche et al., 2009; Hedayatpour et al., 2014).

Previous studies investigating low and high-frequency stimulation across the entire quadriceps muscle group have demonstrated greater reductions in low-frequency torque compared to high-frequency following exercise-induced injury (Brown et al., 1996/1997; Child et al., 1999;

Baptista et al., 2009, Kamandulis et al., 2017). Kamandulis et al. (2017, personal communication) found that 100Hz stimulated torque of the entire quadriceps muscle group one hour following (approximately the same time as our post stimulations were recorded) 100 drop jumps was reduced by roughly 15% in young recreationally active subjects compared to approximately a 51.3% decline in 20 Hz torque. Animal studies have traditionally demonstrated that 57-75% of early strength loss following injury is attributed to E-C uncoupling (Ingalls, 1998; Warren, 2001). Discrepancies between our study and those studies could lie within the method of injury induction. Injury studies involving laboratory animals typically produce a series of high-force eccentric contractions in muscles by supramaximal electrical stimulation of the nerves or muscles resulting in 100% myofiber activation. In contrast, injury studies involving humans running downhill produce a series of relatively moderate intensity contractions. At low to moderate exercise intensities, most of the motor units recruited are low threshold (Henneman et al., 1965a; Henneman et al., 1965b). Although running downhill amplifies the magnitude of the eccentric contraction compared to level running, the degree of muscle activation is relatively low when compared to an MVC. Both animal (Armstrong et al., 1984) and human (Mair et al., 1992) studies document that volitional running activities appear to preferentially injure slow-Type I myofibers in quadricep muscles. Although fast Type II myofibers exhibit greater intrinsic susceptibility to eccentric contraction-induced muscle injury than slow-type myofibers (Friden, 1983; Jones, 1986; Lieber and Friden, 1988; Warren, 1994), the likelihood that a given fiber-type gets injured depends more on the prior contractile history of the muscle (Warren, 1994) and motor unit recruitment patterns used during the injurious exercise (Semmler, 2014). We can determine that the downhill running was at a relatively moderate intensity (65% of heart rate reserve) and the degree of individual muscle activation of running on a level treadmill was only

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"18.5-32.2%" of that of the MVC at 20-degree joint angle and that running downhill is known to recruit less quadriceps muscle than level running (Cai et al., 2010). Because of this it is unlikely that significant high threshold motor units and Type II fibers were used during exercise and differences in injury between the quadriceps muscles would not likely be based on fiber type distribution across the individual muscles.

Greater decreases in low-frequency (i.e., 20 Hz) stimulation torque compared to high-frequency (i.e., 80 Hz) stimulation torque may be explained by force- $Ca^{2+}$  and force-frequency relationships. As stimulation frequency increases there is an increase in the contribution of the force produced by high-threshold motor units (Wüst, 2008). Based on force-calcium and quadriceps torque-frequency relationships, we estimate that a majority of the Type I fibers between the stimulating electrodes will be near maximally activated during 20 Hz torque stimulation while Type II fibers will be sub-maximally (~20% of peak isometric force) activated at this frequency. At 80 Hz, Type I fibers will be maximally activated whereas the Type II fibers will be near maximally activated (~80% of peak isometric force). Therefore, the 20 Hz torque deficit (-25.5%) immediately following DHR likely reflects significant strength loss of injured Type I fibers that operate on the steep portion of force-calcium relationship. At 80 Hz, injured Type I fibers would be predicted to lose less force than at 20 Hz because of the force-calcium relationship, whereas uninjured Type II fibers are still able to produce near maximal force, thus accounting for small reductions (8-9%) in 80 Hz force after injury. At 48H, MVC torque, 80 Hz torque, and EMG RMS remained at similar levels to immediately post. 48H MVC torque was not as low as immediately post (25.3% vs. 14%) however, this might be explained by the recovery of 20 Hz torque and 20/80 Hz ratio (e.g., EC uncoupling) at 48H. Our results show that a decrease

in muscle activation (i.e., EMG RMS) and injury to type I fibers contributed to the loss of maximal volitional torque following eccentric exercise.

Nervous System Response to Torque Deficits and Differential Injury During Locomotion We hypothesized that eccentric contraction-induced muscle injury would alter knee extensor muscle activation patterns during treadmill walking and running. All three knee extensor muscles had a trend of increased EMG RMS during walking following injury (VM = 17.9%, RF = 35.8%, VL = 32.2%) and running EMG RMS of the knee extensors was greater immediately after the downhill run. The increases in running knee extensor muscle activation (i.e., EMG RMS) were due to the injury since no changes (p = 1.0) were seen in the muscles of the control group at any time point. The collective EMG data indicates that there is a global increase in muscle activation during locomotion after muscles have suffered varying degrees of exercise-induced injury. Significant variations in recruitment thresholds, discharge rates, motor unit conduction velocities and synchronization occur up to one week following eccentric exercise (Semmler, 2014) which could explain changes in EMG RMS. Increases in quadriceps muscle activation during submaximal tasks have been demonstrated following ischemia in combination with knee extension exercise (Pierce et al., 2006). This increase in muscle activation is likely to account for the decreases in the force-generating capacity of injured skeletal muscles (Pierce et al., 2006). Previous research has also shown an increase in quadriceps muscle activation at submaximal intensities following eccentric contractions (Martin et al., 2004; Ehrström, 2018). There were also significant changes to the activation of the TA during walking and running trials for the injury group. During downhill running, the tibialis anterior has an increase in eccentric work which has been shown to lead to significant losses of strength, increases in damage and soreness,

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and changes in EMG RMS following the bout (Eston et al., 1995; Giandolini et al., 2017). Downhill running produces similar peak dorsiflexion angles as flat running (Eston et al., 1995) explaining why changes in activation were not seen in the soleus muscles of the injury group. Our results show a global increase in knee extensor and dorsiflexor muscle activation to maintain walking and running speeds immediately following exercise-induced injury.

#### Neuromuscular Control of Balance following Downhill Running

We hypothesized that eccentric contraction-induced muscle injury would impair balance, as indicated by the prolonged trajectory of the center of pressure (COP) during all three experimental conditions during quiet standing. There was an increase in COP length that occurred immediately following downhill running. Balance did recover to baseline levels (p = 1.0) at 48H for the injury group. The control group had improved balance at 48H with decreases in COP length which were less ( $p \le 0.036$ ) than both baseline and immediately following treadmill walking. Previous studies have demonstrated increases in COP length immediately following strenuous running but no change with non-fatiguing exercise (Nardone et al., 1997/1998; Wiest et al., 2011). Increases in COP length and area were shown following fatiguing exercise in the elderly (Nam et al., 2013). Nardone and coworkers (1998) determined that changes in balance following strenuous exercise are short-lived and only last for a few minutes. Our findings also show that DHR impacted standing balance immediately following but not at 48H. It is likely that exercise-induced injury in the TA impacted standing balance for the injury group immediately post. The tibialis anterior is identified as an important postural muscle for maintaining upright standing posture (Michel-Pellegrino et al., 2006). We found that TA activation increased during locomotion immediately following the downhill run. During this time there was also an increase in COP length for the injury group. TA muscle EMG RMS, /MF and

COP length returned to baseline levels at 48H. We presume that injury to the TA correlated with the decrease in balance immediately post but other studies have shown no changes in COP variability or path length following eccentric injury to the dorsiflexors and plantarflexors (McIntosh et al., 2018). There was also qualitative evidence of a greater increase in reliance on the somatosensory systems in the injury group to maintain balance. Although increases in PQ immediately after (40.5%) and 48H (20.5%) after the downhill run were not statistically significant (p = 0.208), a low statistical power (Observed Power = 0.316) does not engender confidence in stating there are no meaningful changes.

#### Use of Stimulation to Evaluate Individual Muscle Strength

Results from the pilot study indicated a high degree of repeatability when using stimulation to generate individual knee extensor torque. Both the Biodex and Vicon scored high intraclass correlation coefficients (> 0.9) representing excellent reliability (Koo and Li et al., 2016). When evaluated separately for stimulation frequencies, the Biodex was considered to have good reliability for both 20 & 80 Hz with ICCs of 0.86 for both settings. 80 Hz stimulation calculated from Vicon motion capture was also considered to have good reliability with an ICC of 0.9. However at 20 Hz, Vicon motion capture was found to have only moderate reliability with an ICC of 0.7. This ultimately led us to use the Biodex as the form of individual muscle torque measurement in our main study. We were also able to add secondary data in terms of the reliability of the Biodex to collect individual muscle torque. On the familiarization day for our main study, subjects were stimulated 3 times at 20 Hz on all three knee extensor muscles and 2 times at 80 Hz for the rectus femoris. Using the familiarization as a first day of data collection and comparing the means to each subject's baseline (i.e., Pre) individual torque values, it further

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demonstrated the excellent test-retest reliability of the Biodex overall (ICC = 0.972) and individually at 20 Hz (ICC = 0.911) and 80 Hz (ICC = 0.94). Some of time periods between the familiarization days and pre trials were multiple weeks apart, further attesting the reliability of these methods in order to assess individual muscle torque. We believe that the results from both studies validate the use of these methods to collect individual muscle torque.

There are limitations to our method of evaluating individual muscle strength. First, the percentage of fibers that are recruited with each stimulation is unknown. It is unlikely that all the myofibers of an individual muscle are recruited during these stimulations and therefore the full degree of muscle strength or injury would not be known. In addition, we could only measure the force produced by the three superficial muscles of the quadriceps muscle group. We are unable to measure potential baseline and post torque ratios of the vastus intermedius since it runs deep to the rectus femoris. Another limitation includes the inability to know the degree of activated fibers in neighboring synergist muscles during each stimulation. Despite this, the baseline sum of the three tested muscles did equate to 52.5% of the subjects' MVC. Total 80 Hz torque was significantly correlated ( $r^2 = 0.494$ ; p < 0.001) with MVC 90° torque (Figure 5.1). Another limitation includes the inability to know the degree of synergist muscles during each stimulation in neighboring synergist muscles during each stimulation includes the inability in neighboring synergist muscles during each stimulation.

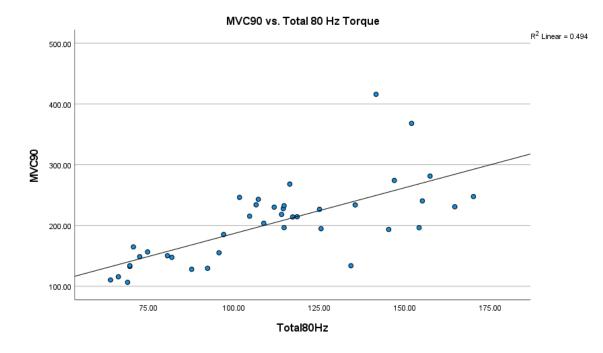


Figure 5.1 Linear regression analysis comparing MVC 90° torque to Total 80 Hz torque.

#### **Application of Findings**

Data from both our main study and pilot study show an excellent test-retest reliability when using the Biodex to measure submaximal tetanic contraction at both stimulation frequencies (i.e., 20 and 80 Hz) for individual muscles. Our methodology offers an assessment approach to evaluate strength and function in individual skeletal muscles across the body. This is the first study of its kind to compare torque produced both low-frequency (20 Hz) and high-frequency (80 Hz) stimulations across the individual muscles of a synergist muscle group in humans. Our study has confirmed differential injury across synergists muscles due to significantly higher soreness in the VL than the RF and VM at 48H. We were not able to show statistical differences in 20 or 80 Hz stimulated torque across the knee extensor muscle group following downhill running. However, with the four-way interaction for normalized torque remaining close to significance and study being underpowered ( $p \ge 0.095$ , Observed Power  $\le 0.467$ ) we cannot with confidence say that there are not differences in stimulated torque deficits between the observed muscles. Being that this is the first time that differential muscle injury between synergists has been tested, future research should evaluate the potential stimulated strength deficits that could exist in different muscle groups, different populations and for different modalities of exerciseinduced injury and evaluate the impact on joint mechanics. It has been previously proposed that unbalanced and asymmetric activities can create differential fatigue across muscles and thereby a kinetic and kinematic imbalance could result in musculoskeletal injury (Kumar, 2010). Strength imbalances of a working muscle group can promote instability of the joint (Yaggie & McGregor, 2002; Fitzgerald et al., 2004; Khalaj et al., 2020) and this instability could potentially lead to injuries such as ligament sprains or tendinopathies. Any exercise or activity that causes differential strains on a muscle group, may result in leaving a person more susceptible to a secondary injury due to altered joint mechanics. It has been shown that other markers of injury (i.e., soreness, swelling) differ across synergist muscles following eccentrically biase exercise. If strength imbalances exist between synergists muscles following exercise-induced injury, recommendations for physical activity and training would likely be altered to prevent further injury.

## Conclusions

This study provides evidence in humans that there are differences in susceptibility to exercise related injury across a synergist group with the VL having the greatest soreness at 48 hours. Most of the MVC torque loss was correlated with a decrease in muscle activation (i.e., EMG RMS). Intrinsic force depression (i.e., 20 and 80 Hz torque) also explained declines in maximal strength post injury. The injury caused increases in muscle activation to the injured group during

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submaximal exercise, a decrease in activation during maximal contractions, decreases in low and high frequency stimulation torque, decreases in 20/80 Hz torque ratio, and caused a decrease in standing balance immediately following 60 minutes of downhill running.

# REFERENCES

Alexander, G. E., & Crutcher, M. D. (1990). Functional architecture of basal ganglia circuits: neural substrates of parallel processing. Trends in neurosciences, 13(7), 266-271.

Alkner, B. A., Tesch, P. A., & Berg, H. E. (2000). Quadriceps EMG/force relationship in knee extension and leg press. Medicine & Science in Sports & Exercise, 32(2), 459.

Altenburg, T. M., Degens, H., van Mechelen, W., Sargeant, A. J., & de Haan, A. (2007). Recruitment of single muscle fibers during submaximal cycling exercise. Journal of applied physiology, 103(5), 1752-1756.

Altringham, J. D., & Johnston, I. A. (1982). The pCa-tension and force-velocity characteristics of skinned fibres isolated from fish fast and slow muscles. The Journal of Physiology, 333(1), 421-449.

Amarantini, D., Rao, G., & Berton, E. (2010). A two-step EMG-and-optimization process to estimate muscle force during dynamic movement. Journal of biomechanics, 43(9), 1827-1830.

Andersen, R. A., & Buneo, C. A. (2002). Intentional maps in posterior parietal cortex. Annual review of neuroscience, 25(1), 189-220.

Armstrong, R. B., Ogilvie, R. W., & Schwane, J. A. (1983). Eccentric exercise-induced injury to rat skeletal muscle. Journal of Applied Physiology, 54(1), 80-93.

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

Balnave, C. D., & Thompson, M. W. (1993). Effect of training on eccentric exercise-induced muscle damage. Journal of Applied Physiology, 75(4), 1545-1551.

Balnave, C. D., & Allen, D. G. (1995). Intracellular calcium and force in single mouse muscle fibres following repeated contractions with stretch. The Journal of Physiology, 488(1), 25-36.

Baptista, R. R., Scheeren, E. M., Macintosh, B. R., & Vaz, M. A. (2009). Low-frequency fatigue at maximal and submaximal muscle contractions. Brazilian Journal of Medical and Biological Research, 42(4), 380-385.

Baroni, B. M., Geremia, J. M., Rodrigues, R., De Azevedo Franke, R., Karamanidis, K., & Vaz, M. A. (2013). Muscle architecture adaptations to knee extensor eccentric training: rectus femoris vs. vastus lateralis. Muscle & nerve, 48(4), 498-506.

Baumann, C. W., Green, M. S., Doyle, J. A., Rupp, J. C., Ingalls, C. P., & Corona, B. T. (2014). Muscle injury after low-intensity downhill running reduces running economy. The Journal of Strength & Conditioning Research, 28(5), 1212-1218.

Baumann, C. W., Rogers, R. G., Gahlot, N., & Ingalls, C. P. (2014). Eccentric contractions disrupt FKBP 12 content in mouse skeletal muscle. Physiological Reports, 2(7), e12081.

Baumann, C. W., Rogers, R. G., Otis, J. S., & Ingalls, C. P. (2016). Recovery of strength is dependent on mTORC1 signaling after eccentric muscle injury. Muscle & nerve, 54(5), 914-924.

Beato, M., Madruga-Parera, M., Piqueras-Sanchiz, F., Moreno-Pérez, V., & Romero-Rodriguez, D. (2019). Acute Effect of Eccentric Overload Exercises on Change of Direction Performance and Lower-Limb Muscle Contractile Function. *Journal of Strength and Conditioning Research*.

Beltman, J. G. M., De Haan, A., Haan, H., Gerrits, H. L., Van Mechelen, W., & Sargeant, A. J. (2004). Metabolically assessed muscle fibre recruitment in brief isometric contractions at different intensities. European journal of applied physiology, 92(4), 485-492.

Benarroch, E. E. (2006). Basic neurosciences with clinical applications. Elsevier Health Sciences.

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(9), 1605.

Blaszczyk, J. W., Bacik, B., & Juras, G. (2003). Clinical assessment of postural stability. Journal of Mechanics in Medicine and Biology, 3(02), 135-144.

Blazevich, A. J. (2006). Effects of physical training and detraining, immobilization, growth and aging on human fascicle geometry. Sports medicine, 36(12), 1003-1017.

Borejdo, J., & Morales, M. F. (1977). Fluctuations in tension during contraction of single muscle fibers. Biophysical Journal et al., 20(3), 315-334.

Bottaro, A., Yasutake, Y., Nomura, T., Casadio, M., & Morasso, P. (2008). Bounded stability of the quiet standing posture: an intermittent control model. Human movement science, 27(3), 473-495.

Brandenberger, K., Warren, G. L., Christopher, P., Otis, J. S., & Andrew, J. (2021). Downhill running impairs activation and strength of the elbow flexors. Journal of Strength and Conditioning Research. doi: 10.1519/JSC.000000000003111

Brockett, C., Warren, N., Gregory, J. E., Morgan, D. L., & Proske, U. (1997). A comparison of the effects of concentric versus eccentric exercise on force and position sense at the human elbow joint. Brain research, 771(2), 251-258.

Brockett, C. L., Morgan, D. L., Gregory, J. E., & Proske, U. (2002). Damage to different motor units from active lengthening of the medial gastrocnemius muscle of the cat. Journal of Applied Physiology, 92(3), 1104-1110.

Brown, S. J., Child, R. B., Donnelly, A. E., Saxton, J. M., & Day, S. H. (1996). Changes in human skeletal muscle contractile function following stimulated eccentric exercise. European journal of applied physiology and occupational physiology, 72(5-6), 515-521.

Buller, A. J., & Lewis, D. M. (1965). The rate of tension development in isometric tetanic contractions of mammalian fast and slow skeletal muscle. The Journal of physiology, 176(3), 337-354.

Byrne, C., Twist, C., & Eston, R. (2004). Neuromuscular function after exercise-induced muscle damage. Sports medicine, 34(1), 49-69.

Cady, E. B., Jones, D. A., Lynn, J., & Newham, D. J. (1989). Changes in force and intracellular metabolites during fatigue of human skeletal muscle. The Journal of physiology, 418(1), 311-325.

Cai, Z. Y., Hsu, C. C., Su, C. P., Lin, C. F., Lin, Y. A., Lin, C. L., & Hsu, M. C. (2010). Comparison of lower limb muscle activation during downhill, level and uphill running. Isokinetics and Exercise Science, 18(3), 163-168.

Calderón, J. C., Bolaños, P., & Caputo, C. (2014). The excitation–contraction coupling mechanism in skeletal muscle. Biophysical reviews, 6(1), 133-160.

Cannon, J. G., Fielding, R. A., Fiatarone, M. A., Orencole, S. F., Dinarello, C. A., & Evans, W. J. (1989). Increased interleukin 1 beta in human skeletal muscle after exercise. American Journal0020of Physiology-Regulatory, Integrative and Comparative Physiology, 257(2), R451-R455.

Cappellini, G., Ivanenko, Y. P., Poppele, R. E., & Lacquaniti, F. (2006). Motor patterns in human walking and running. Journal of neurophysiology, 95(6), 3426-3437.

Cashaback, J. G., & Cluff, T. (2015). Increase in joint stability at the expense of energy efficiency correlates with force variability during a fatiguing task. Journal of Biomechanics, 48(4), 621-626.

Chan, O., Del Buono, A., Best, T. M., & Maffulli, N. (2012). Acute muscle strain injuries: a proposed new classification system. Knee Surgery, Sports Traumatology, Arthroscopy et al., 20(11), 2356-2362.

Chen, T.C. (2003). Effects of a second bout of maximal eccentric exercise on muscle damage and electromyographic activity. Eur J Appl Physiol 89, 115–121.

Child, R., Brown, S., Day, S., Donnelly, A., Roper, H., & Saxton, J. (1999). Changes in indices of antioxidant status, lipid peroxidation and inflammation in human skeletal muscle after eccentric muscle actions. Clinical science, 96(1), 105-115.

Christie, A., Inglis, J. G., Kamen, G., & Gabriel, D. A. (2009). Relationships between surface EMG variables and motor unit firing rates. Eu0072opean journal of applied physiology, 107(2), 177-185.

Clarkson P.M., Nosaka K., Braun B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. Med Sci Sports Exerc, 24, 512–520.

Clarkson, P. M., & Sayers, S. P. (1999). Etiology of Exercise-Induced Muscle Damage. Canadian Journal of Applied Physiology, 24(3), 234.

Clarkson, P. M., & Hubal, M. J. (2002). Exercise-induced muscle damage in humans. American journal of physical medicine & rehabilitation, 81(11), S52-S69.

Cleak, M. J., & Eston, R. G. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. British Journal of Sports Medicine, 26(4), 267-272.

Cleary, M. A., Sitler, M. R., & Kendrick, Z. V. (2006). Dehydration and symptoms of delayedonset muscle soreness in normothermic men. Journal of Athletic Training, 41(1), 36.

Corona, B. T., Balog, E. M., Doyle, J. A., Rupp, J. C., Luke, R. C., & Ingalls, C. P. (2010). Junctophilin damage contributes to early strength deficits and EC coupling failure after eccentric contractions. American Journal of Physiology-Cell Physiology, 298(2), C365-C376.

Crouzier, M., Hug, F., Dorel, S., Deschamps, T., Tucker, K., & Lacourpaille, L. (2019). Do individual differences in the distribution of activation between synergist muscles reflect individual strategies?. Experimental Brain Research, 237(3), 625-635.

Dankaerts, W., O'Sullivan, P. B., Burnett, A. F., Straker, L. M., & Danneels, L. A. (2004). Reliability of EMG measurements for trunk muscles during maximal and sub-maximal voluntary isometric contractions in healthy controls and CLBP patients. Journal of Electromyography and Kinesiology, 14(3), 333-342.

Dartnall, T. J., Nordstrom, M. A., & Semmler, J. G. (2008). Motor unit synchronization is increased in biceps brachii after exercise-induced damage to elbow flexor muscles. Journal of Neurophysiology, 99(2), 1008-1019.

Dartnall, T. J., Rogasch, N. C., Nordstrom, M. A., & Semmler, J. G. (2009). Eccentric muscle damage has variable effects on motor unit recruitment thresholds and discharge patterns in elbow flexor muscles. Journal of Neurophysiology, 102(1), 413-423.

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(3), 1225-1235.

de Brito Fontana, H., Han, S. W., Sawatsky, A., & Herzog, W. (2018). The mechanics of agonistic muscles. Journal of Biomechanics, 79, 15-20.

Deschenes, M. R., Brewer, R. E., Bush, J. A., McCoy, R. W., Volek, J. S., & Kraemer, W. J. (2000). Neuromuscular disturbance outlasts other symptoms of exercise-induced muscle damage. Journal of the Neurological Sciences, 174(2), 92-99.

Desmedt, J.E. & Godaux, E. (1977). Ballistic contractions in man: characteristic recruitment pattern of single motor units of the tibialis anterior muscle. J Physiol 264, 673–693.

Dichgans J, & Mauritz KH. (1983). Patterns and mechanisms of postural instability in patients with cerebellar lesions. Adv Neurol 39:633–43

Dietz, V. et al. (1986) Obstruction of the swing phase during gait: phase-dependent bilateral leg muscle coordination. Brain Res. 384, 166–169

Dietz, V. (1992). Human neuronal control of automatic functional movements: interaction between central programs and afferent input. Physiological reviews, 72(1), 33-69.

Dietz, V., Zijlstra, W., & Duysens, J. (1994). Human neuronal interlimb coordination during split-belt locomotion. Experimental brain research, 101(3), 513-520.

Dietz, V. (2003). Spinal cord pattern generators for locomotion. Clinical Neurophysiology, 114(8), 1379-1389.

Dillon, E. M., Erasmus, P. J., Müller, J. H., Scheffer, C., & Villiers, R. V. (2008). Differential Forces within the Proximal Patellar Tendon as an Explanation for the Characteristic Lesion of Patellar Tendinopathy. The American Journal of Sports Medicine, 36(11), 2119-2127. doi:10.1177/0363546508319311

Du Bois-Reymond, E. (1849). Untersuchungen uber thierische Elektricitat.

Duchateau, J., & Enoka, R. M. (2016). Neural control of lengthening contractions. Journal of Experimental Biology, 219(2) et al., 197-204.

Dundon, J.M., Cirillo, J. & Semmler, J.G. (2008). Low-frequency fatigue and neuromuscular performance after exercise-induced damage to elbow flexor muscles. J Appl Physiol 105, 1146–1155.

Ehrström, S., Gruet, M., Giandolini, M., Chapuis, S., Morin, J. B., & Vercruyssen, F. (2018). Acute and delayed neuromuscular alterations induced by downhill running in trained trail runners: beneficial effects of high-pressure compression garments. Frontiers in physiology, 1627.

Enoka, R. M., & Duchateau, J. (2017). Rate coding and the control of muscle force. *Cold Spring Harbor perspectives in medicine*, 7(10), a029702.

Eston, R. G., Mickleborough, J., & Baltzopoulos, V. (1995). Eccentric activation and muscle damage: biomechanical and physiological considerations during downhill running. British journal of sports medicine, 29(2), 89-94.

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. Scandinavian journal of medicine & science in sports, 10(4) et al., 199-204.

Farina, D. (2008). Counterpoint: spectral properties of the surface EMG do not provide information about motor unit recruitment and muscle fiber type. Journal of Applied Physiology, 105(5), 1673-1674.

Favero, T. G. (1999). Sarcoplasmic reticulum Ca2+ release and muscle fatigue. Journal of Applied Physiology, 87(2), 471-483.

Ferreira, S. H., Lorenzetti, B. B., Bristow, A. F., & Poole, S. (1988). Interleukin-1β as a potent hyperalgesic agent antagonized by a tripeptide analogue. Nature, 334(6184), 698-700.

Fielding, R. A., Manfredi, T. J., Ding, W., Fiatarone, M. A., Evans, W. J., & Cannon, J. G. (1993). Acute phase response in exercise. III. Neutrophil and IL-1 beta accumulation in skeletal muscle. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 265(1), R166-R172.

Fitzgerald, G. K., Piva, S. R., & Irrgang, J. J. (2004). Reports of joint instability in knee osteoarthritis: its prevalence and relationship to physical function. Arthritis care & research, 51(6), 941-946.

Foley, J. M., Jayaraman, R. C., Prior, B. M., Pivarnik, J. M., & Meyer, R. A. (1999). MR measurements of muscle damage and adaptation after eccentric exercise. Journal of Applied Physiology, 87(6), 2311-2318.

Franchi, M. V., Atherton, P. J., Reeves, N. D., Flück, M., Williams, J., Mitchell, W. K., ... & Narici, M. V. (2014). Architectural, functional and molecular responses to concentric and eccentric loading in human skeletal muscle. Acta Physiologica, 210(3), 642-654.

Freeze, B. S., Kravitz, A. V., Hammack, N., Berke, J. D., & Kreitzer, A. C. (2013). Control of basal ganglia output by direct and indirect pathway projection neurons. Journal of Neuroscience, 33(47), 18531-18539.

Freitas, S. R., Antunes, A., Salmon, P., Mendes, B., Firmino, T., Cruz-Montecinos, C., ... & Vaz, J. R. (2019). Does epimuscular myofascial force transmission occur between the human quadriceps muscles in vivo during passive stretching?. Journal of biomechanics, 83, 91-96.

Fridén, J., Sjöström, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. International journal of sports medicine, 4(03), 170-176.

Fridén, J., & Lieber, R. L. (1998). Segmental muscle fiber lesions after repetitive eccentric contractions. Cell and tissue research, 293(1), 165-171.

Fridlund, A. J., & Cacioppo, J. T. (1986). Guidelines for human electromyographic research. Psychophysiology, 23(5), 567-589.

Fuglevand, A. J., Winter, D. A., & Patla, A. E. (1993). Models of recruitment and rate coding organization in motor-unit pools. Journal of neurophysiology, 70(6), 2470-2488.

Funk, C. D. (2001). Prostaglandins and leukotrienes: advances in eicosanoid biology. science, 294(5548), 1871-1875.

Galvan, A., Kuwajima, M., & Smith, Y. (2006). Glutamate and GABA receptors and transporters in the basal ganglia: what does their subsynaptic localization reveal about their function?. Neuroscience, 143(2), 351–375.

Gandevia, S. C. (2001). Spinal and supraspinal factors in human muscle fatigue. Physiological reviews.

Giandolini, M., Horvais, N., Rossi, J., Millet, G. Y., Morin, J. B., & Samozino, P. (2017). Effects of the foot strike pattern on muscle activity and neuromuscular fatigue in downhill trail running. Scandinavian journal of medicine & science in sports, 27(8), 809-819.

Gibson, H., Carroll, N., Clague, J. E., & Edwards, R. H. (1993). Exercise performance and fatiguability in patients with chronic fatigue syndrome. Journal of Neurology, Neurosurgery & Psychiatry, 56(9), 993-998.

Glenmark, B., Hedberg, G., & Jansson, E. (1992). Changes in muscle fibre type from adolescence to adulthood in women and men. Acta Physiologica Scandinavica, 146(2), 251-259.

Gorassini, M. A., Prochazka, A., Hiebert, G. W., & Gauthier, M. J. (1994). Corrective responses to loss of ground support during walking. I. Intact cats. Journal of neurophysiology, 71(2), 603-610.

Gordon, A. M., Huxley, A. F., & Julian, F. J. (1966). The variation in isometric tension with sarcomere length in vertebrate muscle fibres. The Journal of physiology, 184(1), 170-192.

Gottlieb, G. L., Latash, M. L., Corcos, D. M., Liubinskas, T. J., & Agarwal, G. C. (1992). Organizing principles for single joint movements: V. Agonist-antagonist interactions. Journal of Neurophysiology, 67(6), 1417-1427.

Green, M. S., Doyle, J. A., Ingalls, C. P., Benardot, D., Rupp, J. C., & Corona, B. T. (2010). Adaptation of insulin-resistance indicators to a repeated bout of eccentric exercise in human skeletal muscle. International journal of sport nutrition and exercise metabolism et al., 20(3), 181-190.

Grillner S (1975). Locomotion in vertebrates: central mechanisms and reflex integration. Physiol Rev. 55. 247–304.

Grob, K., Manestar, M., Filgueira, L., Kuster, M. S., Gilbey, H., & Ackland, T. (2018). The interaction between the vastus medialis and vastus intermedius and its influence on the extensor apparatus of the knee joint. Knee Surgery, Sports Traumatology, Arthroscopy, 26(3), 727-738.

Guertin P. A. (2013). Central pattern generator for locomotion: anatomical, physiological, and pathophysiological considerations. Frontiers in neurology, 3, 183.

Hamlin, M. J., & Quigley, B. M. (2001). Quadriceps concentric and eccentric exercise 1: changes in contractile and electrical activity following eccentric and concentric exercise. Journal of science and medicine in sport, 4(1), 88-103.

Han, S. W., Sawatsky, A., de Brito Fontana, H., & Herzog, W. (2019). Contribution of individual quadriceps muscles to knee joint mechanics. Journal of Experimental Biology, 222(6).

Henneman, E., Somjen, G., & Carpenter, D. O. (1965a). Excitability and inhibitibility of motoneurons of different sizes. Journal of neurophysiology, 28(3), 599-620.

Henneman, E., Somjen, G., & Carpenter, D. O. (1965b). Functional significance of cell size in spinal motoneurons. Journal of neurophysiology, 28(3), 560-580.

Hennig, R., & Lømo, T. (1987). Gradation of force output in normal fast and slow muscles of the rat. Acta Physiologica Scandinavica, 130(1), 133-142.

Herzog, W. (2017). Skeletal muscle mechanics: questions, problems and possible solutions. Journal of neuroengineering and rehabilitation, 14(1), 98.

Hessel, A. L., Lindstedt, S. L., & Nishikawa, K. C. (2017). Physiological mechanisms of eccentric contraction and its applications: a role for the giant titin protein. Frontiers in physiology, 8, 70.

Hesselink, M. K., Kuipers, H., Geurten, P., & Van Straaten, H. (1996). Structural muscle damage and muscle strength after incremental number of isometric and forced lengthening contractions., 17(3), 335-341.

Hibberd, M. G., & Trentham, D. R. (1986). Relationships between chemical and mechanical events during muscular contraction. Annual review of biophysics and biophysical chemistry, 15(1), 119-161.

Hirata, Y., Brotto, M., Weisleder, N., Chu, Y., Lin, P., Zhao, X., ... & Pan, Z. (2006). Uncoupling store-operated Ca2+ entry and altered Ca2+ release from sarcoplasmic reticulum through silencing of junctophilin genes. Biophysical journal, 90(12), 4418-4427.

Hody, S., Croisier, J. L., Bury, T., Rogister, B., & Leprince, P. (2019). Eccentric muscle contractions: risks and benefits. Frontiers in physiology, 10, 536.

Hof, A. L., & Van den Berg, J. W. (1981). EMG to force processing I: an electrical analogue of the Hill muscle model. Journal of biomechanics, 14(11), 747-758.

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(2), 492-498.

Howatson, G., Van Someren, K. & Hortobagyi, T. (2007). Repeated bout effect after maximal eccentric exercise. Int J Sports Med 28, 557–563.

Howell, J N, Chleboun, G, Conatser, R, (1993), Muscle stiffness, strength loss, swelling and soreness following exercise-induced injury in humans.. *The Journal of Physiology*, 464 doi: 10.1113/jphysiol.1993.sp019629.

Howell, J. N., Fuglevand, A. J., Walsh, M. L., & Bigland-Ritchie, B. (1995). Motor unit activity during isometric and concentric-eccentric contractions of the human first dorsal interosseus muscle. Journal of Neurophysiology, 74(2), 901-904.

Hubal, M., Rubinstein, S., & Clarkson, P. (2007). Mechanisms of variability in strength loss after muscle-lengthening actions. Medicine & Science in Sports & Exercise, 39(3), 461-468.

Hubley-Kozey, C. L., & Smits, E. (1998). Quantifying synergist activation patterns during maximal plantarflexion using an orthogonal expansion approach. Human Movement Science, 17(3), 347-365.

Hug, F., Goupille, C., Baum, D., Raiteri, B. J., Hodges, P. W., & Tucker, K. (2015a). Nature of the coupling between neural drive and force-generating capacity in the human quadriceps muscle. Proceedings of the Royal Society B: Biological Sciences, 282(1819) et al., 20151908.

Hug F., Tucker K., Gennisson J.L., Tanter M., Nordez A. (2015b). Elastography for muscle biomechanics: toward the estimation of individual muscle force. Exerc Sport Sci Rev, 43:125–33.

Huijing, P. A. (1996). Important experimental factors for skeletal muscle modelling: non-linear changes of muscle length force characteristics as a function of degree of activity. European journal of morphology, 34(1), 47-54.

Huijing, P. A. (1998). Muscle, the motor of movement: properties in function, experiment and modelling. Journal of Electromyography and Kinesiology, 8(2), 61-77.

Huijing, P. A. J. B. M., Baan, G. C., & Rebel, G. T. (1998). Non-myotendinous force transmission in rat extensor digitorum longus muscle. Journal of Experimental Biology et al., 201(5), 683-691.

Huijing, P. A., & Baan, G. C. (2003). Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force. Journal of Applied Physiology, 94(3), 1092-1107.

Huxley, H.E. & Hanson, J. (1954) Changes in the cross-striations of muscle during contraction and stretch and their structural interpretation. Nature 173, 973–976

Huxley, H.E. (1969) The mechanism of muscle contraction. Science 164, 1356–1366.

Huxley A.F., Simmons R.M. (1971) Proposed mechanism of force generation in striated muscle. Nature 233:533–538.

Ingalls, C. P., Warren, G. L., Williams, J. H., Ward, C. W., & Armstrong, R. B. (1998a). EC coupling failure in mouse EDL muscle after in vivo eccentric contractions. Journal of applied physiology, 85(1), 58-67.

Ingalls, C. P., Warren, G. L., & Armstrong, R. B. (1998b). Dissociation of force production from MHC and actin contents in muscles injured by eccentric contractions. Journal of Muscle Research & Cell Motility et al., 19(3), 215-224.

Ingalls, C. P., Wenke, J. C., Nofal, T., & Armstrong, R. B. (2004). Adaptation to lengthening contraction-induced injury in mouse muscle. Journal of Applied Physiology, 97(3), 1067-1076.

Ito, K., Komazaki, S., Sasamoto, K., Yoshida, M., Nishi, M., Kitamura, K., & Takeshima, H. (2001). Deficiency of triad junction and contraction in mutant skeletal muscle lacking junctophilin type 1. The Journal of Cell Biology, 154(5), 1059-1068.

Ivanenko, Y. P., Poppele, R. E., & Lacquaniti, F. (2004). Five basic muscle activation patterns account for muscle activity during human locomotion. The Journal of physiology, 556(1), 267-282.

Ivanenko, Y. P., Cappellini, G., Dominici, N., Poppele, R. E., & Lacquaniti, F. (2005). Coordination of locomotion with voluntary movements in humans. Journal of Neuroscience, 25(31), 7238-7253.

Ivanenko, Y. P., Cappellini, G., Poppele, R. E., & Lacquaniti, F. (2008). Spatiotemporal organization of  $\alpha$ -motoneuron activity in the human spinal cord during different gaits and gait transitions. European Journal of Neuroscience, 27(12), 3351-3368.

Johnson, M., Polgar, J., Weightman, D., & Appleton, D. (1973). Data on the distribution of fibre types in thirty-six human muscles: an autopsy study. Journal of the neurological sciences, 18(1), 111-129.

Kamandulis, S., de Souza Leite, F., Hernández, A., Katz, A., Brazaitis, M., Bruton, J. D., ... & Subocius, A. (2017). Prolonged force depression after mechanically demanding contractions is largely independent of Ca2+ and reactive oxygen species. The FASEB Journal, 31(11), 4809-4820.

Kamen, G., & Caldwell, G. E. (1996). Physiology and interpretation of the electromyogram. Journal of Clinical Neurophysiology, 13(5), 366-384.

Kanosue, K., Yoshida, M., Akazawa, K., Fujii, K. (1979). The number of active motor units and their firing rates in voluntary contraction of human brachialis muscle. Jpn. J. Physiol. 29, 427–443.

Kanzaki, K., Watanabe, D., Kuratani, M., Yamada, T., Matsunaga, S., & Wada, M. (2016). Role of calpain in eccentric contraction-induced proteolysis of Ca2+-regulatory proteins and force depression in rat fast-twitch skeletal muscle. Journal of Applied Physiology, 122(2), 396-405. https://doi.org/10.1152/japplphysiol.00270.2016

Katz, B. (1939). The relation between force and speed in muscular contraction. The Journal of Physiology, 96(1), 45-64.

Kernell, D., & Sjo, H. (1975). Recruitment and firing rate modulation of motor unit tension in a small muscle of the cat's foot. Brain research, 98(1), 57-72.

Kernell, D., Eerbeek, O., & Verhey, B. A. (1983). Relation between isometric force and stimulus rate in cat's hindlimb motor units of different twitch contraction time. Experimental brain research, 50(2-3), 220-227.

Khalaj, N., Vicenzino, B., Heales, L. J., & Smith, M. D. (2020). Is chronic ankle instability associated with impaired muscle strength? Ankle, knee and hip muscle strength in individuals with chronic ankle instability: a systematic review with meta-analysis. British journal of sports medicine, 54(14), 839-847.

Kjær, M. (2004). Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. Physiological reviews, 84(2), 649-698.

Komazaki, S., Ito, K., Takeshima, H., & Nakamura, H. (2002). Deficiency of triad formation in developing skeletal muscle cells lacking junctophilin type 1. FEBS letters, 524(1-3), 225-229.

Koo, T. K., & Li, M. Y. (2016). A guideline of selecting and reporting intraclass correlation coefficients for reliability research. Journal of chiropractic medicine, 15(2), 155-163.

Kumar, S. (2001). Theories of musculoskeletal injury causation. Ergonomics, 44(1), 17-47.

Kupa, E. J., Roy, S. H., Kandarian, S. C., & De Luca, C. J. (1995). Effects of muscle fiber type and size on EMG median frequency and conduction velocity. Journal of applied physiology, 79(1), 23-32.

Lacquaniti, F., Ivanenko, Y. P., & Zago, M. (2012). Patterned control of human locomotion. The Journal of physiology, 590(10), 2189–2199. https://doi.org/10.1113/jphysiol.2011.215137

Landau, E. M. (1978). Function and structure of the ACh receptor at the muscle end-plate. Progress in neurobiology, 10(4), 253-288.

Lauritzen, F., Paulsen, G., Raastad, T., Bergersen, L. H., & Owe, S. G. (2009). Gross ultrastructural changes and necrotic fiber segments in elbow flexor muscles after maximal voluntary eccentric action in humans. Journal of Applied Physiology, 107(6) et al., 1923-1934.

Lee, J., Goldfarb, A. H., Rescino, M. H., Hegde, S., Patrick, S., & Apperson, K. (2002). Eccentric exercise effect on blood oxidative-stress markers and delayed onset of muscle soreness. Medicine & Science in Sports & Exercise, 34(3), 443-448.

Lee, J. C., & Healy, J. (2004). Sonography of lower limb muscle injury. American Journal of Roentgenology, 182(2), 341-351.

Leger, A. B., & Milner, T. E. (2001a). Motor impairment in the human hand following eccentric exercise. European journal of applied physiology, 84(3), 213-220.

Leger, A. B., & Milner, T. E. (2001b). Muscle function at the wrist after eccentric exercise. Medicine and science in sports and exercise, 33(4), 612-620.

Levine, A. J., Lewallen, K. A., & Pfaff, S. L. (2012). Spatial organization of cortical and spinal neurons controlling motor behavior. Current opinion in neurobiology, 22(5), 812-821.

Li, X., Shin, H., Zhou, P., Niu, X., Liu, J., & Rymer, W. Z. (2014). Power spectral analysis of surface electromyography (EMG) at matched contraction levels of the first dorsal interosseous muscle in stroke survivors. Clinical Neurophysiology, 125(5), 988-994.

Lieb, F. J., & Perry, J. (1968). Quadriceps function: an anatomical and mechanical study using amputated limbs. JBJS, 50(8), 1535-1548.

Lieber, R. L., & Fridén, J. (1988). Selective damage of fast glycolytic muscle fibres with eccentric contraction of the rabbit tibialis anterior. Acta Physiologica Scandinavica, 133(4), 587-588.

Lieber, R. L., Woodburn, T. M., & Fridén, J. (1991). Muscle damage induced by eccentric contractions of 25% strain. Journal of Applied Physiology, 70(6), 2498-2507.

Lieber, R. L., & Fridén, J. (1993). Muscle damage is not a function of muscle force but active muscle strain. Journal of Applied Physiology, 74(2), 520-526.

Lieber, R. L., Schmitz, M. C., Mishra, D. K., & Fridén, J. (1994). Contractile and cellular remodeling in rabbit skeletal muscle after cyclic eccentric contractions. Journal of Applied Physiology, 77(4) et al., 1926-1934.

Lieber, R. L., & Fridén, J. (2000). Functional and clinical significance of skeletal muscle architecture. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine, 23(11), 1647-1666.

Lieber, R. L., & Fridén, J. (2001). Clinical significance of skeletal muscle architecture. Clinical Orthopaedics and Related Research (1976-2007), 383, 140-151.

Lieber, R. L. (2002). Skeletal muscle structure, function, and plasticity. Lippincott Williams & Wilkins.

Linssen, W. H., Stegeman, D. F., Joosten, E. M., Binkhorst, R. A., Merks, M. J., Laak, H. J. T., & Notermans, S. L. (1991). Fatigue in type I fiber predominance: a muscle force and surface EMG study on the relative role of type I and type II muscle fibers. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine, 14(9), 829-837.

Lowe, D. A., Warren, G. L., Ingalls, C. P., Boorstein, D. B., & Armstrong, R. B. (1995). Muscle function and protein metabolism after initiation of eccentric contraction-induced injury. Journal of applied physiology, 79(4), 1260-1270.

Maas, H., Jaspers, R. T., Baan, G. C., & Huijing, P. A. (2003). Myofascial force transmission between a single muscle head and adjacent tissues: length effects of head III of rat EDL. Journal of Applied Physiology, 95(5) et al., 2004-2013.

Maas, H., & Sandercock, T. G. (2010). Force transmission between synergistic skeletal muscles through connective tissue linkages. BioMed Research International et al., 2010.

MacKay-Lyons, M. (2002). Central pattern generation of locomotion: a review of the evidence. Physical therapy, 82(1), 69-83.

Mackey, A. L., Rasmussen, L. K., Kadi, F., Schjerling, P., Helmark, I. C., Ponsot, E., ... & Kjaer, M. (2016). Activation of satellite cells and the regeneration of human skeletal muscle are expedited by ingestion of nonsteroidal anti-inflammatory medication. The FASEB Journal, 30(6), 2266-2281.

Maeo, S., Saito, A., Otsuka, S., Shan, X., Kanehisa, H., & Kawakami, Y. (2018). Localization of muscle damage within the quadriceps femoris induced by different types of eccentric exercises. Scandinavian journal of medicine & science in sports, 28(1), 95-106.

Malm, C., Nyberg, P., Engström, M., Sjödin, B., Lenkei, R., Ekblom, B., & Lundberg, I. (2000). Immunological changes in human skeletal muscle and blood after eccentric exercise and multiple biopsies. The Journal of physiology, 529(1), 243-262. Malm, C., Sjödin, B., Sjöberg, B., Lenkei, R., Renström, P., Lundberg, I. E., & Ekblom, B. (2004). Leukocytes, cytokines, growth factors and hormones in human skeletal muscle and blood after uphill or downhill running. The Journal of physiology, 556(3), 983-1000.

Manal, K., & Buchanan, T. S. (2013). An electromyogram-driven musculoskeletal model of the knee to predict in vivo joint contact forces during normal and novel gait patterns. Journal of biomechanical engineering, 135(2).

Marder, E., & Bucher, D. (2001). Central pattern generators and the control of rhythmic movements. Current biology, 11(23), R986-R996.

Marqueste, T., Decherchi, P., Messan, F., Kipson, N., Grélot, L., & Jammes, Y. (2004). Eccentric exercise alters muscle sensory motor control through the release of inflammatory mediators. Brain research, 1023(2), 222-230.

Martin, V., Millet, G. Y., Martin, A., Deley, G., & Lattier, G. (2004). Assessment of low-frequency fatigue with two methods of electrical stimulation. Journal of Applied Physiology, 97(5) et al., 1923-1929.

Mathur, S., Eng, J., & MacIntyre, D. (2005). Reliability of surface EMG during sustained contractions of the quadriceps. Journal of Electromyography and Kinesiology, 15(1), 102-110.

McCully, K. K., & Faulkner, J. A. (1985). Injury to skeletal muscle fibers of mice following lengthening contractions. *Journal of Applied Physiology*, *59*(1), 119.

McCully, K. K., & Faulkner, J. A. (1986). Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. Journal of Applied Physiology, 61(1), 293-299.

McGowan, C. P., Neptune, R. R., Clark, D. J., & Kautz, S. A. (2010). Modular control of human walking: adaptations to altered mechanical demands. Journal of biomechanics, 43(3), 412-419.

McHugh, M. P., Connolly, D. A., Eston, R. G., & Gleim, G. W. (1999). Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. Sports medicine, 27(3), 157-170.

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. Scand J Med Sci Sports 13, 88–97.

McIntosh, E. I., Power, G. A., & Dalton, B. H. (2018). The vestibulomyogenic balance response is elevated following high-intensity lengthening contractions of the lower limb. Neuroscience letters, 675, 120-126.

Mendell, L. M. (2005). The size principle: a rule describing the recruitment of motoneurons. Journal of Neurophysiology, 93(6), 3024-3026.

Merletti, R., & Farina, D. (Eds.). (2016). Surface electromyography: physiology, engineering, and applications. John Wiley & Sons.

Meszaros, A. J., Iguchi, M., Chang, S. H., & Shields, R. K. (2010). Repetitive eccentric muscle contractions increase torque unsteadiness in the human triceps brachii. Journal of Electromyography and Kinesiology et al., 20(4), 619-626.

Michel-Pellegrino, V., Amoud, H., Hewson, D. J., & Duchene, J. (2006, August). Identification of a degradation in postural equilibrium invoked by different vibration frequencies on the tibialis anterior tendon. In 2006 International Conference of the IEEE Engineering in Medicine and Biology Society (pp. 4047-4050). IEEE.

Miles, M. P., Ives, J. C., & Vincent, K. R. (1997). Neuromuscular control following maximal eccentric exercise. European journal of applied physiology and occupational physiology, 76(4), 368-374.

Montgomery, D. C. (2017). Design and analysis of experiments. John wiley & sons.

Moore, A. D. (1966). Synthesized EMG waves and their implications.

Morgan, D. L., & Talbot, J. A. (2002). The addition of sarcomeres in series is the main protective mechanism following eccentric exercise. Journal of Mechanics in Medicine and Biology, 2(03n04), 421-431.

Morton, S. M., & Bastian, A. J. (2004). Cerebellar control of balance and locomotion. The neuroscientist, 10(3), 247-259.

Muretta, J. M., Rohde, J. A., Johnsrud, D. O., Cornea, S., & Thomas, D. D. (2015). Direct realtime detection of the structural and biochemical events in the myosin power stroke. Proceedings of the National Academy of Sciences, 112(46), 14272-14277.

Murphy, R. M., Dutka, T. L., Horvath, D., Bell, J. R., Delbridge, L. M., & Lamb, G. D. (2013). Ca2+-dependent proteolysis of junctophilin-1 and junctophilin-2 in skeletal and cardiac muscle. The Journal of physiology, 591(3), 719-729.

Murray, B. E., Froemming, G. R., Maguire, P. B., & Ohlendieck, K. (1998). Excitationcontraction-relaxation cycle: role of Ca2+-regulatory membrane proteins in normal, stimulated and pathological skeletal muscle. International journal of molecular medicine, 1(4), 677-764.

Nam, H. S., Park, D. S., Kim, D. H., Kang, H. J., Lee, D. H., Lee, S. H., ... & Choi, S. Y. (2013). The relationship between muscle fatigue and balance in the elderly. Annals of rehabilitation medicine, 37(3), 389.

Namavarian, N., Rezasoltani, A., Zavieh, M. K., Tabatabaee, S. M., Lahouti, B., & Nadimi, B. (2017). Rehabilitative Ultrasound imaging to study the gastrocnemius muscles morphology in patients with genu varum and valgum deformities. Journal of Clinical Physiotherapy Research, 2(1), 21-25.

Nardone, A., Tarantola, J., Giordano, A., & Schieppati, M. (1997). Fatigue effects on body balance. Electroencephalography and Clinical Neurophysiology/Electromyography and Motor Control, 105(4), 309-320.

Nardone, A., Tarantola, J., Galante, M., & Schieppati, M. (1998). Time course of stabilometric changes after a strenuous treadmill exercise. Archives of physical medicine and rehabilitation, 79(8), 920-924.

Newham, D. J., Jones, D. A., Ghosh, G., & Aurora, P. (1988). Muscle fatigue and pain after eccentric contractions at long and short length. Clinical Science, 74(5), 553-557.

Nosaka, K., & Clarkson, P. M. (1995). Muscle damage following repeated bouts of high force eccentric exercise. Medicine and science in sports and exercise, 27(9), 1263-1269.

Olree, K. S., & Vaughan, C. L. (1995). Fundamental patterns of bilateral muscle activity in human locomotion. Biological cybernetics, 73(5), 409-414.

Prasartwuth, O., Taylor, J. L., & Gandevia, S. C. (2005). Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. The Journal of physiology, 567(1), 337-348.

Prasartwuth, O., Allen, T. J., Butler, J. E., Gandevia, S. C., & Taylor, J. L. (2006). Lengthdependent changes in voluntary activation, maximum voluntary torque and twitch responses after eccentric damage in humans. The Journal of physiology, 571(1), 243-252.

Paschalis, V., Giakas, G., Baltzopoulos, V., Jamurtas, A. Z., Theoharis, V., Kotzamanidis, C., & Koutedakis, Y. (2007). The effects of muscle damage following eccentric exercise on gait biomechanics. Gait & Posture, 25(2), 236–242. <u>https://doi.org/10.1016/j.gaitpost.2006.04.002</u>

Pasquet, B., Carpentier, A., & Duchateau, J. (2005). Change in muscle fascicle length influences the recruitment and discharge rate of motor units during isometric contractions. Journal of neurophysiology, 94(5), 3126-3133.

Pasquet, B., Carpentier, A. & Duchateau, J. (2006). Specific modulation of motor unit discharge for a similar change in fascicle length during shortening and lengthening contractions in humans. J Physiol 577, 753–765.

Patla, A. E. (1985). Some characteristics of EMG patterns during locomotion: implications for the locomotor control process. Journal of motor behavior, 17(4), 443-461.

Paulin, M. G. (1993). The role of the cerebellum in motor control and perception. Brain, behavior and evolution, 41(1), 39-50.

Peake, J., Nosaka, K. K., & Suzuki, K. (2005). Characterization of inflammatory responses to eccentric exercise in humans.

Penfield, W., & Boldrey, E. (1937). Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. Brain, 60(4), 389-443.

Phinyomark, A., Thongpanja, S., Hu, H., Phukpattaranont, P., & Limsakul, C. (2012). The usefulness of mean and median frequencies in electromyography analysis. Computational intelligence in electromyography analysis-A perspective on current applications and future challenges et al., 195-220.

Pincivero, D. M., Salfetnikov, Y., Campy, R. M., & Coelho, A. J. (2004). Angle-and genderspecific quadriceps femoris muscle recruitment and knee extensor torque. Journal of biomechanics, 37(11), 1689-1697.

Piqueras-Sanchiz, F., Martin-Rodriguez, S., Martinez-Aranda, L. M., Lopes, T. R., Raya-Gonzalez, J., Garcia-Garcia, O., & Nakamura, F. Y. (2019). Effects of moderate vs. high isoinertial loads on power, velocity, work and hamstring contractile function after flywheel resistance exercise. PloS one, 14(2).

Place, N., Matkowski, B., Martin, A., & Lepers, R. (2006). Synergists activation pattern of the quadriceps muscle differs when performing sustained isometric contractions with different EMG biofeedback. Experimental brain research, 174(4), 595-603.

Prasartwuth, O., Taylor, J. L., & Gandevia, S. C. (2005). Maximal force, voluntary activation and muscle soreness after eccentric damage to human elbow flexor muscles. The Journal of physiology, 567(1), 337-348.

Prior, B. M., Jayaraman, R. C., Reid, R. W., Cooper, T. G., Foley, J. M., Dudley, G. A., & Meyer, R. A. (2001). Biarticular and monoarticular muscle activation and injury in human quadriceps muscle. European journal of applied physiology, 85(1-2), 185-190.

Prisk, V., & Huard, J. (2003). Muscle injuries and repair: the role of prostaglandins and inflammation. Histology and histopathology.

Prokop, T., Berger, W., Zijlstra, W., & Dietz, V. (1995). Adaptational and learning processes during human split-belt locomotion: interaction between central mechanisms and afferent input. Experimental brain research, 106(3), 449-456.

Protopapa, F., Hayashi, M. J., Kulashekhar, S., van der Zwaag, W., Battistella, G., Murray, M. M., ... & Bueti, D. (2019). Chronotopic maps in human supplementary motor area. PLoS biology, 17(3), e3000026.

Purves, D. (2018). Neuroscience. United States: Oxford University Press.

Rand, M. K., Wunderlich, D. A., Martin, P. E., Stelmach, G. E., & Bloedel, J. R. (1998). Adaptive changes in responses to repeated locomotor perturbations in cerebellar patients. Experimental brain research, 122(1), 31-43.

Rathbone, C. R., Wenke, J. C., Warren, G. L., & Armstrong, R. B. (2003). Importance of satellite cells in the strength recovery after eccentric contraction-induced muscle injury. American Journal of Physiology-Regulatory, Integrative and Comparative Physiology, 285(6), R1490-R1495.

Reeves, N. D., Maganaris, C. N., Ferretti, G., & Narici, M. V. (2005). Influence of 90-day simulated microgravity on human tendon mechanical properties and the effect of resistive countermeasures. Journal of Applied Physiology,98(6), 2278-2286. doi:10.1152/japplphysiol.01266.2004

Ren, K., & Torres, R. (2009). Role of interleukin-1beta during pain and inflammation. Brain research reviews, 60(1), 57–64. https://doi.org/10.1016/j.brainresrev.2008.12.020

Rowlands, A. V., Eston, R. G., & Tilzey, C. (2001). Effect of stride length manipulation on symptoms of exercise-induced muscle damage and the repeated bout effect. Journal of sports sciences et al., 19(5), 333-340.

Sacco, P., & Jones, D. A. (1992). The protective effect of damaging eccentric exercise against repeated bouts of exercise in the mouse tibialis anterior muscle. Experimental Physiology: Translation and Integration, 77(5), 757-760.

Saito, A., & Akima, H. (2013). Knee joint angle affects EMG–force relationship in the vastus intermedius muscle. Journal of electromyography and Kinesiology, 23(6), 1406-1412.

Sandow, A. (1952). Excitation-contraction coupling in muscular response. The Yale journal of biology and medicine, 25(3), 176.

Sasaki, K., & Neptune, R. R. (2010). Individual muscle contributions to the axial knee joint contact force during normal walking. Journal of biomechanics, 43(14), 2780-2784.

Saxton, J. M., Clarkson, P. M., James, R., Miles, M., Westerfer, M., Clark, S., & Donnelly, A. E. (1995). Neuromuscular dysfunction following eccentric exercise. Medicine and science in sports and exercise, 27(8), 1185-1193.

Schmidt, A. F., & Finan, C. (2018). Linear regression and the normality assumption. Journal of clinical epidemiology, 98, 146-151.

Scott, A., Backman, L. J., & Speed, C. (2015). Tendinopathy: update on pathophysiology. Journal of Orthopaedic & sports physical therapy, 45(11), 833-841.

Semmler, J. G., Tucker, K. J., Allen, T. J., & Proske, U. (2007). Eccentric exercise increases EMG amplitude and force fluctuations during submaximal contractions of elbow flexor muscles. Journal of Applied Physiology, 103(3), 979-989.

Semmler, J. G. (2014). Motor unit activity after eccentric exercise and muscle damage in humans. Acta Physiologica, 210(4), 754-767.

Shang, H., Xia, Z., Bai, S., Zhang, H., Gu, B., & Wang, R. (2019). Downhill Running Acutely Elicits Mitophagy in Rat Soleus Muscle. Medicine and Science in Sports and Exercise, 51(7), 1396–1403. https://doi.org/10.1249/MSS.000000000001906

Shield, A., & Zhou, S. (2004). Assessing voluntary muscle activation with the twitch interpolation technique. Sports Medicine, 34(4), 253-267.

Shin, D. D., Hodgson, J. A., Edgerton, V. R., & Sinha, S. (2009). In vivo intramuscular fascicleaponeuroses dynamics of the human medial gastrocnemius during plantarflexion and dorsiflexion of the foot. Journal of Applied Physiology, 107(4), 1276-1284.

Simmons, R. W., & Richardson, C. (1988). Peripheral regulation of stiffness during arm movements by coactivation of the antagonist muscles. Brain research, 473(1), 134-140.

Simoneau, J. A., & Bouchard, C. (1989). Human variation in skeletal muscle fiber-type proportion and enzyme activities. American Journal of Physiology-Endocrinology And Metabolism, 257(4), E567-E572.

Sleivert, G. G., & Wenger, H. A. (1994). Reliability of measuring isometric and isokinetic peak torque, rate of torque development, integrated electromyography, and tibial nerve conduction velocity. Archives of physical medicine and rehabilitation, 75(12), 1315-1321.

Solomonow, M., Baratta, R., Shoji, H., & D'ambrosia, R. (1990). The EMG-force relationships of skeletal muscle; dependence on contraction rate, and motor units control strategy. Electromyography and clinical neurophysiology, 30(3), 141-152.

Speakman H.G., Weisberg J. (1977) The vastus medialis controversy. Physiotherapy 63:249–254

Stein, R B et al. "The kinetics relating calcium and force in skeletal muscle." Biophysical journal vol. 54,4 (1988): 705-17. doi:10.1016/S0006-3495(88)83006-6

Stephenson, D. G., & Williams, D. A. (1982). Effects of sarcomere length on the force—pCa relation in fast-and slow-twitch skinned muscle fibres from the rat. The Journal of Physiology, 333(1), 637-653.

Stupka, N., Tarnopolsky, M. A., Yardley, N. J., & Phillips, S. M. (2001). Cellular adaptation to repeated eccentric exercise-induced muscle damage. Journal of Applied Physiology, 91(4), 1669-1678. https://doi.org/10.1152/jappl.2001.91.4.1669

Swenson, R. S. (2006). Review of clinical and functional neuroscience. Dartmouth Medical School.

Tax, A. A. M., Van Der Gon, J. D., Gielen, C. C. A. M., & Van den Tempel, C. M. M. (1989). Differences in the activation of m. biceps brachii in the control of slow isotonic movements and isometric contractions. Experimental Brain Research, 76(1), 55-63.

Theeuwen, M. M. H. J., Gielen, C. C. A. M., & Miller, L. E. (1994). The relative activation of muscles during isometric contractions and low-velocity movements against a load. Experimental Brain Research, 101(3), 493-505.

Tillin, N. A., Pain, M. T., & Folland, J. P. (2018). Contraction speed and type influences rapid utilisation of available muscle force: neural and contractile mechanisms. Journal of Experimental Biology, 221(24).

Timmins, R. G., Shield, A. J., Williams, M. D., Lorenzen, C., & Opar, D. A. (2016). Architectural adaptations of muscle to training and injury: a narrative review outlining the contributions by fascicle length, pennation angle and muscle thickness. British Journal of Sports Medicine, 50(23), 1467-1472.

Tsagris, M., & Pandis, N. (2021). Normality test: Is it really necessary?. American journal of orthodontics and dentofacial orthopedics, 159(4), 548-549.

U.S. Department of Labor-Bureau of Labor Statistics. (2015). Nonfatal Occupational Injuries and Illnesses Requiring Days Away From Work et al., 2015.

Van der Hoeven, J. H., Van Weerden, T. W., & Zwarts, M. J. (1993). Long-lasting supernormal conduction velocity after sustained maximal isometric contraction in human muscle. Muscle & Nerve: Official Journal of the American Association of Electrodiagnostic Medicine, 16(3), 312-320.

Verrelst, R., De Clercq, D., Willems, T. M., Victor, J., & Witvrouw, E. (2014). Contribution of a muscle fatigue protocol to a dynamic stability screening test for exertional medial tibial pain. The American journal of sports medicine, 42(5), 1219-1225.

Vijayan, K., Thompson, J. L., Norenberg, K. M., Fitts, R. H., & Riley, D. A. (2001). Fiber-type susceptibility to eccentric contraction-induced damage of hindlimb-unloaded rat AL muscles. Journal of Applied Physiology, 90(3), 770-776.

Vila-Chã, C., Hassanlouei, H., Farina, D., & Falla, D. (2012). Eccentric exercise and delayed onset muscle soreness of the quadriceps induce adjustments in agonist–antagonist activity, which are dependent on the motor task. Experimental brain research, 216(3), 385-395.

Waligora, A. C., Johanson, N. A., & Hirsch, B. E. (2009). Clinical anatomy of the quadriceps femoris and extensor apparatus of the knee. Clinical orthopaedics and related research, 467(12), 3297–3306.

Walmsley, B., Hodgson, J. A., & Burke, R. E. (1978). Forces produced by medial gastrocnemius and soleus muscles during locomotion in freely moving cats. Journal of neurophysiology, 41(5), 1203-1216.

Warren, J. A., Jenkins, R. R., Packer, L., Witt, E. H., & Armstrong, R. B. (1992). Elevated muscle vitamin E does not attenuate eccentric exercise-induced muscle injury. Journal of Applied Physiology, 72(6), 2168-2175.

Warren, G. L., Lowe, D. A., Hayes, D. A., Karwoski, C. J., Prior, B. M., & Armstrong, R. B. (1993a). Excitation failure in mouse soleus muscle injured by eccentric contractions. J Physiol, 468, 487-499.

Warren, G. L., Hayes, D. A., Lowe, D. A., & Armstrong, R. B. (1993b). Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. The Journal of physiology, 464(1), 457-475.

Warren, G. L., Hayes, D. A., Lowe, D. A., Prior, B. M., & Armstrong, R. B. (1993c). Materials fatigue initiates eccentric contraction-induced injury in rat soleus muscle. The Journal of Physiology, 464(1), 477-489.

Warren, G. L., Hayes, D. A., Lowe, D. A., Williams, J. H., & Armstrong, R. B. (1994). Eccentric contraction-induced injury in normal and hindlimb-suspended mouse soleus and EDL muscles. Journal of Applied Physiology, 77(3), 1421-1430.

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

Warren, G. L., Ingalls, C. P., Shah, S. J., & Armstrong, R. B. (1999b). Uncoupling of in vivo torque production from EMG in mouse muscles injured by eccentric contractions. *The Journal of physiology*, *515*(2), 609-619.

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. *Medicine and science in sports and exercise*, *32*(4), 820-829.

Warren, G. L., Ingalls, C. P., Lowe, D. A., & Armstrong, R. B. (2001). Excitation-contraction uncoupling: major role in contraction-induced muscle injury. Exercise and sport sciences reviews, 29(2), 82-87.

Warren, G. L., Ingalls, C. P., Lowe, D. A., & Armstrong, R. B. (2002). What mechanisms contribute to the strength loss that occurs during and in the recovery from skeletal muscle injury?. Journal of Orthopaedic & Sports Physical Therapy, 32(2), 58-64.

Whitehead, N. P., Allen, T. J., Morgan, D. L., & Proske, U. (1998). Damage to human muscle from eccentric exercise after training with concentric exercise. The Journal of physiology, 512(Pt 2), 615.

Wiest, M. J., Diefenthaeler, F., Mota, C. B., & Carpes, F. P. (2011). Changes in postural stability following strenuous running and cycling. Journal of Physical Education and Sport, 11(4), 406.

Wisdom, K. M., Delp, S. L., & Kuhl, E. (2015). Use it or lose it: multiscale skeletal muscle adaptation to mechanical stimuli. Biomechanics and modeling in mechanobiology, 14(2) et al., 195-215.

Wüst, R. C., Morse, C. I., De Haan, A., Jones, D. A., & Degens, H. (2008). Sex differences in contractile properties and fatigue resistance of human skeletal muscle. Experimental physiology, 93(7), 843-850

Yaggie, J. A., & McGregor, S. J. (2002). Effects of isokinetic ankle fatigue on the maintenance of balance and postural limits. Archives of physical Medicine and Rehabilitation, 83(2), 224-228.

Yang, J., & Winter, D. (1983). Electromyography reliability in maximal and submaximal isometric contractions. Archives of physical Medicine and Rehabilitation, 64(9), 417-420.

Yang, F., & Liu, X. (2020). Relative importance of vision and proprioception in maintaining standing balance in people with multiple sclerosis. Multiple Sclerosis and Related Disorders, 39, 101901.

Zot, A. S., & Potter, J. D. (1987). Strucutral aspects of troponin-tropomyosin regulation of skeletal muscle contraction. Annual review of biophysics and biophysical chemistry, 16, 535-559.

## **APPENDICES**

# APPENDIX A: PILOT STUDY

To answer our primary research question, we first conducted a pilot study to determine the optimal method of assessing muscle torque from the individual quadriceps muscles. Two methods were employed to measure torque: 1) Biodex dynamometer and 2) kinematic/kinetic analyses. Although we have used the Biodex to assess the muscle torque of the entire quadriceps group, the sensitivity of the Biodex to measure torque of individual injured quadriceps muscles may be at the limit of the system to yield valid and reliable data. It has been shown that submaximal force (20 Hz) of the knee extensor group can decline to up to 60% immediately following eccentric contractions (Newham et al., 1983; Kamandulis et al., 2017). If at least one of the individual injured muscles examined in the injury study has a similar loss in torque, it is important to determine whether or not the Biodex dynamometer is sensitive enough to pick up the individual muscle torque. Kinematic/kinetic analyses can also be used to calculate the torque produced from direct stimulation of an individual muscle by measuring the acceleration of the movement resulting from the contraction. However, the accuracy and reproducibility of the Vicon system to measure individual muscle force has also not been evaluated. Therefore, in this pilot study we compared the methods of measuring individual quadriceps muscle torque during submaximal and maximal tetanic contractions induced by electrical stimulation.

# **Experimental Design**

Our pilot study tested the validity and reproducibility in two methods of recording individual knee extensor torque. The torque produced by an individual muscle was compared using both kinematic/kinetic analysis and a Biodex dynamometer. The subjects reported for screening and

provided informed consent prior to completing any screening information. The subject filled out the health history questionnaire and was screened for suitability to continue the study. When the subject was cleared for participation then height, weight, blood pressure, heart rate and anthropometric measurements of the leg were conducted. We then provided the subject with a disposable razor to remove hair from areas of the thigh where we placed electrodes. Following these measurements, the subject warmed-up on the treadmill by walking at a self-chosen walking pace for 5 minutes. After the warm-up, the subjects first performed two maximal voluntary contractions (MVCs) on the Biodex at 0°/sec. Peak isometric torque produced by 3 directly stimulated muscle tetanic contractions to each muscle at a low frequency (20 Hz) and 2 at a high frequency (80 Hz) were recorded by the Biodex dynamometer. Following this, the subject's leg was removed from the apparatus used to measure torque on the Biodex and an ankle weight corresponding to 5% of their body weight was wrapped around their ankle. Reflective markers were then placed on the subject's leg. The subject then underwent 3 directly stimulated muscle tetanic contractions at a low frequency (20 Hz) and 2 at a high frequency (80 Hz) for each muscle. The direct muscle stimulation resulted in a shortening (concentric) contraction of the individual muscle and Vicon motion analysis cameras recorded the resulting knee extensions. After these recordings, the subject was scheduled for the second session of data collection within seven days following the first meeting.

On the second day of data collection, the subject warmed-up on the treadmill by walking at their self-chosen walking pace for 5 minutes. After the warm-up, the subjects first performed two maximal voluntary contractions (MVCs) on the Biodex at 0°/sec. Peak isometric torque produced by 3 directly stimulated muscle tetanic contractions to each muscle at a low frequency (20 Hz) and 2 at a high frequency (80 Hz) were recorded by the Biodex dynamometer just as the

first day. These stimulations were then repeated with the ankle weight on and recorded by the Vicon motion capture system. Accuracy and consistency of the kinematic/kinetic approach for the measurement of individual muscle torques were compared to the individual muscle torques measured via the Biodex from both time points.

## Subjects

After providing informed consent, potential subjects were screened using a health assessment form (Appendix E) to ensure that they are free of contraindications to exercise and do not have a history of traumatic lower body injuries such as ACL tears or tendinopathies. Subjects recruited for our pilot study were males ages 18 to 35 who are considered sedentary or recreationally active. Sedentary is operationally defined as spending most of the day in activities requiring minimal energy expenditure or sitting/lying and failing to achieve the American College of Sports Medicine's (ACSM) recommended weekly amount of physical activity (i.e., at least 30 minutes/day of moderate-to-vigorous physical activities at least 5 days of the week). Recreationally active will be defined as low to moderate intensity exercise up to 2 days per week for up to 30 minutes per session of exercise. Any recreationally active subject who participates in activities with repeated plyometric or jumping motions or participates in resistance training of the lower body up to once a week were excluded from our study. Subjects who are required to have medical clearance for exercise after completing the ACSM exercise preparticipation screening are excluded from the study.

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## Direct Electrical Stimulation of Quadriceps Muscle

Electrically induced torque produced by three superficial knee extensor muscles (VL, VM, RF) on the left leg were be evaluated using both kinematic/kinetic analysis and a Biodex dynamometer. Directly stimulated muscle tetanus is a widely used method to determine changes in muscle activation and force development after various types of injury and fatigue in humans (Brown et al., 1996; Mackey et al., 2016; Kamandulis et al., 2017/2019). Pairs of stimulating electrodes were placed on the skin 3 cm apart over one of the quadriceps muscles at a time. The hair on the leg was shaved, the skin where electrodes were placed was wiped with sandpaper and alcohol swabs to increase conductance. Muscles were stimulated by the voltage and current settings of a Digitimer (model DS7AH) electrical stimulator, while the stimulation frequency (20 Hz and 80 Hz) and duration (0.4 s) was set by the "Sync Train" output of a Grass S48 electrical stimulator that is connected to the trigger input of the Digitimer stimulator. Permanent markers was used identify the location of stimulating electrodes for multi-day analyses and photos with measurements were taken for reference. The subjects were seated and strapped to the Biodex chair and asked to completely relax between contractions to relieve muscle tension. The output current from the Digitimer was set to 200 mA and 200mV. After the first series of 3 lowfrequency tetanic contractions at 20 Hz, each muscle was then stimulated at a frequency of 80 Hz for 2 repetitions. Following the last contraction at the high frequency, the wires connected to the stimulator were placed on the stimulating electrodes for the next muscle. All stimulations were separated by 60 seconds of rest.

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Individual Muscle Torque Assessment (Kinematics of Individual Muscle Tetanus) As part of this pilot study, we used two methods to test force produced by the individual quadriceps muscles. We compared results from Vicon motion analysis system (OMG, Oxford, England) and a Biodex dynamometer (Biodex, Corp., Shirley, NY). Kinematic analysis was used to calculate torque produced by VL, VM and RF during individual tetanic contractions (see procedure above). The subject's thigh, chest, and waist were strapped down in the Biodex chair during knee extensor measurements. An ankle weight (5%) was placed around the subject's ankle and an ankle brace is applied to prevent movement of the ankle during contractions. The placement of the subject on the seat was recorded to standardize within subject measurements between trials. Bio-reflective markers were placed on the thigh, knee, shank, ankle and foot to measure limb acceleration and joint angle from a relaxed resting position to the end of each contraction. Peak concentric torque (T) was calculated as  $T=I\alpha+Twt$ , where I is the leg-footweight segment's moment of inertia about the knee,  $\alpha$  is angular acceleration of the same segment, and Twt is torque induced by segment's gravity. I and  $\alpha$  were obtained based on the anthropometric model and the collected kinematic data, respectively. During the assessment, each individual muscle (VM, VL, RF) will undergo 3 tetanic contractions at a low-frequency (20 Hz) and 2 at a high-frequency (80 Hz). The movement of the limb resulting from each contraction was recorded by Vicon cameras and then processed by Vicon Nexus software version 2.7 (OMG, Oxford, England). The Vicon Plug-In gate model was used to calculate lower limb kinematic data. All contractions were separated by 60 seconds of rest.

## Individual Muscle Torque Assessment (Biodex)

Participants were strapped into the Biodex using seatbelts and had their shin strapped against the padded bar on the lever arm of the Biodex dynamometer. The Biodex assessment is a unilateral

isometric contraction at 90° of knee flexion. Stimulations at low frequency (20 Hz) and high frequency (80 Hz) will produce an isometric contraction of the individual muscles. The torque values during muscle tetanus (procedure above) will be registered by the Biodex. The subject is asked to completely relax between contractions to relieve muscle tension. All contractions are separated by 60 seconds of rest.

## Maximal Voluntary Contraction Torque Assessment (Biodex)

Maximal voluntary contractile muscular strength of the quadriceps muscles was evaluated using the Biodex dynamometer. Participants were strapped into the Biodex using a seatbelt and contracted their quadriceps muscles with their shin against the padded bar of the Biodex dynamometer. Subjects performed two maximal voluntary isometric contractions at 0°/sec. The isometric contraction at 0°/sec was performed with the leg at 90° of knee flexion. One minute of recovery was given after the first contraction. Maximal voluntary torque is defined as the mean of the greatest torque values achieved from the two contractions.

## Muscle EMG

Hamstring muscle EMG data was recorded during all torque assessments. Muscle EMG was recorded using surface electrodes placed on the skin using double-sided adhesive tape and wrapping to keep electrodes in place. One EMG electrode was placed on the mid-belly of the semitendinosus and biceps femoris muscles. The EMG root mean square (RMS) was measured and used to estimate the level of muscle activation during contractions with the kinematic and Biodex evaluations of muscle torque.

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Data Analysis and Statistics

Test-retest data was analyzed using the intraclass correlation coefficient (ICC) and Altman-Bland analysis. Pearson correlations also assessed the agreement between the first and second sessions for the Biodex and the Vicon. A paired t-test was used to compare maximal voluntary contraction torque between the first and second session. All statistical analysis was performed using SPSS version 27 (IBM: Armonk, NY). Values in the results will be reported as means  $\pm$ SD. Statistical significance is set at an  $\alpha$ -level of 0.05.

## Results

## Subjects

A total of 8 subjects completed the research protocol. The average age of the subjects was  $30.3 \pm 3.9$  years. The average BMI of the subjects was  $31.0 \pm 5.0$ . There was no significant difference in the maximal voluntary contraction (MVC) torque between Day 1 and Day 2 ( $p \ge 0.30$ , Day 1 =  $307.68 \pm 34.0$ , Day 2 =  $291.78 \pm 30.5$ ).

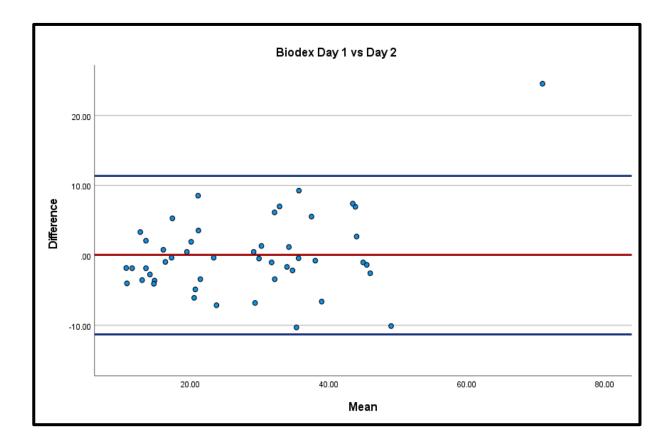
## **Repeatability Analysis**

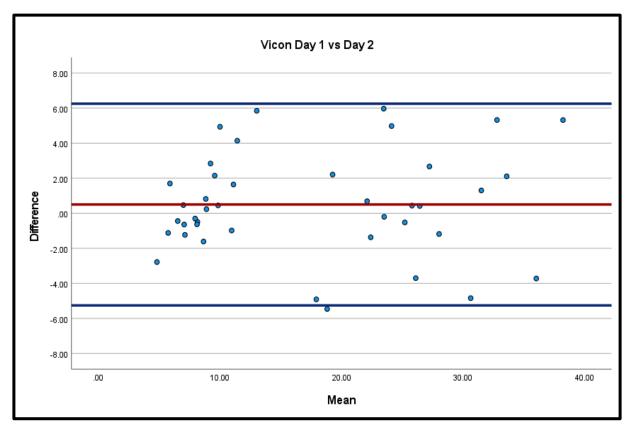
A summary of torque produced by the knee extensor muscles at low and high frequency stimulation across both days is displayed in Table 4.1. Table 4.2 shows the ICC, Cronbach's alpha and Bland-Altman for the Biodex and Vicon torque values between the two days of testing. Both methods show excellent test-retest repeatability with overall ICCs above 0.90 (Koo and Li et al., 2016). The results show a slightly higher overall ICC for the kinematic calculation of knee torque compared to the Biodex dynamometer. When accounting for stimulation frequency et al., 20 & 80 Hz torque showed a good test-retest reliability on the Biodex with ICCs of 0.86. 20 Hz stimulation as calculated by kinematic analysis was not as reliable with an ICC of 0.7 however 80 Hz stimulation scored as borderline excellent with an ICC of 0.9. A Bland-Altman plot was used to evaluate the agreement between separate days of measurement for each method. The results determined good agreement for each method with only one data point falling outside the lines of agreement for both the Biodex and the Vicon overall. For Biodex, only two data points for 20 Hz and one data point for 80 Hz fell outside of the lines of agreement when evaluated separately. Bland Altman Plots for both Biodex and Vicon are shown in Figure 4.1.

	Biodex (N•m)		Vicon (N•m)	
	Day1	Day2	Day1	Day2
VM20	14.95 (4.1)	16.48 (4.0)	8.21 (2.3)	7.7 (0.9)
VM80	35.64 (7.5)	36.51 (6.0)	25.87 (3.5)	26.07 (2.6)
RF20	19.25 (9.3)	19.71 (6.7)	9.97 (3.4)	8.88 (1.7)
RF80	45.08 (16.9)	43.13 (9.8)	32.49 (5.2)	31.35 (5.3)
VL20	18.10 (5.5)	17.5 (4.5)	8.57 (3.2)	8.03 (1.8)
VL80	35.74 (4.7)	34.98 (5.6)	22.3 (5.2)	22.67 (4.1)

Table A.2 Intraclass Correlation (ICC), Cronbach's alpha and Limits of Agreement, Pearson's R for the Biodex and Vicon torque.

	ICC (95% IC)	Cronbach's Alpha	Limits of Agreement	Correlation	Significance
	Biodex				
Total	0.95 (0.90-0.97)	0.94	$0.78 \pm 11.37$	r = 0.92	p < 0.001
20Hz	0.86 (0.67-0.94)	0.85	$-0.46 \pm 8.31$	r = 0.77	p < 0.001
80Hz	0.86 (0.67-0.94)	0.85	$0.62 \pm 13.88$	r = 0.79	p < 0.001
	Vicon				
Total	0.98 (0.96-0.99)	0.98	$0.50\pm5.76$	r = 0.96	p < 0.001
20Hz	0.70 (0.29-0.88)	0.71	$0.75\pm4.38$	r = 0.68	p = 0.001
80Hz	0.90 (0.76-0.96)	0.9	$0.19\pm 6.88$	r = 0.83	p < 0.001





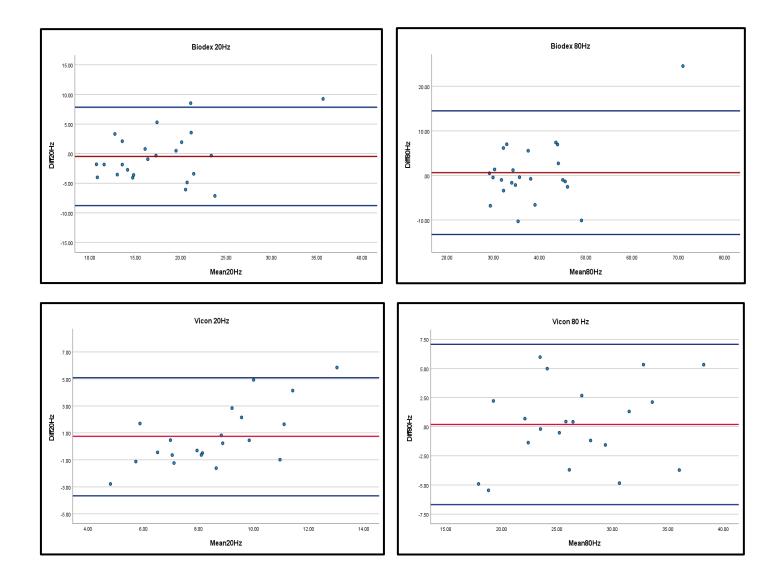


Figure A.1 Bland-Altman Plots for Biodex and Vicon motion capture. Day 1 & 2 were also evaluated for repeatability at 20 Hz and 80 Hz for both Biodex and Vicon.

# APPENDIX B: INSTITUTIONAL REVIEW BOARD APPLICATION

# Expedited/Full Study (Version 1.3)

1.0 General Information				
*Please enter the full title of your study:				
The Effect of Eccentric Contraction-Induced Injury On Individual Quadriceps Muscles				
*Please enter a short title for your own personal reference.				
Differential Exercise-Induced Injury * This field allows you to enter an abbreviated version of the Study Title to quickly identify this study.				
2.0 Add Department(s)				
2.1 Your department is listed below. Click "add" to add an additional department or select t check box next to the department and select "remove" to remove it. PLEASE DO NOT LE "GSU - Georgia State University" AS YOUR PRIMARY DEPARTMENT.:	Construction and the second			
Primary Dept?     Department Name       Image: Construction of the state o				
3.0 Assign Study Personnel				
3.1 *Please add a Principal Investigator for the study:				
Ingalls, Christopher				
3.2 If applicable, please select the Research Staff personnel (If you are adding a GSU student, staff or faculty member and their name does not appear in the list of personnel, ask that person to log-in to iRIS with his/her campus ID and password which will populate their name in the list. If you are adding personnel from outside GSU and their name does not appear in the list, they can be added with the form available at https://irbaccountrequest.gsu.edu/)				
A) Additional Investigators				
Brandenberger, Kyle Co-Investigator Otis, Jeffrey Co-Investigator Rawdon, Christopher L Student PI Yang, Feng, PhD Co-Investigator				
B) Research Support Staff				

Jackson, Mekensie H		
Student		
Middleton, Ryan C		
Student		
3.3 *Please add a Study Contact:		
Ingalls, Christopher		
Middleton, Ryan C		
Rawdon, Christopher L		
The Chudu Casherb(-) will receive all increments a shore webifications along with the Driverial		
The Study Contact(s) will receive all important system notifications along with the Principal Investigator. (e.g. The project contact(s) are typically either the Study Coordinator or the Principal		
Investigator themselves).		
4.0 Additional Personnel Information		
4.1 * Human Subjects Training is a requirement for approval. Have you and your research	team me	mbers
completed Human Subjects Training? For step-by-step directions on checking researc	h team	
members' training, please click here.		
© Yes O No		
• Yes O No		
4.2. * Polow is the DI you colorted Blocce confirm that the DI is aligible to come as	the Drin	ainal
4.2 * Below is the PI you selected. Please confirm that the PI is eligible to serve as Investigator for this study. In general, the PI must be a current, full time facult		
member (no students may serve as a PI). For more information see 4.5.3 of the	Carl Contract of C	
Christopher Ingalls		
Is the PI eligible?		
© Yes O No		
4.3 * Below is the department you selected. Please confirm that the department listed is t	ne correct	t
department for the study. GSU - Georgia State University can NOT be listed as the department	irtment.	
GSU - Kinesiology &Health		
Is this the correct department?		
⊙ Yes O No		
5.0 General Research Information		
5.1 * Describe in lay terms the purpose of the research including the research question an hope to gain.	a wnat yo	ou
	1	
Background		
Exercise-induced injury is the most common type of injury to skeletal muscle and is classified as a g		
injury. This injury is caused by eccentric contractions (i.e., active lengthening) that are used to dece		
limbs during movement. Eccentric contraction-induced muscle injury is characterized delayed onset	erate	
limbs during movement. Eccentric contraction-induced muscle injury is characterized delayed onset muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation and swelling, a		
muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation and swelling, a reduced range of motion. Although this injury is marked by minor muscle cellular damage, human ar	nd	
muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation and swelling, a reduced range of motion. Although this injury is marked by minor muscle cellular damage, human ar animal model studies indicate that maximal muscle strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after the strength can be reduced by 50% immediately after	nd id er	
muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation and swelling, a reduced range of motion. Although this injury is marked by minor muscle cellular damage, human ar	nd id er ve	
muscle soreness, minor damage to subcellular myofibrillar architecture, inflammation and swelling, a reduced range of motion. Although this injury is marked by minor muscle cellular damage, human ar animal model studies indicate that maximal muscle strength can be reduced by 50% immediately aft the performance of unaccustomed eccentric contractions and can take weeks to fully recover. We ha	nd ad er ve	

We have also shown that eccentric contraction-induced muscle injury alters the nervous activation of both injured and uninjured skeletal muscle. Moreover, increased inflammation due to eccentric exercise can decrease activity of Group IV afferent nerves altering muscle sensory motor control. Unlike more severe forms of musculoskeletal injury that may require immobilization and/or surgery, individuals with grade I muscle injuries continue with their activities of daily living and may even continue to exercise despite significant soreness and muscle weakness. However, changes in gait mechanics (i.e., stride length and frequency) during locomotion are seen following eccentric contraction-induced muscle injury. Although muscle weakness and altered tendon strain patterns are known risk factors associated with the development of overuse musculoskeletal injuries (e.g., tendinopathies), it is unclear whether the muscle weakness and/or altered muscle activation patterns associated with grade I injuries help contribute to the development of tendinopathy.

It is currently unknown whether agonistic muscles of the same muscle group become injured to the same degree when undergoing unaccustomed eccentric contractions. For example, eccentric contractions are known to injure and reduce the maximal strength the quadriceps muscles group, but whether the vastus medialis (VM), vastus lateralis (VL), vastus intermedias (VI), and rectus femoris (RF) are injured to the same extent is unclear. If differential muscle injury is present within the quadriceps muscle group and the nervous system is unable to fully compensate for differential strength deficits, then it is likely that altered stress and strain patterns will be imparted onto the quadriceps tendon and patellar ligament during locomotion.

#### Purpose

Our primary research question is the following: Will the individual muscles of the quadriceps group experience the same degree of injury after undergoing an unaccustomed bout of eccentrically-biased exercise? The goal of our study is to measure the degree of strength loss across three different knee extensor muscles (vastus medialis, vastus lateralis, and rectus femoris) following eccentric contraction-induced injury. Understanding the extent of functional deficits of individual quadriceps muscles after injury should allow for better training or rehabilitation strategies that minimize the risk associated with developing secondary soft-tissue injuries associated with the knee. We also question whether heterogeneous injury will alter quadricep muscle activation patterns (i.e., electromyography [EMG]) for maintenance of balance and locomotion. Finally, we will determine if balance will be impacted by the alterations in torque and muscle activation patterns following injury. To test these research questions, we will assess the following before and after injury: 1) torque produced by individual quadricep muscles via electrical stimulation, 2) quadricep muscle EMG during standing, walking, running and maximal voluntary contraction and 3) the postural sway during standing under different conditions by alerting the visual input (eyes-open vs. eyes-closed) and the compliance of the standing surface (firm surface vs. foam pad).

# 5.2 \* Describe how human subjects will be involved. If there is interaction with participants, describe the proposed procedures for research.

If you are using secondary data the Secondary Data Analysis application can be used. If you would like to continue with this application, describe the content of the data, the source, and clarify if all data are currently existing at this time.

Do not describe recruitment information, informed consent procedures, or confidentiality information in this section. That information is requested elsewhere in the application.

#### Experimental Design

On Day 0 of data collection, the subject will report for the initial screening and provides informed consent prior to completing any screening information. The subject then fills out the health history questionnaire and is screened for suitability to continue the study. If the subject is cleared for participation, then height, weight and anthropometric measurements of the legs will be conducted first. The subject is then familiarized with the muscle soreness, knee pain, and limb circumference measurements. Then the subject will have reflective markers placed on their lower limbs before undergoing the postural sway assessment. During the first "eyes-open" recording, a kinematic analysis of the subject's standing posture will also be conducted. Following this assessment, the subject will be instructed to walk at their "normal walking pace" between two markers that are placed 10 meters apart. Using the average time of three walking trials, we will calculate the walking velocity that will be used during locomotion assessments and the experimental protocol. We will then have the subject jog on the treadmill for roughly 5 minutes while wearing a HR monitor. The jogging speed that corresponds with roughly 70% of the subject's agepredicted maximum heart rate (HRmax) at steady state will be used for locomotion assessments and the experimental protocol. The subject is then familiarized with the maximal voluntary contractile (MVC) and individual muscle strength assessments which will be recorded on a Biodex dynamometer (Biodex Medical Systems, Inc., Shirley, NY). All settings for the Biodex dynamometer will be logged and used for all subsequent testing. Following the familiarization protocol, the subject will be scheduled for two remaining data collection sessions.

Day 1 of data collection will take place 5-7 days after Day 0, and then Day 2 will take place 48 hours after Day 1. The subject will be randomly placed into either the downhill run protocol (INJ) or control exercise protocol (CON). On Day 1, the subject returns to the lab and will be measured for baseline (Pre) muscle soreness, knee pain and limb circumference. The researchers will then apply EMG electrodes over the guadriceps, hamstring, tibialis anterior and gastrocnemius muscles which will be secured by athletic wrap. EMG data will be collected to estimate levels of muscle activation during all assessments. To prepare the skin for electrode placement, we will provide the subject with a razor to remove hair from the areas we will be placing the electrodes. In addition, we will rub sandpaper on the skin of the application sites to help remove dead skin cells and then wipe with a rubbing alcohol pad. To ensure consistency of electrode placement throughout the study, the researchers will outline the electrode placement using indelible marker. Both stimulating electrodes and EMGs are secured by athletic wrap to secure their placement on the subject. The subject will complete their pre-injury postural assessment. Then the subject completes the pre-injury locomotion assessments where EMG is recorded during three trials of walking and running on the treadmill. EMG electrodes are removed from the tibialis anterior and gastrocnemius medialis following the locomotion assessment. Following the walking and running trials, the subject will then be assessed for maximal voluntary knee extensor strength on the Biodex. The researchers will remove the EMG electrodes on the non-dominant quadriceps muscles before testing the subject's individual muscle strength via electrical stimulation. The subject will then perform the 60-minute downhill running injury protocol or the 30-minute flat walking control exercise protocol. After a 5 to 10-minute break following the completion of the experimental protocol, subjects will be measured for post-injury (Post) muscle soreness, knee pain and limb circumference. The subject will have all EMGs reapplied then undergo the post-injury postural sway and locomotion assessments. Post-injury strength assessments will conclude the data collection for Day 1. The subject is then instructed to return to the lab in 48 hours for Day 2 of data collection. On Day 2, the subject is measured for post-injury muscle soreness, knee pain and limb circumference. EMG electrodes are reapplied before completing the participant's posture assessment plus walking and running assessments. The subject is then placed on the Biodex for the 48 hours post-injury MVC strength and individual knee extensor torque assessments. This will conclude all necessary testing and data collection for the subject in this study.

#### Procedures

#### Muscle Soreness and Knee Pain

Perceived soreness of individual knee extensor muscles will be measured using a myometer using procedures similar to Newham et al., 1983.22 The force transducer of the myometer (Manual Muscle Tester, Lafayette Instrument) will be applied at the midbelly and distal portions of the vastus medialis, rectus femoris and vastus lateralis while the subject is in a supine position. The researchers will apply 45N of force to each site. The subject is then asked to rate the pain from a 1 to 100 mm visual analog scale with 1 indicating the least amount of soreness and 100 indicating the greatest amount of soreness. The average of the two sites will serve as an overall soreness score for a given knee extensor muscle. Total quadricep muscle soreness and anterior knee pain will be evaluated by having the subjects step onto and off a 12 inch box. Soreness and pain are subjective measures, so the participants will indicate their level of soreness/pain by placing a tick mark on a scale form 1-100 with 1 indicating the least amount of soreness/pain. Muscle soreness and knee pain will be measured three times per time point and the average reported.

#### Limb Circumference

Thigh circumference will be assessed at the midpoint of the anterior superior iliac spine (ASIS) and suprapatellar pica (SPP) of both legs with an anthropometric tape. Subjects will be asked to stand in a fully relaxed in the anatomical position before being instructed to put all their weight on the opposite leg while three measurements are taken. Measurement sites will be marked with indelible marker to ensure consistent measurements and the average will be reported.

#### Muscle EMG

Bilateral quadriceps muscle (VL; RF; VM), hamstring muscle (biceps femoris; semitendinosus) EMG data will be recorded during voluntary maximal isometric strength, balance assessments and gait evaluations. Additionally, tibialis anterior (TA) and gastrocnemius medialis (GM) EMG data will be recorded during balance assessments and gait evaluations. Muscle EMG will be recorded using surface electrodes placed on the skin using double-sided adhesive tape and wrapping to keep electrodes in place. One EMG electrode will be placed on the mid-belly of each quadricep, hamstring, and calf muscles (TA; GM). A total of 14 electrodes will be used: 7 on the left side and 7 on the right side of the body. The EMG root mean square (RMS) will be measured from the raw data and used to determine the level of muscle activation during maximal isometric contractions on the Biodex as well as the locomotion and balance trials.

#### Kinematic Analysis

A baseline standing and running kinematic evaluation will be conducted during the familiarization day. Reflective markers will be placed on the ASIS, mid-thigh, knee, shank, ankle and foot of both legs. The position of these markers will be measured using a nine-camera motion capture system (Vicon, UK) at 100 Hz. Marker paths will be low-pass filtered using 4th-order Butterworth filter with a cutoff frequency of 7 Hz. During the standing analysis, the subject will stand still with feet at hip width and arms at their side while marker positions are recorded. 10-seconds of gait data will be collected three times while

researchers are determining running speeds on the treadmill on Day 0. Stride length and frequency will be recorded using the Vicon Nexus system during each 10 second recording period for running. These recordings will be analyzed off-line at a later date.

#### Balance Assessment with Posturography

Balance during quiet stance will be quantified by measuring spontaneous sway as the participant stands on two side-by-side force plates. A trial of 30 seconds will be tested for each of the 3 following conditions: eyes open, eyes closed, and eyes open while standing on a block of compliant foam (10-cm thick, Aeromat Fitness Product, CA). Subjects will be told to remain as still as possible under all conditions with feet shoulder width apart and arms resting at the sides in a comfortable standing position. The bilateral ground reaction forces will be measured by the respective force plate. The center of pressure (COP) trace during each trial will be determined by the filtered ground reaction force. The total COP trajectory length will be calculated. Proprioception quotient (PQ) and Romberg's quotient (RQ) will be calculated using the COP trajectory length based on the methods previously described by Yang and Liu (2020).27 EMG data will be collected from the quadricep, hamstring, tibialis anterior and gastrocnemius muscles of both legs during this assessment. Kinematic data will be recorded via Vicon motion capture during the eyes open trials on the familiarization day as mentioned above.

#### Locomotion Assessments

Participants will begin by walking for 3 minutes at the determined speed from Day 0 testing. This initial 3 minutes of walking will be the participant's warm-up. The participant will then continue to walk at the same self-selected speed for an additional 3 minutes. Leg muscle EMG data will be recorded during the last 10 seconds of the final three minutes. The walking speed will remain constant throughout the testing period. Immediately after the walking trials, the subjects will run on the treadmill at a speed that corresponds to 70% of their age-predicted maximum heart rate determined from Day 0 testing. Subjects will warm-up at the speed that yields 70% of age-predicted heart rate for 3-minutes followed by 3-minutes of kinematic data collection. Running speeds for each participant will remain constant throughout the testing period. Leg muscle EMG data will be recorded during the last 10 seconds of the final three minutes.

#### Maximal Voluntary Contraction Torque Assessment (Biodex)

Maximal voluntary contractile muscular strength of the quadriceps muscles will be evaluated using the Biodex dynamometer. Participants will be strapped into the Biodex using a seatbelt and chest harness and will contract the quadriceps muscles with their shin against the padded bar of the Biodex dynamometer. Subjects will perform three maximal voluntary isometric contractions at 0°/sec with the leg at 20°, 45° and 90° of knee flexion. One minute of recovery will be given after each contraction. Maximal voluntary torque is defined as the greatest torque value achieved from the three contractions at each joint angle. EMG data will be collected from the non-dominant quadriceps and hamstring muscles during this assessment.

#### Direct Electrical Stimulation of Quadriceps Muscle

Electrically induced torque produced by three superficial knee extensor muscles (vastus lateralis, vastus medialis, rectus femoris) on the non-dominant leg will be evaluated using either kinematic/kinetic analysis or a Biodex dynamometer. Directly stimulated muscle tetanus is a widely used method in order to determine changes in muscle activation and force development after various types of injury and fatigue in humans.23-26 The hair on the leg will shaved, and the skin wiped with alcohol. Pairs of stimulating electrodes separated by 3 cm will be placed on the skin over one of the quadriceps muscles at a time. Muscles will be stimulated by the voltage and current settings of a Digitimer (model DS7AH) electrical stimulator, while the stimulation frequency (20 Hz and 80Hz) and duration (0.4 s) will be set by the "Sync Train" output of a Grass S48 electrical stimulator that is connected to the trigger input of the Digitimer stimulator. Permanent markers will be used identify the location of stimulating electrodes for multi-day analyses. The subjects will be seated and strapped to the Biodex chair and asked to completely relax between contractions to relieve muscle tension. A series of contractions will be used to determine the voltage and current for eliciting tetanic contractions that are typically 2-5-times that of the stimulation that causes initial muscle contraction. Specifically, the output current from the Digitimer will be incrementally increased (from 0 mA, with an incremental step of 10-20 mA) until a maximum twitch torque is reached despite an increase in current intensity. After the first series of 3 low-frequency tetanic contractions at 20 Hz, each muscle will be stimulated at a frequency 80 Hz for 2 repetitions. The order of quadriceps muscle activation will be randomized for each subject. All stimulations will be separated by 60 seconds of rest.

#### Downhill Running Protocol

Half of the subjects will perform the downhill running protocol (INJ) on Day 1. The downhill running protocol is designed to induce injury through a series of repeated eccentric contractions. Subjects will complete 60 minutes of downhill running on a -14% at a HR range of 70-85% of their age-predicted HR max. Subjects will be granted a break every 10-15 minutes, each break lasting a maximum 5 minutes.

#### Control Exercise Protocol

Half of the subjects will perform the control walking protocol (CON) on Day 1. Reduced times of level

treadmill walking are chosen to serve as an exercise control that would likely minimize the risk of recreational participants from experiencing muscle injury from running for 60 minutes. Subjects will complete 30 minutes of level walking (0%) at a speed which will be calculated based on their normal walking pace for 10 meters. Subjects will be granted a break every 10-15 minutes, each break lasting a maximum 5 minutes.

#### Statistics

Power analysis of pilot data estimating expected difference of 25% in the 80 Hz pre-post torque ratio between groups at an alpha level of 0.05 and a power of 0.8 has indicated a minimum sample size of 16 subjects for this study. Separate group (downhill vs. level walking) by time (Pre, Post, 48h) by muscle (VL, VM, RF) analysis of variance (ANOVA) with repeated measures on time, will be used to evaluate changes in individual muscle soreness, torque via 20 Hz/80 Hz electrical stimulation, and muscle activation (EMG RMS) during the posture and locomotion trials as well as maximal strength testing. Separate group (downhill vs. level walking) by time (Pre, Post, 48h) by knee joint angle (20, 45, 90 degrees) ANOVA with repeated measures on time and knee joint angle will be used to evaluate changes in maximal voluntary knee extensor strength. Separate group (downhill vs. level walking) by time (Pre, Post, 48h) ANOVA will be used to evaluate changes in total muscle soreness, knee pain, thigh circumference and COP for each of the three standing conditions. We will use a ratio of a muscle's capacity of producing torque following injury (T2) to the torque produced prior to injury (T1). This formula is calculated as: r = T2 / T1. From this ratio we can thus determine variability between the three muscles. With this variability metric, we can capture the heterogeneity of the impact of the injury protocol on the torque production capacity amongst the muscles of the quadriceps group. In the event of significant statistical interactions, simple main effect analyses will be used. Bonferonni test will be used as the post-hoc test when indicated. An a-level will be set at 0.05. Statistical analysis will be conducted using SPSS (IBM) version 27 and Excel (Microsoft Office 16).

The research team will observe restrictions imposed by Georgia State University and relevant government or public health authorities in the conduct of research activities.

5.3 \* State who will be conducting each of the procedures detailed above. If there are multiple procedures or populations, be sure to state who will be conducting each procedure or working with each population.

Only individuals listed on the application will be interacting with the subjects. Each person on this application has been trained in the measurement of muscle strength, EMG and kinematics. The student investigator (Chris Rawdon) will be the person primarily collecting data with the assistance of Mekensie Jackson. Dr. Chris Ingalls will assist or conduct the procedures when needed. Drs. Yang, Brandenberger and Otis will assist when needed.

5.4 \* Will the study involve interaction with participants? Interaction includes any contact with people including, but not limited to, online interaction, survey distribution, or hiring a company or third person that will interact with people.

• Yes O No

## 6.0 Funding, Dissertation, or Protocol

### 6.1 \* Will the research be funded?

O Yes 💿 No

A research protocol must be uploaded in the study document section during submission. If a study plan is included in the thesis, dissertation, or prospectus, it can be uploaded to meet this requirement.

## 6.2 \* Is this study or any part of the study contributing to a dissertation, thesis, or capstone project?

## • Yes O No

Is this research being conducted ONLY for a dissertation, thesis, or capstone project?

0	Yes 💿 No	
7.0	Study Information	
7.1	* Will this study be submitted to another IRB for review and approval?	
0	Yes 💽 No	
7.2	* Will another institution rely on GSU IRB's review for any research activity?	
0	Yes 💿 No	
7.3	* Does your study involve the use of Protected Health Information (PHI) as such term is define HIPAA, obtained from a Covered Entity? For more information on the definitions of PHI and Co Entity or other terms related to HIPAA, please see the IRB Manual at gsu.edu/irb.	
0	Yes 💿 No	
7.4	* Will the study involve the use or possible exposure to infectious or potentially infectious mate (e.g. blood, bodily fluids, mucosal swabs, tissue samples, etc.)	erial?
0	Yes 💿 No	
7.5	* Does the study involve the use of non-human animals? (e.g. dogs, mice, non-human primates	, etc.)
0	Yes 💿 No	
7.8	* Will your study involve data from student education records (e.g. class work, grades, attenda records, communications, projects, classroom tests, standardized tests, journals, SAT/ACT scoretc.) that are protected by FERPA? This list is not exhaustive. Please see section 1.6 of the IRB manual for more information on FERPA records.	
0	Yes 💿 No	
8.0	Location	
8.1	* Will the study be conducted outside of the United States?	
0	Yes 💿 No	
8.2	* Is there a research location located outside of Georgia State University?	
0	Yes 💿 No	
9.0	Investigational Information	
9.1	* Will the study involve the use of FDA approved drugs? Please note: GSU's IRB can only review studies that use FDA approved drugs for approved uses Please contact the IRB office if you are using a drug not approved by the FDA.	
0	Yes 💿 No	

9.2 * Will the study involve an investigational device?			
O Yes 💿 No			
9.3 * Will the study involve Radiation or Lasers?			
O Yes 💿 No			
10.0 Additional Information			
10.1 * Will the study involve deception or concealment of any information?			
O Yes 💿 No			
10.2 Do any research personnel need special certifications, training, or special qualifications to conduct the research procedures? If so the individual's name and qualifications should be listed along with any certification or licensure number and dates of qualification. This includes studies that utilize venipuncture, EKGs, direct patient care, CPR, EEGs, and studies involving clinical psychologists, physicians, nurses, physical therapists, and others.			
O Yes ⊙ No O N/A			
11.0 Vulnerable Populations			
11.1 * If you are including women, are you recruiting pregnant women because they are pregnant			
you including any procedures that could be more than minimal risk for a pregnant woman or f			
<ul> <li>you including any procedures that could be more than minimal risk for a pregnant woman or f</li> <li>Yes</li> <li>No, I am including women of childbearing age, but the study includes no procedures that are more than minimal risk for the participant or fetus.</li> <li>No, I am excluding women of childbearing age (a study specific justification must be provided elsewhere in the application).</li> <li>No, I am excluding pregnant women (a study specific justification and procedures for the exclusion</li> </ul>			
<ul> <li>you including any procedures that could be more than minimal risk for a pregnant woman or f</li> <li>Yes</li> <li>No, I am including women of childbearing age, but the study includes no procedures that are more than minimal risk for the participant or fetus.</li> <li>No, I am excluding women of childbearing age (a study specific justification must be provided elsewhere in the application).</li> <li>No, I am excluding pregnant women (a study specific justification and procedures for the exclusion must be included in the application)</li> </ul>			
<ul> <li>you including any procedures that could be more than minimal risk for a pregnant woman or f <ul> <li>Yes</li> <li>No, I am including women of childbearing age, but the study includes no procedures that are more than minimal risk for the participant or fetus.</li> <li>No, I am excluding women of childbearing age (a study specific justification must be provided elsewhere in the application).</li> <li>No, I am excluding pregnant women (a study specific justification and procedures for the exclusion must be included in the application)</li> </ul> </li> <li><b>11.2</b> * Are you including any students or trainees in your research?</li> <li>Yes, participants are the students or trainees of a researcher.</li> <li>Participants may be students or trainees, but they are not the students or trainees of anyone on the</li> </ul>			
<ul> <li>you including any procedures that could be more than minimal risk for a pregnant woman or f <ul> <li>Yes</li> <li>No, I am including women of childbearing age, but the study includes no procedures that are more than minimal risk for the participant or fetus.</li> <li>No, I am excluding women of childbearing age (a study specific justification must be provided elsewhere in the application).</li> <li>No, I am excluding pregnant women (a study specific justification and procedures for the exclusion must be included in the application)</li> </ul> </li> <li><b>11.2 * Are you including any students or trainees in your research?</b></li> <li>Yes, participants are the students or trainees of a researcher.</li> <li>Participants may be students or trainees, but they are not the students or trainees of anyone on the research team.</li> </ul>			
<ul> <li>you including any procedures that could be more than minimal risk for a pregnant woman or f</li> <li>Yes</li> <li>No, I am including women of childbearing age, but the study includes no procedures that are more than minimal risk for the participant or fetus.</li> <li>No, I am excluding women of childbearing age (a study specific justification must be provided elsewhere in the application).</li> <li>No, I am excluding pregnant women (a study specific justification and procedures for the exclusion must be included in the application)</li> <li>11.2 * Are you including any students or trainees in your research?</li> <li>Yes, participants are the students or trainees, but they are not the students or trainees of anyone on the research team.</li> <li>Yes, participants are the employees or subordinates?</li> <li>Yes, participants are the employees or subordinates of someone on the research team.</li> <li>Participants may be employees or subordinates, but they are not the employees or subordinates of</li> </ul>			

⊙ No	
11.5 * Are you using prisoners in your study?	
O Yes No	
11.6 * Are you using children (ages 0-17 in Georgia) in your research?	
O Yes ⊙ No	
11.7 * Are you including any adults that may be cognitively or decisionally impaired?	
O Yes O No	
12.0 Population Data	
12.1 * Will enrollment be limited to a specific ethnic, social, or gender group? If so, describe and ju	istify.
In Yes O No ○ Yes O No The study will be limited to male participants ages 18 to 35 who do not have a history of knee joint injuries and are classified as low-risk. Research shows that men and women may respond differently to exercise induced injury (e.g. Sewright et al., Med Sci Sports Exers. 40(2): 242-251, 2008) and that maximal muscle strength of females can fluctuate up to 10% during the month based on hormonal changes (e.g., Phillips et al., J Physiol. 496:551-557, 1996). Since our main research question involved the measurement of maximal isometric knee torque we will be excluding females from this pilot study. If significant findings are found, future planned studies can address the question of differential muscle injury in females as well.	
12.2 * Total number of participants (You cannot enroll more than the total number of participants an amendment.)	without
16	
12.3 * Total number of participants per a year	
16.00	
12.4 * Justification for the number of participants	
Power analysis of pilot data estimating expected difference of 25% in the 80 Hz pre-post torque ratio between groups at an alpha level of 0.05 and a power of 0.8 has indicated a minimum sample size of 16 subjects for this study.	
12.5 * What will be the age range(s) of the participants?	
<ul> <li>□ 0-17</li> <li>☑ 18-89</li> <li>□ 90 and above</li> </ul>	
12.7 * What is the time commitment for each participant? If you are using multiple populations, pr	ovide



There will be three different sessions in total. The first session will last approximately two hours, the second session will last approximately four hours and the third session will last approximately two hours. The total subject participation is expected to be eight hours across the three days.

12.8 \* Describe where the procedures will take place and how privacy will be maintained while conducting procedures. If you are conducting multiple procedures or using multiple populations, be sure to describe where each interaction will take place. Please Note: If research is to be conducted off site and not at a public location, you MUST submit the approval letter from the site stating that the research may be conducted there.

All research will take place in the Georgia State University Biomechanics Laboratory located in G15 of the Sports Arena. The informed consent and health history questionnaire will be completed in the biomechanics room. Data collection in the labs will be completed only in the presence of the research faculty listed on this protocol and/or by Chris Rawdon or Mekensie Jackson (student investigators). Only after students have been added to the approved protocol and approved by the IRB will other graduate students be allowed to be present and assist with data collection. Please note that the biomechanics lab has an office for a small number of graduate students that may not be part of the research project, but this with a door is in the corner of the lab and not visible (or audible when the door is closed) from the testing sight in the lab.

- 12.9 \* Federal regulations require that you include minors (e.g. participants aged 0 17) in your research unless you can justify their exclusion. Are you including minors? If not, check the appropriate box and provide a justification specific to this study in the text box.
- O No, inappropriate due to lack of safety data in studies conducted in adults
- O No, inappropriate with respect to the purpose of the research

O Other

- O Yes, minors are included
- \* Please provide justification for not including minors in your study if applicable.

It is possible that the repeated measurement of maximal strength in subjects younger than 18 years of age, who likely have immature bone growth plates may increase the risk of inducing skeletal injuries (e.g. alvulsion fracture).

12.10 \* Federal regulations require that you include minorities (i.e. minority ethnic, racial, gender groups, etc.) in your research unless you can justify their exclusion. Are you including minorities? If not, describe and provide a justification specific to this study

- O No, minorities are not included
- Yes, minorities are included

#### 13.0 Recruitment

13.1 \* Describe in detail the recruitment plan. Who will be recruited and how (i.e. will the study use a subject pool, announcements, recruitment ads, email, etc.?) If materials such as flyers, emails, advertisements, screen shots from websites, or any other recruitment material is used, it must be uploaded with this application. Do not use the terms 'word of mouth' or 'snowball sampling'. Instead, describe what you will be doing to let people know about the study and how you will invite them to participate.

The participants for will be recruited are sedentary or recreationally active males 18-35 years of age. The researchers will contact known associates and classmates via email and in person for recruitment. The researchers will also post recruitment flyers in common areas. Georgia State students recruited for the study will be informed that they will not receive any credit towards any of their coursework including extra credit. The recruitment script will be as followed:

#### "Dear [subject name].

My name is \_\_\_\_\_\_\_, and I am a [status - graduate student; faculty member] in the Department of Kinesiology and Health Sciences at Georgia State University. I am conducting a research study examining exercise induced injury in skeletal muscle and you are invited to participate in the study. Exercise induced injury is a low grade injury where no architectural distortion of muscle tissue occurs but is characterized by soreness and strength loss in the days that follow unaccustomed exercise. We are determining whether muscles of the same group have the same degree of strength loss and soreness following exercise induced injury. If you agree, you will take part in different walking, running, balance and strength assessments as well as have the strength of your individual quadriceps muscles measured repeatedly via electrical stimulation.

You can participate in this study if you are a male between the ages of 18-35 and are currently sedentary or are recreationally active. If you have a cardiovascular, metabolic or renal disease or have any signs or symptoms of these diseases then you cannot participate. If you have had a traumatic lower body injury (e. g., ligament tear, fracture) you will be excluded from the study. Your total participation for the study is anticipated to be approximately 8 hours total on 3 separate days that work with your schedule. Participation in this study is voluntary. Your identity as a participant will remain anonymous during and after the study.

If you have questions or would like to participate please contact me at [email/phone number]. If you qualify to participate, we will schedule your first session at Georgia State University.

Thank you, [Researcher]"

### 13.2 \* Describe the inclusion and exclusion criteria. State how the inclusion/exclusion criteria will be determined.

Inclusion criteria: sedentary or recreationally males age 18-35 that are cleared for exercise according to the American College of Sports Medicine Preparticipation Screening. Exercise clearance is determined by using the health history questionnaire. Individuals with no current signs or symptoms for cardiovascular or metabolic disease and do not have a known cardiovascular or metabolic disease are cleared to participate.

Exclusion criteria: Females of any age are excluded from the study. The reasoning for this is outlined in Section 12.1. History of significant lower body injury (e.g. knee ligament tears); classified as having signs or symptoms of cardiovascular or metabolic disease on the health history questionnaire; pre-existing heart condition (e.g. heart disease); pre-existing pulmonary condition (e.g. asthma); pre-existing metabolic condition (e.g. diabetes); currently on medications for a heart issue (e.g. amoldipine); participates in low to moderate exercise more than 3 times a week for 30 minutes per session; participates in high-intensity exercise or activities that are plyometric, involved repetitive jumping or resistance training of the lower body. Potential subjects will be asked the above criteria on the health history questionnaire.

13.3 \* Will participants be compensated or incur any costs for their participation? If so, provide details of the compensation (i.e. what the compensation is, the total amount, etc.). Compensation might include money, gifts, food, class credit, or extra credit provided for participation. Any costs to the subjects that may result from participation in the research should also be described. Detail what compensation participants will be given if they do not complete the study. If extra credit is given, describe the assignment of equal difficulty and length that will be provided for the same amount of credit if students wish to not participate in the research. If a lottery or drawing will be used, specific information must be provided to ensure it meets requirements in GSU policy and state law.

#### • Yes O No

There are no financial costs for the subjects. Subjects will receive one \$25 gift card for the session on day 0 and one \$25 gift card for the session on day 1. The subjects will receive a \$50 gift card for the session on day 2. If a subject is withdrawn from the study, they will still receive the gift card for their visit that day.

We have submitted a grant to cover the cost of the subject stipends. If the pending grant is not funded, then stipends will be provided by personal funds of the student investigator and Dr Ingalls.

#### 14.0 Benefits & Risks

#### 14.1 \* Describe the benefits, if any, to the participants and to society from the proposed research. Compensation is not a benefit of participating in research.

Please note: The benefits and risks described in the application must match the benefits and risks described in the informed consent form.

This study is not designed to benefit subjects personally. Overall, we hope to gain information about the effects of exercise-induced injury on the individual quadriceps muscles. We also hope to learn how these changes may impact neuromuscular control while standing upright and during locomotion. Understanding the extent of functional deficits of individual quadriceps muscles after injury should allow for better training or rehabilitation strategies that minimize the risk associated with developing secondary soft-tissue injuries associated with the knee. Minimizing the risk of injury encourages individuals to maintain weekly physical activity to improve overall health and quality of life.

14.2 \* Describe the risks or discomforts, if any, to the participants, whether physical, psychological, or social, and the means proposed to minimize them. If participants may become upset or require medical or psychological attention as a result of the research procedures, a means of addressing attention to these concerns should be described in this section. A participant is at risk in research if he or she may be exposed to a possibility of harm that is greater than that ordinarily encountered in daily life or during routine examinations or tests. Each investigator should make a conscientious assessment of possible harms and disclose them to the IRB.

Some subjects may perceive the electrical stimulation of the skeletal muscle and nerves in their thigh as uncomfortable. However, there is no harm or damage being done to the skin or muscle tissues during stimulation. We will ask the subject for feedback on their perceived pain following each stimulation. The subject will be encouraged to vocalize at any point if they deem the stimulation to be intolerable. If the subject determines that the stimulation is intolerable, the subject will be withdrawn from the study. Any subject who wishes to terminate participation at any time is free to do so.

Shaving cream will be provided to the subjects to avoid injury or cuts in preparation for the electrodes.

The downhill running protocol used in this study is designed to injure certain muscle groups of the legs and will result in soreness within the affected muscles. Participants in the experimental group (i.e., downhill running) will experience some discomfort of their quadriceps (i.e., anterior thigh) and ankle plantar flexor (i.e., calves) muscles starting 12-24 hours after the exercise and the soreness may persist for several days (delayed onset muscle soreness, DOMS). The soreness usually peaks 48 hours after the induction of the muscle injury and subsides thereafter. This pain should be no greater than what someone would experience from an unaccustomed bout of exercise. The subjects will be visually monitored through the test and asked every 5-10 minutes whether they have symptoms including breathlessness, chest pain, nausea, blurred vision, or swelling. In the event of any signs of symptoms mentioned above, the treadmill will be stopped, and testing terminated. In addition, subjects may fall off the treadmill during the test. To minimize this risk, a spotter will stand adjacent to the treadmill to visually monitor the subject and catch them if they fall.

The risks associated with running at 70-85% of the subject's age predicted maximal heart rate include acute myocardial infarction, cardiac arrest, cardiac arrhythmia, stroke, and musculoskeletal injury. However, the risks of these events are extremely small in an apparently healthy population that does not have elevated risk of heart disease. To ensure that subjects are at low risk for these adverse events, the subjects' ages have been restricted to 18-35, the subjects will undergo a risk assessment according to the procedure outlined by the American College of Sports Medicine. The subjects are constantly monitored, and procedures will be terminated if any symptoms listed above are experienced by the subject or if they request termination. Although unlikely, in the case of a life-threatening emergency, researchers will contact Georgia State University police for an ambulance and medical assistance.

There is also a risk of COVID-19 transmission between researchers and subjects. However, we will take the following steps to minimize the spread of COVID-19 between individuals. Upon arrival in the lab space, subjects will be advised to wash their hands for 20 seconds with soap and water or use hand sanitizer. During the experiment, at least six feet distance between researchers and participants will be observed (except when applying the sensors and stimulating electrodes to participants' skin, and anthropometric measurements). The number of researchers present with visitors will be limited to those essential to carry out protocol-defined research procedures.

Lab equipment and chairs/tables to be used in this project will be cleaned using Lysol spray and wipe with paper towel after each completion of the experimental session.

We will strictly follow the restrictions posted by GSU and relevant government or public health authorities in the conduct of research activities, such as at least 6-foot distance between persons and face coverings for those not fully vaccinated, washing hands, cleaning equipment, etc. During some of the data collection procedures, we need to manually apply sensors and electrodes to participants' skin, as well as make anthropometric measurements. The standard precautions (especially the 6-foot distancing) cannot be observed during this process. To address this, we will adopt the following additional measures to reduce the health risk:

· Only one researcher will apply the sensors to participants' skin

 $\cdot$  This researcher will wear a face covering

• The total time that the researcher will be within 6 feet of the participant will be less than 15 minutes during all of these procedures

All participants, visitors, and researchers will be instructed to wash their hands for 20 seconds with soap and water or use hand sanitizer before exiting the research site.

#### 15.0 Participant Data

15.1 \* Will information that personally links the participants to the research be collected?

#### • Yes O No

If **Yes**, state what identifying information will be collected. Identifying information includes (but is not limited to) name, social security or student ID number, date of birth, contact information including email address or phone number, photographs, and audio or video recordings.

Subjects' name, email, address, phone number and date of birth will be collected.

#### 15.2 \* Will photographs, audio recordings, or video recordings be used?

#### • Yes • No

If **Yes**, describe and provide information how any special precautions used to protect photographs, audio or video recordings.

The Vicon motion capturing system will be used to record the subjects movement. However, the cameras used by the motion capturing system only will pick-up the reflective markers placed on the participant's anatomical landmarks and do not reveal the subject's identity.

15.3 \* State where and how any data will be collected, stored, and transported; who will have access to the data and what will be done with it after the study is over; protections for storing or sharing hardcopy and electronic data (flash drive, cloud storage, Drop Box, etc.) If a code sheet will be used to separate identifying information from the participant data describe the means of protecting this document.

If identifiable data are inadvertently collected, please state how it will be managed.

The informed consent and the health history questionnaire used screening will be stored in a locked filling cabinet in Dr. Ingalls's office (G05 of the Sports Arena). The documents will be transported by hand from the lab to the office or from the office to the lab. Research data will be collected via two different computer workstations (Vicon motion capture software & Biodex strength testing system) in the Biomechanics laboratory. The subjects will be assigned a study identification (ID) number and their names and IDs will be temporarily stored on a password protected lab computers, an office (Dr. Ingalls) computer, and a flash drive. The flash drive will be stored in Dr. Ingalls's locked office when not being used. Drs. Ingalls, Yang, Otis and Branderberger as well as Mr. Christopher Rawdon will have access to the data. A separate code sheet will be stored in a filing cabinet in Dr. Ingalls' office. Dr. Ingalls will be the only researcher to have key access to his office and the filing cabinet. When another member of the research staff needs to access this document, they will be accompanied by Dr. Ingalls. Once a subject has completed the study and their ID is stored on the computer in Dr. Ingalls's office, their patient name and associated data will be deleted from all other lab computers. Once all the data collection for the study is completed and data stored in Dr. Ingalls's office computer, the code sheet matching their name with the subject ID will be destroyed.

#### 16.0 Review Categories

16.1 Review Categories

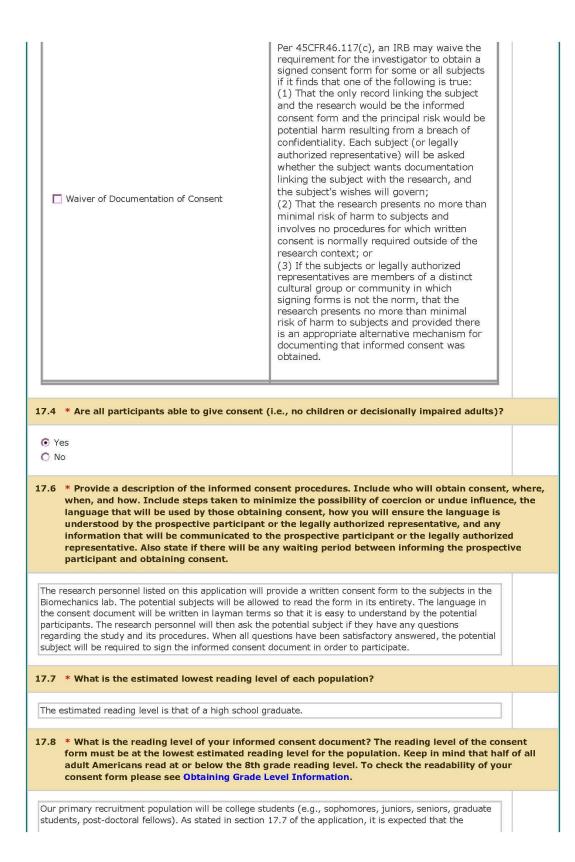
elect Category	Description
▼ Full Board Review	More than minimal risk/does not meet other categories' requirements
	Clinical studies of drugs and medical devices only when condition (a) or (b) is met.
	(a) Research on drugs for which an investigational new drug application (21 CFR Part 312) is not required. (Note: Research on marketed drugs that significantly increases the risks or decreases the acceptability of the risks associated with the use of the product is not eligible for expedited review.)
Expedited - Category 1	(b) Research on medical devices for which
	(i) an investigational device exemption application (21 CFR Part 812) is not required; or
	(ii) the medical device is cleared /approved for marketing and the medical device is being used in accordance with its cleared/approved labeling.
Expedited - Category 2	Collection of blood samples by finger stick, heel stick, ear stick, or venipuncture as follows:
	(a) from healthy, nonpregnant adults who weigh at least 110 pounds. For these subjects, the amounts drawn may not exceed 550 ml in an 8 week period and collection may not occur more frequently than 2 times per week; or
	(b) from other adults and children, considering the age, weight, and health of the subjects, the collection procedure, the amount of blood to be collected, and the frequency with which it will be collected. For these subjects, the amount drawn may not exceed the lesser of 50 ml or 3 ml per kg in an 8 week period and collection may not occur more frequently than 2 times per week.
	Collection of biological specimens by noninvasive means. Examples are:
	(a) hair and nail clippings;
	(b) teeth routinely shed or extracted;
	(c) excreta and external secretions;
	(d) uncannulated saliva;
Expedited - Category 3	(e) placenta removed after delivery;
	(f) amniotic fluid collected in accordance with accepted prophylactic techniques;
	(h) mucosal or skin cells collected by scraping,

	(i) sputum collected after saline mist nebulization
	Collection of data through noninvasive procedures (not involving general anesthesia or sedation) routinely employed in clinical practice, excluding procedures involving x-rays or microwaves. Where medical devices are employed, they must be cleared/approved for marketing. (Studies intended to evaluate the safety and effectiveness of the medical device are not generally eligible for expedited review, including studies of cleared medical devices for new indications.) Examples:
Expedited - Category 4	(a) physical sensors that are applied either to the surface of the body or at a distance and do not involve input of significant amounts of energy into the subject or an invasion of the subject's privacy;
	(b) weighing or testing sensory acuity;
	(c) magnetic resonance imaging;
	(d) electrocardiography, electroencephalography, thermography, detection of naturally occurring radioactivity, electroretinography, ultrasound, diagnostic infrared imaging, doppler blood flow, and echocardiography;
	(e) moderate exercise, muscular strength testing, body composition assessment, and flexibility testing where appropriate given the age, weight, and health of the individual.
Expedited - Category 5	Research involving materials (data, documents, records, or specimens) that have been collected, or will be collected solely for nonresearch purposes (such as medical treatment or diagnosis). (NOTE: Some research in this category may be exempt from the HHS regulations for the protection of human subjects. 45 CFR 46.101(b)(4). This listing refers only to research that is not exempt.)
Expedited - Category 6	Collection of data from voice, video, digital, or image recordings made for research purposes.
Expedited - Category 7	Research on individual or group characteristics or behavior (including, but not limited to, research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices, and social behavior) or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies. (NOTE: Some research in this category may be exempt from the HHS regulations for the protection of human subjects. 45 CFR 46.101 (b)(2) and (b)(3). This listing refers only to research that is not exempt.)

### 17.0 Informed Consent

17.1	* Directions: Check all applicable consent procedures.	These procedures must be approved by the
	IRB.	

ame	Description
✓ Signed Consent Required	Signed consent will be sought from the subject or the subject's legally authorized representative.
Waiver of Consent or Waiver/Alteration of the required elements of consent	Per 45 CFR 46.116(f), an IRB may approve a consent procedure which does not include, or which alters, some or all of the elements of informed consent provided the IRB finds and documents that: (1) The research involves no more than minimal risk to the subjects; (2) The research could not practicably be carried out without the requested waiver or alteration; (3) If the research involves using identifiable private information or identifiable biospecimens, the research could not practicably be carried out without using such information or biospecimens in an identifiable format; (4) The waiver or alteration will not adversely affect the rights and welfare of the subjects; and (5) Whenever appropriate, the subjects or legally authorized representatives will be provided with additional pertinent information after participation. and (6) the research is not FDA-regulated OR Waiver of Consent Process-Demonstration Project (1) The research or demonstration protocol is designed to study, evaluate, or otherwise examine: Public benefit or service programs. Prosedures for obtaining benefits or services under those programs. Possible changes in or alternatives to those programs or procedures. Possible changes in or alternatives to those programs or procedures. Possible changes in or alternatives to those programs or procedures. Possible changes in or alternatives to those programs. (3) The research cannot practicably be carried out without the waiver or alteration. and (4) The research is not FDA-regulated



students and employees of Georgia State University that are recruited for this study will be at minimum high school graduates (i.e., reading level of 12). The informed consent used in our pilot study for subjects aged 18-35 had a Flesch-Kincaid Grade Level of 10.6. The reading level of our current informed consent of 10.8 is sufficiently below the expected reading level of our target population.

17.9 \* Does the population include participants that are non-English speaking?

O Yes ⊙ No

#### **18.0 Conflict of Interest**

18.1 \* Does the PI, Co-Investigators, or other research staff including their spouse and dependents have a significant financial conflict of interest defined as: - An equity interest that, when aggregated for the Investigator or research staff and their spouse or dependents meets all of the following tests: Exceed \$5,000 in value as determined through reference to public prices or other reasonable measures of fair market value, represents more than a 5% ownership interest in any single entity, and value is affected by the outcome of the research; or - Salary, royalties or other payments that, when aggregated for the Investigator or research staff and their spouse and dependents over the next 12 months, are expected to exceed \$5,000 and value is affected by the outcome of the research.

O Yes 💿 No

18.2 \* Does the PI, Co-Investigators, or other research staff including their spouse and dependents have: - A board or executive relationship related to the research regardless of compensation. - Proprietary interest related to the research including by not limited to a patent, trademark, copyright, or licensing agreement.

O Yes 💿 No

#### 19.0 Endorsement

**19.1** \* Please affirm the following endorsement statements:

- I will not begin this research study before receiving a formal letter of IRB approval;
- I will document informed consent according to my approved procedure;
- I will notify GSU IRB through the iRIS system of any non- compliance, deviations, unanticipated problems, or suspensions/terminations;
- I will renew my IRB application before expiration (if applicable), submit a status check form, or submit a study closure form;
- I will gain IRB approval before altering the research study and/or consent forms;
- I will notify the IRB if there are any changes in my contact information.

💿 I agree

### APPENDIX C: INSTITUTIONAL REVIEW BOARD LETTER OF APPROVAL



INSTITUTIONAL REVIEW BOARD

Mail: P.O. Box 3999 Atlanta, Georgia 30302-3999 Phone: 404/413-3500 In Person: 3rd Floor 58 Edge wood FWA: 00000129

June 21, 2021

Principal Investigator: Christopher Ingalls

Key Personnel: Brandenberger, Kyle; Ingalls, Christopher, Jackson, Mekensie H; Otis, Jeffrey; Rawdon, Christopher L; Yang, Feng, PhD

Study Department: Kinesiology & Health

Study Title: The Effect of Eccentric Contraction-Induced Injury On Individual Quadriceps Muscles

Submission Type: Submission Response for Initial Review Submission Form

Review Type: Full Board Review

IRB Number: H21635

Reference Number: 365445

The above referenced study was reviewed and given pending approval under the Full board review process by the Georgia State University Institutional Review Board (IRB) on 06/17/2021. This approval became effective on June 18, 2021 after all pending issues were addressed and is valid through 06/16/2022 in accordance with 45 CFR 46.111. The IRB has reviewed and approved the research protocol and any informed consent forms, recruitment materials, and other research materials that are marked as approved in the application. The approval period is listed above. Research that has been approved by the IRB may be subject to further appropriate review and approval or disapproval by officials of the Institution.

It is the Principal Investigator's responsibility to ensure that the IRB's requirements as detailed in the Institutional Review Board Policies and Procedures For Faculty, Staff, and Student Researchers (available at gsu.edu/irb) are observed, and to ensure that relevant laws and regulations of any jurisdiction where the research takes place are observed in its conduct.

Federal regulations require researchers to follow specific procedures in a timely manner. For the protection of all concerned, the IRB calls your attention to the following obligations that you

have as Principal Investigator of this study.

- For any changes to the study, an Amendment Application must be submitted to the IRB. The Amendment Application must be reviewed and approved before any changes can take place.
- 2. Any unanticipated problems occurring as a result of participation in this study must be reported immediately to the IRB using the Unanticipated Problem Form.
- Principal investigators are responsible for ensuring that informed consent is properly documented in accordance with 45 CFR 46.116.
  - The Informed Consent Form (ICF) used must be the one reviewed and approved by the IRB with the approval dates stamped on each page.
  - •
- 4. For any research that is conducted beyond the approval period, a Continuing Review Form must be submitted at least 30 days prior to the expiration date. The Continuing Review Form must be approved by the IRB before the expiration date else automatic termination of this study will occur. If the study expires, all research activities associated with the study must cease and a new application must be approved before any work can continue.
- 5. When the study is completed, a Study Closure Form must be submitted to the IRB.

All of the above referenced forms are available online at <u>http://protocol.gsu.edu</u>. Please do not hesitate to contact the Office of Research Integrity (404-413-3500) if you have any questions or concerns.

Sincerely,

an Cale Kyuger

Ann Kruger, IRB Chair

### APPENDIX D: INFORMED CONSENT FOR PILOT STUDY

Georgia State University Informed Consent

Title: Pilot study of individual quadriceps muscle torque Principal Investigator: Dr. Christopher Ingalls Co-Investigator: Feng Yang; Kyle Brandenberger Student Principal Investigator: Christopher Rawdon

#### Introduction and Key Information

You are invited to take part in a research study. It is up to you to decide if you would like to take part in the study.

The purpose of this study is to investigate the accuracy and reliability of two different methods of measuring individual quadriceps muscle torque.

Your role in the study will be for two hours on two separate days that work with your schedule. You will be asked to do the following: Have three muscles of your left quadriceps muscle be electrically stimulated in order to measure torque produced by the individual muscles.

The risks of being in this study include: Direct electrical stimulation may result in brief discomfort of the thigh muscles.

This study not designed to benefit you. Overall, we hope to gain information about which of the two methods of torque measurement will produce the most accurate and reliable results of individual quadriceps muscle torque.

#### Purpose

The purpose of the study is to investigate the reliability and accuracy of the Biodex dynamometer and Vicon motion capturing cameras in measuring individual quadriceps muscle torque. You are invited to take part in this research study because you are a male between the ages of 18 and 35 have a low risk of heart disease, either do not exercise regularly or are recreationally active and have no history of significant knee injuries. A total of 10 people will be invited to take part in this study.

#### Procedures

If you decide to take part, you will report to the Georgia State Biomechanics Laboratory in the Sports Arena (Room G15). Each session will last between one and two hours. We will ask you to not exercise in the three days leading up to the study and the days in between your two sessions.

When you arrive you will be asked to complete a health history form. If you have fewer than 2 risk factors for heart disease and have no history of a traumatic leg injury, then you will be asked to continue with the study. After you complete your health history questionnaire, we will measure your height, weight, blood pressure and heart rate as well as take some measurements of your left leg. You will then warm-up on our Biodex dynamometer for 3 sets of 10 leg extensions on your left leg. The researchers will then strap your left thigh, waist and torso to the Biodex chair. Pairs of stimulating electrodes will be placed on the individual muscles of your left knee extensor muscles (i.e., quadriceps). To assess activation of antagonist muscles during quadriceps muscle activation, two electromyography (EMG)

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electrodes will be placed on your hamstring to measure the electrical activity of these muscles. Your hamstring muscles will not be stimulated at any point during the study. In order to prepare the skin for electrode placement, we will provide you with a razor in order to remove hair from the areas we will be placing the electrodes. The researchers will stimulate your individual quadriceps muscles at a low frequency (10-20Hz) and high frequency (80-100Hz) each lasting for a 0.4 second duration. Our Vicon motion capturing cameras will record the "kick" of your lower leg produced by the electrical activation of your individual quadriceps muscles. Three to five contractions will be recorded for each muscle for each frequency for a total of 30 contractions. If at any time you find that these stimulations are too uncomfortable, you should withdraw from the study. The researchers will then strap your shin to the lever of the Biodex dynamometer. The researchers will repeat the stimulations of the individual muscles at a low frequency (10-20Hz) and high frequency (80-100Hz) each lasting for a 0.4 second duration and measure torque via the Biodex dynamometer. You will then perform two maximal voluntary contractions at each of the following velocities: 0, 45, 90, 210 °/sec. There will be one minute of rest in between contractions. This will conclude the data collection for the first session. We will then schedule your second session within seven days following the first data collection day.

When you come in for the second session you will warm-up on our Biodex dynamometer for 3 sets of 10 leg extensions on your left leg. The researchers will then strap you to the Biodex chair. Pairs of stimulating electrodes will be placed on the individual muscles of your left knee extensor muscles and two electromyographs (EMGs) will be placed on two of your hamstring muscles. The researchers will stimulate your individual quadriceps muscles at a low frequency (10-20Hz) and high frequency (80-100Hz) each lasting for a 0.4 second duration. Our Vicon motion capturing cameras will record the "kick" movement of the lower leg produced by electrical activation of your individual quadriceps muscles. The researchers will repeat the stimulations of the individual muscles at a low frequency (10-20Hz) and high frequency (80-100Hz) each lasting for a 0.4 second duration. You will then perform two maximal voluntary contractions at each of the following velocities: 0, 45, 90, 210 °/sec. This will conclude your participation in the study.

#### **Future Research**

Researchers will remove information that may identify you and may use your data for future research. If we do this, we will not ask for any additional consent from you.

#### Risks

Direct electrical stimulation may result in discomfort of the thigh muscles. The stimulation of the muscles and nerves within your thigh could be perceived as uncomfortable or painful. If you find that these stimulations are too uncomfortable, you should withdraw from the study. To reduce risk during the experiment, trained personnel will supervise the testing. You may develop a mild rash or skin irritation where the electrodes were attached. This irritation usually goes away on its own without medical treatment. No injury is expected from this study, but if you experience any health-related problem besides temporary muscle discomfort, you may contact either Dr. Christopher Ingalls (cingalls@gsu.edu) or Christopher Rawdon (crawdon1@gsu.edu) as soon as possible. Georgia State University and the research team have not set aside funds to compensate for any injury.

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GSU IRB NUMBER: H20435 APPROVED IRB APPROVAL DATE: 02/26/2020

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#### **Benefits**

This study is not designed to benefit you personally. Overall, we hope to gain information about the validity and reliability of the Biodex dynamometer and Vicon motion capturing technology in measuring individual quadriceps muscle torque.

#### **Alternatives**

The alternative to taking part in this study is to not take part in the study

#### Voluntary Participation and Withdrawal

You do not have to be in this study. If you decide to be in the study and change your mind, you have the right to drop out at any time. You may skip questions or stop participating at any time. You may refuse to take part in the study or stop at any time. This will not cause you to lose any benefits to which you are otherwise entitled.

#### **Confidentiality**

We will keep your records private to the extent allowed by law. The following people and entities will have access to the information you provide:

• Drs. Christopher Ingalls, Feng Yang, Kyle Branderberger , Mr. Christopher Rawdon and Mr. Marcus Adan

- GSU Institutional Review Board
- Office for Human Research Protection (OHRP)

We will use a study code rather than your name on study records. The information you provide will be stored in a locked filing cabinet in the office of Dr. Ingalls, temporarily on a password protected lab computer and flash drive while the flash drive is transported to Dr. Ingalls office, and a password-protected computer in Dr. Ingalls office. A study code will be kept in a secure location separate from the study data in Dr. Ingalls' office; the study code will be destroyed after completion of the study. When we present or publish the results of this study, we will not use your name or other information that may identify you.

We will keep your personal information private. Your privacy will be kept to the extent allowed by law. The health information you give us will be used in this research study. We will remove all information that can identify you. We will share it with other people for this research study. If you decide you want to be in this study it means that you agree to let us use and share your personal health information for the reasons we have listed in this consent form.

While we are doing this research, the research team may use only the personal health information that you have given us. The people that will be able to look at your personal health information is: Dr. Christopher Ingalls, Dr. Feng Yang, Dr. Kyle Brandenberger, Mr. Chistopher Rawdon and Mr. Marcus Adan. They will look at it so they can work on this research study. We may also share your health information with the Georgia State University Institutional Review Board (IRB). Your personal health

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information may be shared by the people or places we have listed, but it will be shared in a way that does not fall under the protection of federal regulations that apply to the privacy of health information. This research may be shown to other researchers. This research may be published, but we will take steps to make sure that you cannot be identified.

If you sign this consent form you are letting us use your personal health information until the end of the study. You have the right to say that you do not want us to use your personal health information after we have collected it. If you decide you don't want us to use your information anymore you must write a letter asking us not to use your information. You may not be able to look at or get a copy of your health information that you gave us while we are doing the research; however you will be able to look at or get a copy at the end of the study.

#### **Contact Information**

Contact Dr. Christopher Ingalls at (404) 413-8377 and cingalls@gsu.edu or Christopher Rawdon at (678) 689-8009 and crawdon1@gsu.edu

- If you have questions about the study or your part in it
- If you have questions, concerns, or complaints about the study
   \*Contact the GSU Office of Human Research Protections at 404-413-3500 or irb@gsu.edu
- · If you have questions about your rights as a research participant
- If you have questions, concerns, or complaints about the research

The IRB at Georgia State University reviews all research that involves human participants. You can contact the IRB if you would like to speak to someone who is not involved directly with the study. You can contact the IRB for questions, concerns, problems, information, input, or questions about your rights as a research participant. Contact the IRB at 404-413-3500 or <u>irb@gsu.edu</u>.

#### **Consent**

We will give you a copy of this consent form to keep. If you are willing to volunteer for this research, please sign below.

Printed Name of Participant			
Signature of Participant		Date	
Principal Investigator or Researche	r Obtaining Consent	Date	
	4	GSU	IRB NUMBER: H20435

### APPENDIX E: INFORMED CONSENT FOR DIFFERENTIAL INJURY STUDY

Georgia State University Informed Consent

Title: The Effect of Eccentric Contraction-Induced Injury On Individual Quadriceps Muscles Principal Investigator: Dr. Christopher Ingalls Co-Investigators: Drs. Feng Yang, Kyle Brandenberger, and Jeff Otis Student Principal Investigator: Christopher Rawdon

#### Introduction and Key Information

You are invited to take part in a research study. It is up to you to decide if you would like to take part in the study.

The purpose of this study is to investigate the effects of downhill running on strength loss, soreness, and activation of the individual quadriceps muscles.

Your role in the study will be for approximately eight hours in total on three separate days that work with your schedule.

You will be asked to do the following:

- Consent to take part of this study.
- Walk and run for an extended duration.
- Take part in a balance assessment
- Take part in a maximum strength assessment.
- Have three muscles of your left thigh be electrically stimulated to measure force produced by the individual muscles. In total, there will be 50 stimulations across the three days with each stimulation lasting .4 seconds.
- Have the activation of you hamstring muscles recorded.
- Rate your level of soreness and pain.

The risks of being in this study include:

- Direct electrical stimulation may result in brief discomfort of the thigh muscles.
- If selected in the downhill running group, soreness and weakness of your leg muscles may persist for 5-7 days.

This study is not designed to benefit you. Overall, we hope to gain information about whether unaccustomed exercise will injure individual thigh muscles differently, which may then predispose a person to developing a secondary injury of tendons, ligaments, or bones.

#### Purpose

The primary goal of our studies is to measure the degree of strength loss across the different knee extensor muscles (vastus medialis, vastus lateralis, and rectus femoris) following unaccustomed exercise that causes injury. You are invited to take part in this research study because you are a male between the ages of 18 and 35, either do not exercise regularly or are recreationally active, do not require medical clearance for exercise and have no history of significant knee injuries. A total of 16 people will be invited to take part in this study.

Version Date: 6-17-21

#### **Procedures**

If you decide to take part, you will report to the Georgia State Biomechanics Laboratory in the Sports Arena (Room G15). There will be three different sessions in total. The first session will last approximately two hours, the second session will last approximately four hours and the third session will last approximately two hours. We will ask you to not exercise in the three days leading up to the study and in the days in between your sessions.

When you arrive for your first session, you will be asked to complete a health history form. If you do not have any signs or symptoms for heart disease and have no history of a traumatic leg injury, then you will be asked to continue with the study. After you complete your health history questionnaire, we will measure your height, weight, blood pressure and heart rate as well as take some measurements of your left leg. You will then complete a soreness assessment where first we will rate overall soreness of your non-dominant leg by stepping onto a box and then apply pressure using a myometer on the individual muscles of your quadriceps to assess individual muscle soreness. You will then take part in a balance assessment where you will maintain upright posture with eyes open, closed and eyes open on a foam pad while we record ground reaction force. Next, we will determine the appropriate range of treadmill walking and running velocities that will be used during assessments and either of the experimental protocols. The researchers will then strap your left thigh, waist, and torso to the Biodex chair and strap your shin to the lever of the Biodex dynamometer. You will then perform two maximal voluntary contractions at 20°, 45°, and 90° of knee flexion. There will be one minute of rest in between contractions. Pairs of stimulating electrodes will be placed on rectus femoris muscle of your left leg. To assess activation of antagonist muscles during quadriceps muscle activation, two electromyography (EMG) electrodes will be placed on your hamstring to measure the electrical activity of these muscles. Your hamstring muscles will not be stimulated at any point during the study. To prepare the skin for electrode placement, we will provide you with a razor in order to remove hair from the areas we will be placing the electrodes. In addition, we will first rub sandpaper on the skin of the application sites to help remove dead skin cells and then wipe with a rubbing alcohol pad. The researchers will stimulate your rectus femoris muscles at a low frequency (20Hz) for two repetitions and high frequency (80Hz) for two repetitions each lasting for a 0.4 second duration and measure torque via the Biodex dynamometer. If at any time during the study you find that these stimulations are too uncomfortable, you should withdraw from the study. This will conclude the data collection for the first session. You will be randomly placed into either the downhill run protocol (INJ) or control exercise protocol (CON). We will then schedule your second session five to seven days following the first data collection day.

When you come in for the second session you will be measured for baseline (Pre) muscle soreness and limb circumference. The researchers will then apply EMG electrodes to your leg muscles which will be secured by athletic wrap. You will then complete the postural assessment while EMG is recorded. Following this assessment, you will walk on the treadmill at the previously determined speed for five minutes. We will be recording the EMG of your leg muscles during the last ten seconds of the final three minutes. Then you will run on the treadmill at the previously determined speed for five minutes. We will be recording the EMG of your leg muscles during the last ten seconds of the final three minutes. Following the walking and running trials, you will then be assessed for maximal voluntary knee extensor strength on the Biodex. Pairs of stimulating electrodes will be placed on the individual muscles of your left knee extensor muscles (i.e., quadriceps). The researchers will stimulate your individual quadriceps muscles for three times at a low frequency (20Hz) and then twice at a high frequency (80Hz) each lasting for a 0.4 second duration. The individual torques will be recorded by the Biodex dynamometer. You will then perform either the 60-minute downhill running injury protocol or the 30-minute walking control exercise protocol as determined by your group selection. After a 5 to 10-minute break you will then be

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measured again for post-injury muscle soreness, limb circumference, balance, walking and running assessments. Post-injury strength assessments (i.e., MVC and electrical stimulation) will conclude your data collection for the second session.

You will then return to the lab in 48 hours for the last day of data collection. You will be measured for post-injury muscle soreness and limb circumference. EMG electrodes are reapplied before completing the posture assessment plus walking and running assessments. You will then be placed in the Biodex for the 48 hours post-injury strength assessments. This will conclude all necessary testing and data collection for the subject in this study.

#### Future Research

Researchers will remove information that may identify you and may use your data for future research. If we do this, we will not ask for any additional consent from you.

#### <u>Risks</u>

Direct electrical stimulation may result in discomfort of the thigh muscles. The stimulation of the muscles and nerves within your thigh could be perceived as uncomfortable or painful. If you find that these stimulations are too uncomfortable, you should withdraw from the study. To reduce risk during the experiment, trained personnel will supervise the testing. You may develop a mild rash or skin irritation where the electrodes will be attached. This irritation usually goes away on its own without medical treatment.

If selected for the downhill running group, the protocol is designed to cause temporary soreness and weakness of your leg muscles and should be gone within 5-7 days. We ask you not to exercise outside of the study. We also ask you not to consume over-the-counter pain medications as these may influence your injury and confound study results. There is also the possibility that acute myocardial infarction, cardiac arrest, cardiac arrhythmia, stroke, and musculoskeletal injury could occur during exercise while participating this study. However, the risks of these events is extremely small in an apparently healthy population that does not have elevated risk of heart disease. These cardiovascular risks are also small considering that you will be walking at submaximal intensities for a brief period (30 minutes) if you are in the control group. The cardiovascular risk is also small in the downhill running group because running downhill is less stressful metabolically than level running. There is also a risk of falling off the treadmill while walking or running which may cause injury. If you experience any health-related problem besides muscle soreness, you may contact either Dr. Christopher Ingalls (cingalls@gsu.edu) or Christopher Rawdon (crawdon1@gsu.edu) as soon as possible. Georgia State University and the research team have not set aside funds to compensate for any injury.

#### **Benefits**

This study is not designed to benefit you personally. Overall, we hope to gain information about the effects of exercise-induced injury on the individual quadriceps muscles. We also hope to learn how these changes may impact neuromuscular control while standing upright and during locomotion.

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#### **Compensation**

You will receive one \$25 gift card for the first session and one \$25 gift card for the second session. You will receive a \$50 gift card for the third session. If you are withdrawn from the study or decide to stop participating, you will still receive the gift card for the visit that day.

#### **Alternatives**

The alternative to taking part in this study is to not take part in the study

#### Voluntary Participation and Withdrawal

You do not have to be in this study. If you decide to be in the study and change your mind, you have the right to drop out at any time. You may refuse to take part in the study and/or stop participating at any time.

#### **Confidentiality**

We will keep your records private to the extent allowed by law. The following people and entities will have access to the information you provide:

- Drs. Christopher Ingalls, Feng Yang, Kyle Branderberger, Jeff Otis, Mr. Christopher Rawdon, and Ms. Mekensie Jackson
- GSU Institutional Review Board
- Office for Human Research Protection (OHRP)

We will use a study code rather than your name on study records. The information you provide will be stored in a locked filing cabinet in the office of Dr. Ingalls, temporarily on a password protected lab computer and flash drive while the flash drive is transported to Dr. Ingalls office, and a password-protected computer in Dr. Ingalls office and as encrypted files on a GSU sponsored cloud server. A study code will be kept in a secure location separate from the study data in Dr. Ingalls' office; the study code will be destroyed after completion of the study. When we present or publish the results of this study, we will not use your name or other information that may identify you.

We will keep your personal information private. Your privacy will be kept to the extent allowed by law. The health information you give us will be used in this research study. We will remove all information that can identify you. We will share it with other people for this research study. If you decide you want to be in this study it means that you agree to let us use and share your personal health information for the reasons we have listed in this consent form.

While we are doing this research, the research team may use only the personal health information that you have given us. The people that will be able to look at your personal health information are: Dr. Christopher Ingalls, Dr. Feng Yang, Dr. Kyle Brandenberger, Dr. Jeff Otis, Mr. Christopher Rawdon, and Ms. Mekensie Jackson. They will look at it so they can work on this research study. We may also share your health information with the Georgia State University Institutional Review Board (IRB). Your personal health information may be shared by the people or places we have listed, but it will be shared in a way that does not fall under the protection of federal regulations that apply to the privacy of health

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information. This research may be shown to other researchers. This research may be published, but we will take steps to make sure that you cannot be identified.

If you sign this consent form you are letting us use your personal health information until the end of the study. You have the right to say that you do not want us to use your personal health information after we have collected it. If you decide you do not want us to use your information anymore you must write a letter asking us not to use your information. You may not be able to look at or get a copy of your health information that you gave us while we are doing the research; however you will be able to look at or get a copy at the end of the study.

#### **Contact Information**

Contact Dr. Christopher Ingalls at (404) 413-8377 and cingalls@gsu.edu or Christopher Rawdon at (678) 689-8009 and crawdon1@gsu.edu:

- If you have questions about the study or your part in it
- If you have questions, concerns, or complaints about the study
- \*Contact the GSU Office of Human Research Protections at 404-413-3500 or irb@gsu.edu
- If you have questions about your rights as a research participant
- If you have questions, concerns, or complaints about the research

The IRB at Georgia State University reviews all research that involves human participants. You can contact the IRB if you would like to speak to someone who is not involved directly with the study. You can contact the IRB for questions, concerns, problems, information, input, or questions about your rights as a research participant. Contact the IRB at 404-413-3500 or irb@gsu.edu.

#### <u>Consent</u>

We will give you a copy of this consent form to keep. If you are willing to volunteer for this research, please sign below.

Printed Name of Participant

Signature of Participant

Date

Principal Investigator or Researcher Obtaining Consent

Date

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### APPENDIX F: HEALTH HISTORY QUESTIONNAIRE

### Health History Questionnaire Department of Kinesiology and Health Georgia State University

### **Health History**

All information given is personal and confidential. The information will enable us to better understand you and your health and fitness habits.

Name	e		<u>.</u>	Date	2
Addr	ess			Home Phone	
City/	State		Zip Code		
E-ma	uil				
Occu	pation			Other Phone	
Birth	Date	Gender	Height	Weight	Ethnicity
I. **** Have (please yes	Sig you even e circle yes no no no	gns and Symptoms ************************************	owing: ess or numbness i st or with mild ex	********************** n the chest, neck, ja certion.	**************************************
yes yes		<ul><li>5. Ankle swelling.</li><li>6. Rapid pulse or heart rate</li></ul>			
yes yes		<ol> <li>7. Intermittent cramping.</li> <li>8. Known heart murmur.</li> </ol>			
yes If you	u answere How o Have y	9. Unusual shortness of bre ed yes to any of the above— ften do you experience the s you ever discussed the symp	symptom?	r?	
	Explai	n the symptom in more deta	il:		

*******	********	*****	******
	lajor Risk Factors		
******	* * * * * * * * * * * * * * * * * * * *	*****************	******
yes no	1. Do you have a body mass index	$x \ge 30$ or a waist girth >10	00 cm?
yes no	2. Have you had a fasting glucose least 2 separate occasions.	of $\geq$ 110 mg/dl confirme	d by measurements on at
yes no	3. Has your father or brother expension your mother or sister expension of the second		
yes no	4. Do you currently smoke or quit	within the past 6 months	?
yes no	5. Has your doctor ever told you the	hat you have high blood J	pressure?
yes no	6. Do you have high cholesterol? Total cholesterol:	HDL:	Date tested:
yes no 7. Do you have a sedentary lifestyle? (sitting most of the day in your job with no regular physical activity)			
III. M ******	**************************************	****	
Have you eve	er had any of the following? Circle	all that apply:	
heart attack	angioplasty	heart surgery	coronary artery disease
angina	hypertension	heart murmur	heart clicks
asthma	emphysema	bronchitis	stroke
anemia	phlebitis	emboli	cancer
osteoporosis	emotional disorders	eating disorders	
Any special j	problems not listed above:		
If any of the	above are circled, please give details	and explain:	

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******	************	*****
	eneral	
********	***************************************	*****
yes no	1. Are you pregnant?	
yes no	2. Do you have arthritis or any bone or joint problem? If yes, please explain:	
yes no	3. Do you currently exercise? If yes, how long have you been exercising? What do you do and how often?	
yes no	4. Are you taking any medication, vitamins or supplements? Name them and their dosage (list both prescribed and over-the-counter n Drug name and dosage / purpose of drug / prescribed or over-the- dosage / purpose of drug / purpose of drug / prescribed or over-the- dosage / purpose of drug / purpose of dru	nedications)
	5. Has your physician ever told you that you have reduced kidney functi ever been diagnosed or treated for kidney stones? certifies that all of the above is true, to the best of my knowledge.	
Signature:	Date:	
Signature.	Datt	
STAFF USE	ONLY	
Stratification	(circle one): Low Risk Moderate Risk	High Risk
Resting blood	Pressure:Resting heart rate:	
yes no	Do meds affect BP or HR?	
Date:	Initials:	

### APPENDIX G: DATA COLLECTION SHEET

Subject ID: \_\_\_\_\_

Age:

Height:

Weight:

<b>Familiarization</b>	Day 1		Day 2
Resting HR:	Resting HI	R:	Resting HR:
Resting BP:	Resting BP	:	Resting BP:
Anthropomorphic Measure	ements	Left Leg	Right Leg
		<u> </u>	0 0
Q-Angle:			
Leg Length (cm):			
Shank Length (cm):			
Foot Length (cm):			
Ankle Height (cm):			
Feet Distance at Shoulder Width (cm):			

Fam <sup>L</sup>	R Pre L	R Post L	<sup>R</sup> 48HL	R
Thigh Circ:	Thigh Circ:	Thigh Circ:	Thigh Circ:	
Knee ROM:	Knee ROM:	Knee ROM:	Knee ROM:	

# Forces for Individual Soreness

Fam	Pre	Post	48H
LVM			
L RF L VL			
R VM			
R RF			
R VL			
Biodex Chair Position	ing		
Chair Height:	Back Positio	n:	Chair Position:
U			
Dynamometer Positio	on: Leg Atta	chmont	
Dynamometer i ositio	II. Leg Atta	ciiiieiit.	
Walking & Jogging Spe	<u>eeds</u>		
10m Trial 1 (s):	Average (s	5):	m/s:
10m Trial 2 (s):		- ) -	1m/s = 2.237
10m Trial 3 (s):			

# Walking Speed (mph):

**Jogging Speed** (mph @ ~70% age-predicted HRM):

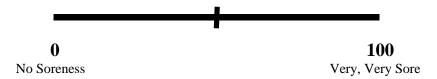
# Pain Scores Individual Stimulation (0-100)

Fam

VM low freq:	RF low freq:	VL low freq:
VM high freq:	RF high freq:	VL high freq:
Pre		
VM low freq:	RF low freq:	VL low freq:
VM high freq:	RF high freq:	VL high freq:
Post		
VM low freq:	RF low freq:	VL low freq:
VM high freq:	RF high freq:	VL high freq:
48H		
VM low freq:	RF low freq:	VL low freq:
VM high freq:	RF high freq:	VL high freq:

# Rating of Exercise Induced – LEFT Muscle Soreness Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



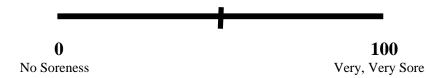
# Rating of Exercise Induced – RIGHT Muscle Soreness Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



# Rating of Exercise Induced – LEFT Knee Pain Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



# Rating of Exercise Induced – RIGHT Knee Pain Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



APPENDIX J: INDIVIDUAL MUSCLE SORENESS SCALE

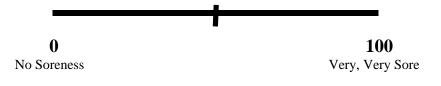
# Rating of Exercise Induced–Muscle Soreness (Left VM) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



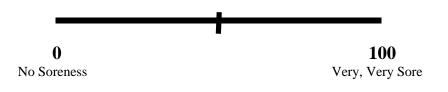
# Rating of Exercise Induced–Muscle Soreness (Left RF) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



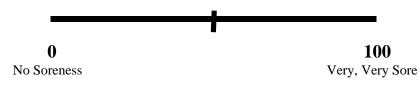
# Rating of Exercise Induced–Muscle Soreness (Left VL) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



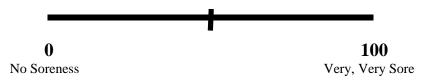
# Rating of Exercise Induced–Muscle Soreness (Right VM) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



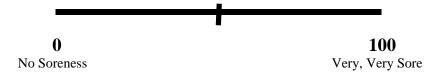
# Rating of Exercise Induced–Muscle Soreness (Right RF) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



Rating of Exercise Induced–Muscle Soreness (Right VL) Visual Analogue Scale: 100 mm

Subject #	
Date	
Time	
Trial/Step # (1-4)	
Soreness Score (mm)	



### APPENDIX K: SUBJECT RECRUITMENT FLYER



# RESEARCH PARTICIPANTS NEEDED

### DO YOU WANT TO BE A PART OF A NOVEL STUDY?

What are the effects of strenuous exercise on our neuromuscular system?



What can you expect during the study?

- Have anthropomorphic measurements taken
- Walk and run on a treadmill
- Undergo balance tests
- Undergo strength tests
- Rate your levels of muscle & joint soreness and pain

### You will be expected to come to the GSU Biomechanics lab on 3 different occasions to perform these tests

### Who qualifies?

- Males
- Ages 18 35
- Does not currently exercise or does not exercise often (< 3x per week)</li>
- Free of injury or illness

You will be compensated \$100 in Visa gift cards for completing the study

For more information or to sign up please contact Chris Rawdon (crawdon1@gsu.edu) or Dr. Christopher Ingalls (cingalls@gsu.edu)

		VM			RF			VL	
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	12.6	14.1	16.0	11.1	12.7	15.4	13.6	14.5	15.6
2	20.2	17.8	-	17.2	13.2	-	22.8	16.8	-
3	21.8	18.5	20.5	14.4	13.4	13.2	21.2	17.8	21.1
4	23.4	20.7	22.6	35.9	28.9	28.4	24.0	19.9	23.6
5	20.3	18.1	21.2	19.4	19.7	20.9	18.7	16.4	18.5
6	18.1	11.8	16.1	19.0	15.3	17.7	14.2	8.6	11.7
7	-	-	-	-	-	-	-	-	-
8	7.50	5.30	7.60	7.20	5.60	6.30	12.90	13.30	14.30
9	9.7	8.0	11.0	23.6	19.7	29.3	18.4	11.8	19.3
10	9.7	4.4	7.3	24.9	14.2	13.9	21.2	9.9	12.1
11	19.0	17.7	22.4	23.0	22.4	23.6	20.9	17.9	20.6
12	6.00	5.70	9.10	15.30	11.10	12.00	14.80	8.60	11.30
13	13.00	9.20	12.40	14.70	12.80	17.40	19.90	13.30	18.60
14	12.9	11.2	-	42.9	38.0	-	30.5	28.6	-
15	10.1	11.20	11.0	16.9	17.3	14.8	18.9	19.6	19.6
Mean	14.6	12.4	14.8	20.4	17.5	17.7	19.4	15.5	17.2
SD	1.5	1.4	1.6	2.4	2.1	1.9	1.2	1.4	1.1

APPENDIX L: 20 HZ STIMULATED TORQUE (N·M)

		VM			RF			VL	
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	33.0	36.8	36.6	29.8	32.3	32.3	38.9	37.3	38.2
2	47.0	45.3	-	45.7	42.0	-	54.2	46.9	-
3	48.8	48.0	41.7	26.4	26.5	23.9	41.1	39.4	39.0
4	41.2	39.8	40.0	83.7	81.0	77.0	45.2	43.8	40.4
5	35.5	34.0	35.0	45.0	49.2	46.4	34.2	42.2	37.1
6	35.0	29.6	33.4	38.4	36.6	37.9	23.5	21.4	21.0
7	-	-	-	-	-	-	-	-	-
8	35.5	34.8	38.7	55.1	42.7	49.2	44.9	34.2	37.1
9	35.0	29.6	33.4	38.4	36.6	37.9	23.5	21.4	21.0
10	30.5	28.3	28.8	76.4	72.4	73.1	48.2	44.6	52.2
11	17.3	19.5	18.0	21.1	20.1	19.6	31.1	31.0	31.9
12	14.7	13.2	13.4	34.2	31.3	27.1	23.5	21.6	23.3
13	23.5	18.6	20.6	25.5	23.7	25.6	32.8	26.6	28.5
14	19.6	20.0	-	78.0	70.7	-	54.4	50.9	-
15	27.7	29.5	29.1	42.5	44.6	38.6	44.2	43.1	47.0
Mean	31.7	30.5	30.7	45.7	43.6	40.7	38.6	36.0	34.7
SD	2.7	2.6	2.5	5.3	4.9	5.1	2.8	2.6	2.7

### APPENDIX M: 80 HZ STIMULATED TORQUE $(N\!\cdot\!M)$

	20°				45°			90°		
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H	
1	105.4	87.7	100.3	177.7	182.4	178.6	246.2	234.1	243.0	
2	106.5	63.0	-	201.2	119.4	-	274.2	133.8	-	
3	147.4	93.6	112.4	203.2	156.1	157.0	268.2	218.2	215.4	
4	142.7	129.6	135.4	253.4	224.9	260.2	247.6	230.9	281.4	
5	112.5	126.1	113.8	191.5	207.6	224.6	196.5	194.7	214.4	
6	83.8	70.7	60.8	144.0	99.4	99.2	185.2	128.0	129.5	
7	135.1	137.4	142.4	177.1	164.2	193.3	237.5	222.2	223.9	
8	97.1	94.0	110.6	167.2	168.6	184.1	233.9	230.2	226.7	
9	124.1	97.1	124.6	206.3	176.4	197.5	240.5	193.5	196.4	
10	65.1	59.1	49.2	135.4	130.6	108.7	203.7	155.1	150.3	
11	72.4	79.1	83.3	117.6	123.0	126.0	132.6	164.8	133.8	
12	93.8	64.2	62.3	117.5	93.1	89.4	148.7	115.6	110.4	
13	75.6	70.7	83.3	127.9	97.1	135.7	147.6	106.5	156.5	
14	120.7	130.1	-	222.7	236.0	-	368.0	415.9	-	
15	78.8	85.8	92.3	165.6	179.3	193.8	227.7	214.1	232.6	
Mean	104.1	92.5	97.7	173.9	157.2	165.2	223.9	197.2	193.4	
SD	6.5	6.6	7.8	10.0	11.4	13.7	14.8	18.8	13.9	

APPENDIX N: MAXIMAL VOLUNTARY CONTRACTION TORQUE  $(N{\cdot}M)$ 

				VM					
	20°			45°			90°		
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	46.66	46.54	81.47	44.44	49.40	68.69	61.84	62.19	78.24
2	106.75	59.23	-	109.32	50.01	-	155.96	67.64	-
3	95.58	91.85	121.28	65.44	83.48	76.79	92.00	107.39	125.96
4	174.68	173.35	148.44	182.44	211.84	181.06	256.60	251.61	205.25
5	101.09	109.16	86.41	103.75	116.35	111.33	86.43	99.82	85.80
6	58.93	58.37	45.80	70.67	45.97	52.47	82.53	69.30	74.73
7	134.98	104.06	149.36	83.71	83.85	95.29	182.75	133.66	196.64
8	168.81	114.48	120.00	186.71	164.33	139.68	258.98	209.92	171.40
9	190.07	160.60	162.37	165.00	173.43	148.77	377.93	257.17	270.23
10	265.23	355.90	292.09	285.00	465.65	301.64	392.49	387.01	256.06
11	42.65	51.26	53.40	34.25	43.31	45.91	65.73	83.95	74.37
12	48.98	53.63	45.44	29.54	38.81	29.94	42.7	41.89	43.97
13	110.33	103.52	119.77	116.39	97.08	145.57	172.14	72.04	152.09
14	76.66	91.59	-	104.04	103.71	-	105.74	112.67	-
15	85.91	83.42	110.23	109.92	100.02	130.72	113.39	80.50	89.66
Mean	113.82	110.46	118.16	112.71	121.82	117.53	163.15	135.78	140.34
SD	15.69	19.37	17.41	17.06	27.03	19.15	27.86	24.27	19.81

APPENDIX O: KNEE EXTENSOR ACTIVATION	(RMS) DURING MVC
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				RF					
	20			45			90		
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	54.41	54.58	59.96	58.17	68.37	56.77	70.09	70.57	70.27
2	35.95	17.20	-	37.57	23.49	-	60.41	35.54	-
3	49.16	39.43	45.13	29.92	33.90	25.89	49.37	37.16	34.22
4	122.40	105.79	137.06	104.96	101.48	137.11	100.62	60.46	92.74
5	120.06	107.50	90.06	103.23	107.19	92.87	107.19	94.90	76.07
6	26.43	34.53	20.80	31.43	30.33	21.30	46.70	48.97	37.97
7	61.01	65.32	59.90	38.65	28.94	37.02	65.41	58.35	78.30
8	47.79	36.03	36.17	51.09	48.41	41.87	82.66	101.15	100.07
9	86.00	66.97	70.30	85.63	80.67	75.47	178.93	134.77	142.23
10	125.28	113.15	199.72	144.43	170.15	183.26	190.50	219.61	172.66
11	22.66	24.30	37.32	14.86	23.00	27.42	22.88	35.92	32.30
12	34.38	48.64	30.82	24.91	29.82	18.96	39.66	33.47	26.37

13	38.1	47.94	58.94	42.18	44.17	63.94	66.07	55.68	83.72
14	67.73	71.72	-	106.86	97.12	-	113.16	112.51	-
15	60.18	70.97	76.34	78.01	83.56	99.22	93.72	126.01	141.11
Mean	63.44	60.27	70.96	63.46	64.71	67.78	85.82	81.67	83.69
SD	8.66	7.47	13.07	9.51	10.48	13.21	11.88	12.77	12.34

				VL					
		20			45			90	
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	46.40	45.24	43.15	47.18	51.45	42.43	52.58	51.16	47.59
2	37.07	27.42	-	38.22	21.92	-	49.25	32.54	-
3	54.29	58.33	59.79	32.50	50.64	29.75	69.01	54.67	46.03
4	69.58	106.06	79.54	61.07	89.20	81.77	77.36	81.56	67.06
5	96.33	95.23	95.99	91.75	92.27	100.40	87.98	85.61	92.49
6	25.43	24.93	21.53	25.37	21.13	19.37	23.77	22.20	23.23
7	66.96	72.73	69.01	41.17	36.82	45.44	62.78	55.55	64.53
8	75.10	68.96	55.38	81.44	84.20	63.78	97.76	95.70	91.39
9	138.93	135.57	98.80	105.97	129.27	87.43	115.17	105.50	101.03
10	139.33	222.48	162.46	134.24	258.51	172.61	135.07	148.35	129.94
11	17.48	24.45	22.52	15.32	20.71	20.18	20.53	26.65	23.88
12	59.14	47.14	34.38	42.03	37.04	26.98	59.64	38.91	37.38
13	30.47	39.52	37.8	33.69	34.69	42.37	57.8	42.88	64.19
14	127.52	109.86	-	176.88	147.43	-	182.79	179.78	-
15	35.31	110.42	48.25	40.70	85.67	49.07	57.39	100.51	70.99
Mean	67.96	79.22	63.74	64.50	77.40	60.12	76.59	74.77	66.13
SD	10.12	13.26	10.34	11.31	15.89	11.34	10.60	11.38	8.37

				171	Л				
		20		VN				00	
<u> </u>		<u>20</u>	4011		45 D	4011		<u>90</u>	4011
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	109.06	112.97	111.22	104.23	109.68	92.01	102.63	108.14	85.33
2	91.37	92.53	-	84.17	87.85	-	81.42	79.31	-
3	114.93	107.84	104.88	92.93	89.64	98.36	99.04	96.49	86.58
4	97.03	91.95	110.90	80.17	77.63	94.35	77.77	75.15	87.07
5	93.64	99.85	102.00	91.89	92.18	99.90	105.37	107.48	116.14
6	112.37	107.47	101.77	101.77	98.87	91.93	88.10	98.97	85.50
7	131.34	145.88	99.52	106.48	98.40	89.55	123.23	138.03	88.11
8	137.46	135.12	128.81	131.23	137.09	130.36	140.61	126.88	128.16
9	83.36	87.14	89.33	89.47	84.88	86.22	82.94	83.86	80.61
10	88.20	103.67	98.13	79.30	98.23	83.57	71.47	103.23	78.13
11	97.46	97.46	121.84	91.41	96.67	116.08	99.45	99.08	113.15
12	134.37	145.65	121.76	124.07	137.16	127.96	108.01	123.26	123.44
13	99.33	112	106.13	93.34	108.88	96.06	90.2	137.35	89.98
14	91.37	92.53	-	84.17	87.85	-	81.42	79.31	-
15	140.05	146.34	137.15	133.84	134.86	131.80	115.95	103.37	106.04
Mean	108.09	111.89	110.26	99.23	102.66	102.93	97.84	103.99	97.56
SD	4.84	5.27	3.63	4.43	4.83	4.62	4.71	5.06	4.62

APPENDIX P: KNEE EXTENSOR ACTIVATION (MF) DURING MV
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				RF	7					
		20			45			90		
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H	
1	125.73	120.01	118.07	114.77	112.59	107.08	95.63	93.80	84.84	
2	126.67	120.69	-	119.46	120.78	-	95.41	93.44	-	
3	123.44	122.87	116.17	110.71	106.75	105.18	97.35	100.17	97.56	
4	114.14	110.39	118.18	102.97	97.96	108.69	93.85	96.38	94.31	
5	86.00	85.12	93.59	81.21	76.59	91.69	67.76	69.18	82.57	
6	126.07	113.60	124.77	113.07	106.40	108.23	81.90	82.40	81.93	
7	121.65	119.45	111.92	116.68	116.96	107.51	97.37	105.41	88.39	
8	123.15	115.94	111.48	116.47	105.29	99.86	85.71	82.49	77.50	
9	86.25	84.11	82.64	88.84	79.81	83.64	66.49	65.18	63.86	
10	103.80	106.20	109.23	93.00	103.33	103.43	69.43	77.30	73.40	
11	158.24	125.51	141.94	158.23	122.11	153.44	122.52	99.67	152.50	
12	187.36	106.95	116.56	173.12	98.78	108.38	132.46	99.69	102.3	

13	132.83	126.2	133.84	123.07	118.56	132.15	117.2	98.2	114.33
14	126.67	120.69	-	119.46	120.78	-	95.41	93.44	-
15	101.96	98.71	94.70	95.74	82.81	88.29	79.60	75.19	74.58
Mean	122.93	111.76	113.31	115.12	104.63	107.51	93.21	88.80	91.39
SD	6.37	3.35	4.31	6.04	3.77	4.86	4.86	3.12	6.06

				VL					
		20			45			90	
Subject #	Pre	Post	48H	Pre	Post	48H	Pre	Post	48H
1	174.38	176.29	184.26	162.72	166.70	171.41	132.23	133.90	134.48
2	188.75	182.20	-	181.00	174.66	-	150.41	141.53	-
3	198.63	185.16	195.33	165.20	149.02	181.10	119.04	124.48	133.25
4	166.62	151.32	171.40	160.99	153.02	166.75	121.46	124.24	124.81
5	124.69	120.15	123.88	110.57	97.77	105.80	83.95	83.30	78.56
6	197.90	171.87	186.93	186.67	158.47	177.50	151.70	130.93	144.40
7	179.22	176.20	176.39	163.61	165.40	172.85	146.16	154.33	146.47
8	217.38	188.33	171.57	205.50	189.35	170.56	177.62	154.72	156.26
9	154.41	146.44	132.38	139.81	138.56	125.75	109.29	107.38	90.47
10	158.80	146.77	164.43	140.47	130.87	166.30	128.20	127.07	138.13
11	197.59	171.81	173.27	186.26	184.51	191.29	166.12	151.49	153.18
12	112.19	173.93	182.94	110.62	154.63	159.46	104.71	139.45	116.2
13	188	176.31	185.79	170.97	154.21	167.64	140.13	129.89	142.34
14	188.75	182.2	-	181	174.66	-	150.41	141.53	-
15	168.75	101.11	165.06	153.09	82.81	150.12	116.56	106.02	69.75
Mean	174.40	163.34	170.28	161.23	151.64	162.04	133.20	130.02	125.25
SD	7.06	6.33	5.55	6.74	7.39	6.17	6.20	4.89	7.58

		•••	ALKINU			
			VM			
		Non-Dominant Leg		]	Dominant Leg	-
Subject #	Pre	Post	48H	Pre	Post	48H
1	5.24	5.77	5.14	4.87	6.0	4.62
2	9.42	13.24	-	10.16	14.6	-
3	6.34	12.40	4.23	6.23	11.1	6.66
4	4.45	3.04	6.44	3.34	2.3	5.39
5	5.38	4.49	3.74	5.96	4.7	4.01
6	6.59	3.89	6.05	6.04	6.2	3.32
7	-	-	-	-	-	-
8	8.85	8.34	8.56	3.39	3.0	3.65
9	4.07	4.18	6.38	11.40	11.5	12.43
10	12.07	21.39	15.57	16.77	19.4	25.13
11	5.43	7.86	8.81	7.09	7.1	8.02
12	6.95	8.65	8.68	7.59	3.42	8.50
13	10.30	9.15	10.25	8.30	9.01	7.73
14	7.58	7.83	-	9.15	8.19	-
15	14.68	12.34	8.16	8.49	7.47	5.16
Mean	7.67	8.76	7.67	7.77	8.14	7.89
SD	0.79	1.26	0.88	0.90	1.22	1.66

APPENDIX Q: KNEE EXTENSOR VOLUNTARY ACTIVATION (RMS) DURING
WALKING

			RF			
	1	Non-Dominant Leg			Dominant Le	g
Subject #	Pre	Post	48H	Pre	Post	48H
1	5.39	5.95	4.81	2.90	3.0	2.99
2	3.03	5.22	-	2.99	3.3	-
3	2.00	6.61	2.63	1.80	3.0	2.66
4	3.56	3.29	2.98	2.15	2.3	1.81
5	3.87	3.43	3.04	3.12	3.1	2.32
6	3.10	2.35	3.78	3.61	2.3	3.44
7	-	-	-	-	-	-
8	2.85	2.94	3.03	2.87	2.2	2.26
9	4.75	5.22	5.41	2.74	2.7	2.20
10	6.99	14.94	14.16	6.57	17.2	10.34
11	3.97	4.45	3.42	3.25	2.8	2.62
12	4.57	4.16	4.87	4.57	2.23	4.83
13	4.12	4.23	4.10	5.16	4.84	3.54

14	5.05	5.09	-	3.24	3.44	-
15	5.05	3.59	3.48	4.75	2.62	2.66
Mean	4.16	5.11	4.64	3.55	3.93	3.47
SD	0.33	0.79	0.86	0.33	1.00	0.64

			VL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	7.40	8.28	7.66	5.80	6.9	8.87
2	7.81	10.00	-	9.64	15.1	-
3	5.31	13.70	7.09	6.54	10.8	5.45
4	6.03	4.62	5.81	3.53	5.3	-
5	7.13	5.85	5.53	9.32	9.1	5.81
6	4.28	2.34	4.59	7.89	4.3	-
7	-	-	-	-	-	-
8	6.74	6.11	6.91	7.60	7.8	7.81
9	10.26	11.75	12.04	12.33	11.8	10.95
10	8.98	15.82	11.92	14.12	33.7	17.59
11	5.98	6.66	7.61	-	-	8.66
12	7.25	8.57	7.49	8.32	5.87	7.99
13	7.80	7.49	8.96	7.60	7.78	4.60
14	12.14	11.14	-	8.07	7.57	-
15	9.02	15.04	8.66	10.60	9.90	9.72
Mean	7.58	9.10	7.86	8.57	10.46	8.75
SD	0.53	1.03	0.63	0.73	2.02	1.11

			BF			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	8.34	9.32	9.35	4.30	3.7	4.72
2	9.51	8.49	-	4.03	4.0	-
3	7.81	8.88	7.71	2.29	2.7	2.49
4	7.51	7.50	7.64	3.15	2.2	1.80
5	10.64	17.33	6.67	4.71	3.4	2.54
6	10.15	8.31	10.49	-	2.2	4.23
7	-	-	-	-	-	-
8	9.51	9.13	9.62	3.26	3.3	3.90

9	13.57	12.55	13.43	5.22	4.9	4.31
10	12.00	7.36	10.95	8.09	16.3	11.78
11	3.14	-	7.20	3.88	4.1	3.35
12	12.29	10.91	13.31	11.63	12.57	9.27
13	9.63	11.34	11.95	9.66	9.48	9.25
14	9.49	9.04	-	9.55	9.32	-
15	11.12	11.20	10.60	11.16	10.39	10.33
Mean	9.62	10.10	9.91	6.23	6.33	5.66
SD	0.65	0.71	0.64	0.88	1.15	0.96

			TA			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	18.29	17.43	19.65	21.98	24.1	24.79
2	24.06	28.17	-	16.19	25.6	-
3	18.39	41.12	23.52	27.31	19.5	33.13
4	15.60	18.99	17.01	18.25	19.1	14.65
5	23.04	25.33	20.90	15.29	16.1	16.09
6	16.25	13.83	12.93	14.40	14.8	12.85
7	30.79	30.57	25.22	45.94	40.4	43.52
8	36.08	33.89	41.27	33.30	40.0	27.16
9	24.56	28.51	23.77	27.74	34.5	28.63
10	20.24	33.46	28.08	24.18	33.9	26.97
11	23.01	19.44	18.48	21.00	19.9	20.15
12	23.87	27.22	22.69	20.06	25.83	21.02
13	20.06	25.83	21.02	23.87	27.22	22.69
14	39.08	37.16	-	33.65	29.27	-
15	19.17	17.74	16.85	15.99	19.68	21.20
Mean	23.50	26.58	22.41	23.94	25.99	24.07
SD	1.72	1.98	1.85	2.15	2.05	2.19

			SOL			
		Non-Dominant Leg			Dominant Leg	5
Subject #	Pre	Post	48H	Pre	Post	48H
1	17.54	16.65	17.05	15.04	16.8	18.93
2	22.01	26.45	-	15.46	14.0	-
3	12.64	13.56	10.58	14.93	11.8	12.19

4	13.81	14.44	13.69	15.64	16.4	14.78
5	14.07	12.71	13.34	17.94	15.5	14.97
6	11.68	10.58	13.10	13.98	12.0	14.10
7	17.81	17.98	19.92	22.04	21.2	24.03
8	20.97	23.32	21.02	25.38	24.0	24.31
9	29.58	28.21	27.86	18.06	17.2	18.10
10	20.60	24.73	21.92	25.87	25.8	27.93
11	12.68	12.83	11.58	14.37	13.3	14.76
12	18.52	18.43	20.94	13.20	12.82	13.19
13	30.07	25.07	23.21	25.70	23.77	23.10
14	32.22	29.97	-	22.77	24.28	-
15	12.21	17.86	17.53	26.38	20.09	20.24
Mean	19.09	19.52	17.83	19.12	17.93	18.51
SD	1.71	1.57	1.38	1.25	1.22	1.34

			VM			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	104.17	107.59	86.39	107.36	108.3	99.45
2	85.08	77.63	-	90.62	91.1	-
3	61.24	82.30	104.84	115.48	115.8	128.39
4	93.58	84.60	129.25	122.46	100.2	177.29
5	128.16	108.77	112.33	121.45	91.9	104.76
6	96.17	86.52	96.03	107.49	94.8	109.82
7	-	-	-	-	-	-
8	114.51	123.33	115.75	143.37	132.7	159.01
9	91.20	94.25	123.84	109.18	96.7	97.90
10	99.77	96.87	90.71	104.57	122.3	149.39
11	92.10	95.93	93.98	84.52	86.7	93.93
12	147.18	112.53	113.86	108.86	169.26	95.51
13	99.29	103.92	103.03	122.53	115.51	125.20
14	125.02	126.03	-	129.67	127.70	-
15	104.27	148.25	131.13	120.22	118.26	133.57
Mean	102.98	103.47	108.43	113.41	112.23	122.85
SD	5.39	5.04	4.16	3.90	5.66	7.63

APPENDIX R: KNEE EXTENSOR VOLUNTARY ACTIVATION (M	(MF) DURING WALKING
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			RF			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	106.51	104.99	103.91	135.68	127.2	126.90
2	116.86	110.30	-	110.87	118.7	-
3	125.99	100.72	122.28	141.23	108.7	134.58
4	131.92	103.42	113.91	131.23	120.8	134.23
5	125.91	128.14	134.80	159.30	144.9	154.74
6	128.04	124.12	120.15	130.46	113.8	144.07
7	-	-	-	-	-	-
8	105.37	112.85	111.87	126.81	132.1	121.76
9	82.56	83.31	87.66	116.69	102.4	115.11
10	115.26	117.70	116.38	109.92	121.4	108.54
11	116.27	101.91	89.00	131.67	139.0	115.44
12	144.59	104.29	113.73	127.88	100.72	115.31

13	102.16	104.09	104.87	118.11	110.33	114.27
14	123.32	108.61	-	111.46	106.65	-
15	118.13	99.63	95.02	132.05	115.16	115.20
Mean	117.35	107.43	109.47	127.38	118.70	125.01
SD	3.89	2.87	3.88	3.48	3.43	3.91

			VL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	153.61	157.33	161.29	138.24	130.2	148.69
2	109.70	162.03	-	167.85	159.0	-
3	148.96	158.02	160.20	161.65	143.3	146.91
4	173.53	158.57	177.19	158.87	182.3	163.04
5	121.73	128.19	119.17	116.02	122.7	114.37
6	169.33	162.08	157.12	173.47	177.1	-
7	-	-	-	-	-	-
8	172.94	174.99	170.59	171.69	161.5	172.47
9	143.11	169.30	164.58	129.21	130.1	126.31
10	166.19	175.54	178.86	176.93	213.0	201.08
11	138.76	140.86	137.25	-	-	140.56
12	185.35	175.61	166.70	158.39	172.85	155.73
13	161.22	163.48	177.25	180.26	161.25	166.26
14	111.55	115.68	-	165.00	172.97	-
15	146.41	88.94	140.75	148.30	157.63	151.64
Mean	150.17	152.19	159.25	157.38	160.30	153.37
SD	6.06	6.53	5.06	5.17	6.61	6.67

			BF			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	148.65	156.92	147.35	123.24	127.9	122.64
2	157.33	168.46	-	118.63	142.0	-
3	228.12	174.10	222.22	130.70	115.2	106.93
4	286.89	264.62	296.16	122.45	96.5	117.70
5	169.75	216.76	158.34	146.33	145.0	155.40
6	203.28	206.81	143.78		158.0	136.07
7	-	-	-	-	-	-

8	197.56	209.10	196.38	159.39	157.0	148.84
9	148.29	149.20	145.79	142.29	142.2	137.73
10	135.02	146.48	139.90	143.16	142.4	149.45
11	105.17	-	195.34	125.25	120.6	149.69
12	122.99	133.85	104.82	144.21	135.13	130.41
13	138.15	135.95	140.65	201.97	202.01	209.32
14	203.36	182.13	-	192.39	190.80	-
15	134.62	140.51	141.59	146.53	156.62	149.81
Mean	169.94	175.76	169.36	145.89	145.10	142.83
SD	12.58	10.39	14.12	6.87	7.16	7.12

			ТА			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	143.43	148.81	117.21	163.44	154.3	156.11
2	166.71	159.15	-	150.17	129.1	-
3	174.38	149.85	185.49	163.23	140.0	162.26
4	140.02	137.71	128.45	163.25	150.7	161.65
5	135.62	140.90	132.58	146.45	150.9	164.11
6	173.81	169.90	169.65	179.34	168.5	161.17
7	161.42	165.75	167.23	153.65	148.4	133.00
8	156.45	143.69	148.00	136.66	143.4	145.74
9	197.46	171.57	185.34	161.75	157.6	148.02
10	144.62	147.36	157.45	144.59	127.1	151.43
11	128.01	130.91	128.73	123.29	125.5	114.01
12	154.87	138.78	143.97	148.43	113.80	153.77
13	160.09	136.26	158.32	160.16	164.59	163.79
14	146.79	144.41	-	125.07	126.15	-
15	135.87	141.06	123.55	135.67	142.54	137.32
Mean	154.64	148.41	149.69	150.34	142.84	150.18
SD	4.59	3.15	6.13	3.89	3.95	3.94

			SOL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	157.24	154.29	157.52	160.22	151.8	151.69
2	174.49	170.31	-	182.02	183.1	-

	3	165.22	155.90	164.29	167.84	162.3	173.75
	4	150.08	155.70	152.61	164.03	163.0	167.92
	5	145.48	149.49	143.12	132.00	139.2	140.67
	6	165.33	170.76	174.19	161.27	174.1	174.58
	7	177.83	177.70	174.67	172.34	174.0	163.16
	8	161.92	165.88	153.84	171.16	171.1	169.98
	9	143.04	155.69	144.39	168.74	181.7	165.30
	10	167.02	183.44	179.10	153.27	165.2	161.78
	11	134.10	141.43	144.47	140.47	145.6	143.10
	12	175.10	177.42	155.29	179.90	182.30	182.43
	13	163.15	160.37	163.87	176.73	169.20	173.22
	14	176.37	175.55	-	166.51	170.94	-
	15	173.63	153.17	145.87	146.89	147.99	143.50
-	Mean	162.00	163.14	157.94	162.89	165.44	162.39
	SD	3.36	3.06	3.31	3.59	3.45	3.62

			VM			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	20.69	16.23	14.73	19.84	23.9	19.96
2	29.12	34.29	-	29.88	38.5	-
3	15.33	20.64	19.73	22.48	26.5	18.42
4	54.74	57.98	46.75	27.32	34.8	24.88
5	25.25	25.29	22.51	20.36	19.5	18.66
6	16.69	17.84	14.75	15.46	15.2	13.13
7	36.54	33.19	39.56	32.37	31.93	32.11
8	69.42	72.85	67.00	42.44	44.1	42.56
9	34.07	33.19	35.15	65.13	-	68.72
10	60.26	83.83	72.10	-	93.1	109.92
11	12.87	14.28	22.86	14.32	13.5	27.06
12	31.69	32.09	31.27	29.32	44.23	32.36
13	29.08	30.49	27.50	19.08	21.19	19.42
14	34.77	35.59	-	32.22	33.44	-
15	32.94	25.77	25.65	20.38	23.06	14.59
Mean	33.56	35.57	33.81	27.90	33.07	33.98
SD	4.10	5.09	4.90	3.40	5.12	7.25

# APPENDIX S: KNEE EXTENSOR VOLUNTARY ACTIVATION (RMS) DURING RUNNING

			RF			
	Non-Dominant Leg				Dominant Le	g
Subject #	Pre	Post	48H	Pre	Post	48H
1	14.05	13.31	10.24	10.83	10.0	9.55
2	7.79	11.19	-	9.68	7.2	-
3	6.12	8.84	7.49	4.08	5.2	5.31
4	12.05	13.29	12.06	10.66	10.1	8.07
5	11.02	10.74	9.43	8.35	8.4	6.61
6	5.60	5.50	6.57	6.91	6.1	6.14
7	8.62	7.86	8.54	8.85	7.25	7.72
8	16.47	17.33	15.93	12.75	10.4	11.13
9	14.97	14.27	15.45	7.10	7.9	6.33
10	39.77	65.84	68.53	30.24	49.6	37.94
11	12.65	13.54	9.11	11.54	11.0	6.42

14.09	15.64	11.69	12.35	15.68	11.88
9.44	10.66	10.54	10.08	14.81	10.08
7.72	6.58	-	13.31	13.20	-
7.15	7.04	7.54	8.99	6.75	7.42
12.50	14.78	14.86	11.05	12.24	10.35
2.06	3.63	4.36	1.46	2.69	2.27
	9.44 7.72 7.15 12.50	9.4410.667.726.587.157.0412.5014.78	9.4410.6610.547.726.58-7.157.047.5412.5014.7814.86	9.4410.6610.5410.087.726.58-13.317.157.047.548.9912.5014.7814.8611.05	9.4410.6610.5410.0814.817.726.58-13.3113.207.157.047.548.996.7512.5014.7814.8611.0512.24

			VL			
		Non-Dominant Leg			Dominant Leg	-
Subject #	Pre	Post	48H	Pre	Post	48H
1	16.07	15.39	14.71	18.51	17.8	20.55
2	26.80	20.95	-	23.10	22.8	-
3	14.49	21.37	15.23	17.28	17.6	11.52
4	19.75	23.16	18.15	18.61	27.5	20.79
5	20.11	19.30	21.21	28.46	23.7	19.25
6	8.71	8.81	8.78	15.02	16.4	13.54
7	19.93	14.94	17.64	23.07	23.86	27.14
8	27.52	27.79	26.52	25.11	27.3	22.73
9	22.32	23.59	25.65	25.95	30.5	24.40
10	31.20	38.50	35.71	48.32	57.8	44.04
11	17.14	15.26	14.23	10.7	10.6	13.71
12	13.72	20.12	17.94	24.50	26.85	22.62
13	15.84	16.11	17.09	13.34	15.84	13.50
14	41.50	36.85	-	16.64	14.98	-
15	26.24	28.43	18.11	25.14	20.92	18.77
Mean	21.42	22.04	19.31	22.25	23.63	20.97
SD	2.05	2.03	1.81	2.21	2.74	2.24

			BF			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	16.57	15.99	15.39	13.47	11.5	13.65
2	13.02	12.90	-	11.25	9.2	-
3	10.76	11.82	11.09	5.55	5.7	8.28
4	12.03	12.15	12.80	8.67	8.1	7.25
5	15.47	23.12	15.08	15.92	11.7	12.37
6	14.98	13.16	15.28	6.46	11.6	7.16

7	19.57	16.67	18.62	10.51	10.81	13.73
8	16.80	15.65	15.82	14.89	12.8	14.20
9	25.72	22.88	26.42	12.02	13.2	11.43
10	26.27	30.43	29.04	32.44	40.9	44.32
11	31.07	25.6	14.64	28.10	21.6	9.19
12	21.31	20.29	22.16	18.26	21.34	19.28
13	17.34	18.53	18.66	14.11	13.99	13.69
14	15.83	14.70	-	13.32	12.75	-
15	16.21	34.04	17.01	15.64	15.24	14.17
Mean	18.20	19.20	17.85	14.71	14.70	14.52
SD	1.42	1.70	1.39	1.81	2.09	2.55

			TA			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	40.39	38.15	35.23	56.93	64.6	50.12
2	49.96	61.61	-	31.03	60.1	-
3	42.14	55.68	49.07	82.84	38.7	79.85
4	38.08	51.62	41.42	35.10	37.6	30.51
5	25.32	23.25	25.52	18.75	15.2	19.45
6	28.97	24.69	22.96	28.08	23.1	18.41
7	60.80	63.10	55.15	94.98	85.0	99.02
8	81.04	70.23	86.86	59.44	72.3	57.28
9	47.98	47.29	47.09	48.80	53.6	52.72
10	67.45	88.53	91.26	64.38	77.8	85.75
11	24.96	24.59	27.05	28.04	25.8	32.92
12	46.79	38.42	46.53	63.07	49.71	46.59
13	26.54	40.30	29.89	27.07	44.26	39.77
14	70.23	72.54	-	73.69	66.61	-
15	32.36	39.74	33.29	41.22	37.43	39.36
Mean	45.53	49.32	45.49	50.23	50.12	50.13
SD	4.39	4.82	5.80	5.70	5.20	6.65

			SOL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	39.15	33.59	35.18	35.31	37.3	37.68

2	31.72	37.78	-	22.71	26.6	-	
3	23.09	22.62	21.77	27.95	18.1	16.71	
4	27.62	32.73	24.55	28.41	28.9	25.75	
5	23.73	20.10	24.67	25.29	23.3	25.90	
6	22.83	19.89	21.41	21.75	19.2	22.43	
7	42.36	35.50	37.89	38.94	36.8	39.82	
8	39.92	38.29	37.57	39.85	40.8	39.85	
9	33.20	36.30	35.23	23.91	21.0	21.77	
10	32.04	28.65	29.19	32.98	35.6	40.01	
11	27.60	24.35	18.08	24.68	22.0	19.85	
12	27.01	20.70	26.58	21.07	19.57	18.93	
13	30.49	26.53	20.35	22.61	25.96	24.49	
14	39.06	35.29	-	33.11	32.25	-	
15	22.23	26.72	22.18	25.58	25.93	22.74	
Mean	30.80	29.27	27.28	28.28	27.55	27.38	
SD	1.69	1.68	1.86	1.56	1.84	2.32	

			VM			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	88.21	104.66	96.08	99.74	97.6	101.65
2	85.89	69.85	-	96.32	78.3	-
3	95.51	90.21	102.24	110.54	115.6	120.20
4	77.78	74.83	89.45	99.04	93.5	107.06
5	81.57	85.52	89.66	79.13	89.6	88.16
6	94.37	87.00	89.16	109.94	97.8	95.51
7	98.74	114.89	81.38	135.52	126.30	125.09
8	108.51	105.29	108.48	142.12	146.2	140.34
9	70.60	83.52	73.26	101.53	82.7	79.66
10	92.10	95.48	95.37	-	112.9	149.28
11	72.85	73.03	79.50	77.11	74.8	79.91
12	77.08	105.95	89.56	98.05	90.98	94.81
13	94.11	103.28	91.63	100.02	103.19	105.58
14	106.04	114.26	-	89.59	90.40	-
15	125.86	140.47	125.99	109.68	102.89	113.18
Mean	91.28	96.55	93.21	103.45	100.18	107.73
SD	3.70	4.72	3.60	4.66	4.71	5.75

			RF				
		Non-Dominant Leg			Dominant Leg		
Subject #	Pre	Post	48H	Pre	Post	48H	
1	85.99	84.84	92.82	99.02	99.2	101.19	
2	99.92	91.38	-	82.24	96.0	-	
3	101.93	93.04	99.76	103.45	96.5	91.65	
4	90.42	88.26	92.19	82.18	80.6	85.97	
5	73.50	80.54	83.61	85.00	78.2	89.67	
6	108.28	103.46	109.87	102.95	97.7	112.14	
7	88.37	95.53	80.04	80.00	95.38	87.45	
8	98.64	90.76	91.09	95.95	100.1	95.95	
9	71.99	77.90	76.07	81.57	85.7	86.86	
10	128.46	110.64	122.08	99.01	132.2	112.12	
11	81.91	73.69	72.66	81.78	75.5	75.48	
12	148.13	90.56	86.38	92.82	96.32	92.26	

13	95.95	107.97	93.30	101.25	104.20	99.92
14	101.07	105.86	-	102.01	100.93	-
15	94.36	96.01	82.11	83.50	95.27	81.99
Mean	97.93	92.70	90.92	91.52	95.59	93.28
SD	4.91	2.73	3.64	2.33	3.34	2.89

			VL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	132.02	138.52	132.33	129.95	130.8	137.32
2	110.16	130.58	-	144.13	143.6	-
3	144.39	152.73	159.45	140.45	143.8	141.48
4	145.85	139.80	153.79	153.46	142.8	151.99
5	94.67	93.85	90.71	62.75	71.0	72.74
6	159.20	150.35	157.15	158.64	151.0	158.76
7	134.58	160.27	155.49	125.89	123.89	115.38
8	156.57	152.55	161.08	130.12	127.9	133.87
9	134.86	147.07	154.25	114.11	113.1	119.61
10	165.41	165.19	159.57	164.87	199.2	187.45
11	103.06	107.57	110.32	111.6	117.6	113.92
12	103.46	163.66	148.85	136.07	154.40	147.74
13	144.39	149.62	158.42	149.15	139.54	148.17
14	110.58	114.16	-	144.37	154.59	-
15	108.95	88.35	124.74	125.14	134.30	130.67
Mean	129.88	136.95	143.55	132.71	136.50	135.32
SD	5.74	6.20	5.95	6.16	6.80	7.28

			BF			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	117.68	123.11	124.80	109.11	116.8	111.28
2	100.06	128.17	-	95.19	109.7	-
3	111.34	115.71	110.14	105.62	104.8	88.99
4	128.24	141.98	116.95	102.71	101.1	103.19
5	110.64	114.95	133.28	91.62	100.4	105.82
6	127.68	121.40	100.16	113.40	72.9	118.90
7	120.37	108.38	94.33	108.77	115.39	98.53

8	144.85	147.06	147.06	149.27	157.7	152.24
9	139.44	142.51	139.47	112.84	115.1	109.35
10	150.92	162.88	124.07	132.41	133.7	141.92
11	92.83	110.2	113.08	106.64	110.7	112.18
12	82.48	96.62	78.98	111.53	116.46	105.91
13	125.06	112.28	117.12	131.84	138.71	132.96
14	139.54	142.02	-	140.00	143.20	-
15	120.77	81.38	112.14	123.59	132.49	129.44
Mean	120.79	123.24	116.28	115.64	117.94	116.21
SD	4.80	5.31	4.93	4.11	5.18	4.83

			TA			
	Non-Dominant Leg				Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	139.37	142.94	121.27	152.77	142.8	157.55
2	174.24	170.57	-	146.75	126.8	-
3	165.24	144.58	150.75	131.16	130.2	135.59
4	140.34	144.06	134.29	160.18	150.2	170.12
5	125.94	130.70	122.44	128.03	138.2	146.92
6	160.55	163.55	164.16	169.09	157.0	158.13
7	158.61	162.78	151.95	146.65	149.7	136.01
8	149.61	137.47	144.03	138.34	144.1	143.05
9	199.61	176.44	194.96	164.21	158.3	159.97
10	158.59	141.65	148.21	152.06	130.1	144.46
11	127.77	125.10	128.18	123.28	123.6	108.41
12	159.01	154.92	163.94	146.46	124.88	167.75
13	147.14	142.48	156.45	150.62	162.37	162.15
14	137.76	135.02	-	121.01	127.18	-
15	111.90	135.80	109.93	117.61	134.84	129.88
Mean	150.38	147.20	145.43	143.21	140.02	147.69
SD	5.38	3.77	6.03	4.00	3.27	4.65

			SOL			
		Non-Dominant Leg			Dominant Leg	5
Subject #	Pre	Post	48H	Pre	Post	48H
1	128.98	128.35	137.16	129.32	128.0	128.43
2	153.72	151.41	-	158.67	161.7	-

	3	137.22	137.18	138.07	138.01	146.8	141.54
	4	115.57	121.22	122.35	135.22	139.6	141.63
	5	126.66	134.44	128.70	121.26	123.4	120.68
	6	131.79	146.37	138.86	127.35	140.3	140.55
	7	123.61	150.72	141.56	150.66	153.6	141.42
	8	134.13	129.82	126.28	148.29	137.7	145.33
	9	119.31	125.69	115.86	133.93	153.6	141.62
	10	142.81	155.26	151.22	131.55	142.4	139.90
	11	118.04	121.08	126.75	121.39	124.8	130.31
	12	154.39	162.33	135.34	161.97	158.36	166.06
	13	134.77	144.60	138.36	159.03	157.14	153.05
	14	157.66	153.90	-	147.41	152.64	-
	15	126.51	125.49	120.62	129.98	139.10	130.78
-	Mean	133.68	139.19	132.39	139.60	143.94	140.10
	SD	3.34	3.42	2.64	3.42	3.06	3.05

		RQ			PQ	
Subject #	Pre	Post	48H	Pre	Post	48H
1	1.55	1.97	0.96	1.18	2.27	2.59
2	2.71	4.27	-	1.27	5.44	-
3	0.72	0.62	3.60	1.51	4.79	4.89
4	1.79	1.17	1.35	1.95	0.96	2.05
5	-	-	-	-	-	-
6	0.78	2.61	2.34	1.53	3.86	7.22
7	2.58	0.99	4.58	3.47	1.12	2.58
8	1.08	0.87	2.32	4.47	2.65	1.34
9	0.49	1.28	0.91	1.75	5.14	3.65
10	1.49	0.45	1.77	3.61	0.64	1.67
11*	1.67	1.14	2.11	0.89	1.61	1.37
12	0.51	0.6	3.8	1.48	2.13	2.83
13	6.09	3.42	0.68	7.26	5.56	0.91
14	2.71	4.27	-	1.27	5.44	-
15	1.83	2.26	.91	5.81	7.35	1.16
Mean	1.86	1.85	2.11	2.68	3.50	2.69
SD	0.37	0.34	0.36	0.51	0.55	0.51

# APPENDIX U: ROMBERG'S QUOTIENT AND PROCIOCEPTION QUOTIENT

\*Dropped from analysis. Raw data was irregular.

		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	0.0	0.0	4.0	0.0	0.0	2.0
2	0.0	9.5	-	3.0	55.7	-
3	0.0	1.7	14.0	0.0	0.0	15.0
4	0.0	8.0	18.0	0.0	0.0	25.0
5	0.3	0.0	0.0	0.0	0.7	0.0
6	0.0	1.0	9.0	0.0	0.0	9.7
7	0.0	0.0	0.0	0.0	0.0	0.0
8	3.0	10.0	1.3	0.0	3.5	1.8
9	0.0	18.3	16.7	1.0	7.0	27.3
10	0.7	19.0	18.7	2.0	17.0	14.0
11	0.0	0.0	0.0	0.7	1.3	0.0
12	0.0	0.0	12.0	0.0	0.0	10.5
13	0.8	0.8	6.8	1.0	1.5	11.2
14	0.0	0.0	-	0.0	0.0	-
15	0.0	0.2	0.0	0.7	2.8	0.0
Mean	0.3	4.6	7.7	0.6	6.0	9.0
SD	0.2	1.7	2.0	0.2	3.6	2.5

## APPENDIX V: KNEE EXTENSOR SORENESS ON STEP TEST

# APPENDIX W: KNEE PAIN

		Left Leg			Right Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	0.0	0.0	0.0	5.0	10.0	12.0
2	2.7	16.3	-	2.3	55.3	-
3	0.0	0.0	23.0	0.0	0.0	16.0
4	0.0	0.0	0.0	0.0	0.7	0.0
5	0.3	0.0	0.0	0.0	0.0	1.0
6	0.0	0.0	1.0	0.3	0.0	0.0
7	0.0	0.0	0.0	0.0	0.0	0.0
8	0.0	4.0	0.0	0.2	0.8	0.2
9	0.0	1.7	0.3	0.3	3.3	0.0
10	0.0	0.0	0.0	0.0	0.0	0.0
11	0.0	0.7	0.0	0.0	0.0	0.0
12	0.0	0.0	5.7	0.0	0.0	6.0

13	0.0	0.3	0.0	0.0	0.3	1.0
14	0.0	0.0	-	0.0	0.0	-
15	0.7	5.8	0.5	0.5	6.3	0.7
Mean	0.2	1.9	2.3	0.6	5.1	2.8
SD	0.2	1.1	1.7	0.3	3.5	1.4

### APPENDIX X: INDIVIDUAL MUSCLE SORENESS

			VM			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	0.00	0.00	0.00	0.00	0.00	0.00
2	0.75	3.50	-	0.75	13.50	-
3	0.00	0.00	3.50	0.00	0.00	1.00
4	0.00	3.00	6.50	0.00	5.50	3.00
5	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	0.00	0.00	0.00	0.25
7	0.00	0.00	0.00	0.00	0.00	0.50
8	0.25	4.75	0.00	0.75	0.25	0.88
9	0.00	0.50	15.50	0.50	6.00	10.00
10	0.00	2.50	4.75	0.00	0.00	3.75
11	1.00	0.00	0.00	0.00	0.00	0.38
12	0.00	0.13	10.25	0.00	1.63	19.63
13	0.50	2.00	1.75	0.25	0.50	1.38
14	0.00	0.00	-	0.00	2.25	-
15	1.13	6.88	0.00	0.50	2.25	0.00
Mean	0.24	1.55	3.25	0.18	2.13	3.14
SD	0.10	0.54	1.31	0.07	0.93	1.51

			RF			
	Ν	Non-Dominant Le	g		Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	0.00	0.00	0.00	0.00	0.00	0.00
2	1.50	0.75	-	0.75	2.50	-
3	0.00	0.00	4.50	0.00	0.00	5.00
4	0.00	0.00	3.50	0.00	0.00	3.00
5	0.00	0.00	0.00	0.00	0.25	0.00

6	0.00	0.50	5.75	0.00	0.00	6.00
7	0.00	0.00	0.00	0.00	0.00	0.50
8	0.38	4.88	0.00	0.00	4.50	1.00
9	1.00	1.25	14.00	1.00	0.00	3.00
10	0.00	2.00	0.00	0.00	0.75	5.25
11	0.38	0.38	0.00	0.00	0.00	0.75
12	0.00	0.00	12.75	0.00	0.75	13.00
13	0.00	0.00	3.63	0.00	0.63	3.13
14	0.00	0.00	-	0.00	0.00	-
15	0.25	0.63	0.75	0.00	1.00	0.00
Mean	0.23	0.69	3.45	0.12	0.69	3.13
SD	0.11	0.32	1.29	0.08	0.31	0.97

			VL			
		Non-Dominant Leg			Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	0.00	0.00	0.75	0.00	0.00	1.00
2	0.75	1.50	-	0.00	1.00	-
3	0.00	0.00	20.50	1.00	0.00	2.25
4	0.00	0.00	10.00	0.00	0.00	8.25
5	0.00	0.00	0.00	0.00	0.00	0.00
6	0.00	0.00	9.00	0.00	1.00	8.25
7	0.00	0.00	0.00	0.00	0.75	0.00
8	0.00	2.50	0.00	0.13	3.50	0.88
9	1.00	2.50	21.00	0.50	0.25	9.00
10	0.50	1.50	8.00	0.50	2.50	7.25
11	0.25	0.38	0.25	0.00	0.13	0.50
12	0.00	1.38	9.88	0.00	0.50	13.13
13	0.50	2.13	6.38	0.75	0.88	5.75
14	0.00	0.00	-	0.00	0.00	-
15	0.50	2.13	6.38	0.75	0.88	5.75
Mean	0.23	0.93	7.09	0.24	0.76	4.77
SD	0.08	0.26	1.94	0.09	0.25	1.14

	Ν	on-Dominant	Leg		Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	55.0	55.4	55.3	55.0	55.5	55.0
2	65.8	67.0	-	65.2	66.5	-
3	64.5	65.5	66.0	64.0	65.5	65.0
4	60.0	60.0	61.5	60.0	60.0	61.5
5	56.0	56.0	55.3	56.0	56.0	54.8
6	63.8	66.1	65.0	64.7	67.0	65.7
7	62.8	62.7	64.0	62.0	62.5	63.8
8	54.0	53.5	53.0	56.0	56.0	54.5
9	51.6	51.5	51.0	50.0	51.9	52.4
10	45.2	44.0	45.0	45.0	44.7	46.0
11	63.5	63.7	64.0	62.0	62.5	63.0
12	54.0	54.3	54.9	54.8	56.0	56.0
13	49.4	49.6	49.4	49.6	49.0	49.7
14	63.0	63.5	-	62.5	64.0	-
15	62.3	62.0	61.7	61.0	62.0	62.0
Mean	58.1	58.3	57.4	57.9	58.6	57.6
SD	1.6	1.7	1.8	1.5	1.7	1.7

# APPENDIX Y: LIMB CIRCUMFERENCE

# APPENDIX Z: KNEE RANGE OF MOTION

		Non-Dominant Leg		]	Dominant Leg	
Subject #	Pre	Post	48H	Pre	Post	48H
1	138.0	135.0	139.5	135.0	136.0	135.5
2	138.0	133.5	-	130.0	130.0	-
3	129.0	121.0	130.0	126.8	123.0	125.0
4	141.0	139.0	139.0	133.0	138.0	138.0
5	142.0	143.0	142.0	138.0	141.0	141.0
6	135.0	133.5	135.0	136.0	129.0	144.0
7	136.0	139.5	140.5	137.0	139.0	138.0
8	132.0	138.5	136.5	136.0	139.0	141.0
9	141.0	139.0	140.5	137.0	133.0	138.5
10	157.0	155.0	161.0	147.0	148.0	159.0
11	129.0	124.5	132.0	132.0	127.0	130.0
12	123.0	141.0	126.0	125.0	131.0	133.0

13	137.0	135.5	135.0	131.5	135.0	134.0
14	140.5	143.0	-	130.0	130.0	-
15	139.0	139.0	140.0	139.0	139.0	141.0
Mean	137.2	137.3	138.2	134.2	134.5	138.3
SD	1.9	2.0	2.2	1.4	1.6	2.2

# APPENDIX AA: MVC TORQUE ANALYSIS

#### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Angle	Sphericity Assumed	286920.655	2	143460.327	67.394	.000	.838	134.788	1.000
	Greenhouse-Geisser	286920.655	1.375	208718.538	67.394	.000	.838	92.645	1.000
	Huynh-Feldt	286920.655	1.602	179136.217	67.394	.000	.838	107.944	1.000
	Lower-bound	286920.655	1.000	286920.655	67.394	.000	.838	67.394	1.000
Angle * Group	Sphericity Assumed	7173.709	2	3586.854	1.685	.205	.115	3.370	.322
	Greenhouse-Geisser	7173.709	1.375	5218.467	1.685	.215	.115	2.316	.263
	Huynh-Feldt	7173.709	1.602	4478.838	1.685	.212	.115	2.699	.285
	Lower-bound	7173.709	1.000	7173.709	1.685	.217	.115	1.685	.225
Error(Angle)	Sphericity Assumed	55345.614	26	2128.677					
	Greenhouse-Geisser	55345.614	17.871	3096.985					
	Huynh-Feldt	55345.614	20.822	2658.040					
	Lower-bound	55345.614	13.000	4257.355					
Time	Sphericity Assumed	6473.451	2	3236.725	6.519	.005	.334	13.038	.871
	Greenhouse-Geisser	6473.451	1.910	3388.711	6.519	.006	.334	12.453	.859
	Huynh-Feldt	6473.451	2.000	3236.725	6.519	.005	.334	13.038	.871
	Lower-bound	6473.451	1.000	6473.451	6.519	.024	.334	6.519	.656
Time * Group	Sphericity Assumed	10774.367	2	5387.184	10.850	.000	.455	21.700	.982
	Greenhouse-Geisser	10774.367	1.910	5640.147	10.850	.000	.455	20.727	.978
	Huynh-Feldt	10774.367	2.000	5387.184	10.850	.000	.455	21.700	.982
	Lower-bound	10774.367	1.000	10774.367	10.850	.006	.455	10.850	.861
Error(Time)	Sphericity Assumed	12909.205	26	496.508					
	Greenhouse-Geisser	12909.205	24.834	519.822					
	Huynh-Feldt	12909.205	26.000	496.508					
	Lower-bound	12909.205	13.000	993.016					
Angle * Time	Sphericity Assumed	1185.089	4	296.272	2.561	.049	.165	10.246	.683
	Greenhouse-Geisser	1185.089	2.355	503.228	2.561	.086	.165	6.032	.512
	Huynh-Feldt	1185.089	3.131	378.558	2.561	.066	.165	8.019	.600
	Lower-bound	1185.089	1.000	1185.089	2.561	.134	.165	2.561	.317
Angle * Time * Group	Sphericity Assumed	1347.788	4	336.947	2.913	.030	.183	11.652	.746
	Greenhouse-Geisser	1347.788	2.355	572.315	2.913	.062	.183	6.860	.569
	Huynh-Feldt	1347.788	3.131	430.530	2.913	.044	.183	9.119	.663
	Lower-bound	1347.788	1.000	1347.788	2.913	.112	.183	2.913	.353
Error(Angle*Time)	Sphericity Assumed	6014.768	52	115.669					
	Greenhouse-Geisser	6014.768	30.615	196.467					
	Huynh-Feldt	6014.768	40.697	147.794					
	Lower-bound	6014.768	13.000	462.674					

a. Computed using alpha = .05

Measure: MEASURE\_1

Measur				Mean			95% Confiden Differ	
Group	Angle	(I) Time	(J) Time	Difference (I- J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	1	2	-2.586	5.625	1.000	-18.030	12.859
			3	-7.107	4.483	.411	-19.416	5.202
		2	1	2.586	5.625	1.000	-12.859	18.030
			3	-4.521	4.908	1.000	-18.000	8.957
		3	1	7.107	4.483	.411	-5.202	19.416
			2	4.521	4.908	1.000	-8.957	18.000
	2	1	2	-5.972	6.720	1.000	-24.426	12.482
			3	-18.131	6.458	.044	-35.866	397
		2	1	5.972	6.720	1.000	-12.482	24.426
			3	-12.160	7.890	.442	-33.826	9.507
		3	1	18.131	6.458	.044	.397	35.866
			2	12.160	7.890	.442	-9.507	33.826
	3	1	2	-4.771	12.096	1.000	-37.987	28.444
			3	.025	9.513	1.000	-26.097	26.147
		2	1	4.771	12.096	1.000	-28.444	37.987
			3	4.797	12.732	1.000	-30.164	39.757
		3	1	025	9.513	1.000	-26.147	26.097
			2	-4.797	12.732	1.000	-39.757	30.164
Inj	1	1	2	23.874	5.261	.002	9.427	38.321
			3	14.943	4.193	.010	3.429	26.458
		2	1	-23.874	5.261	.002	-38.321	-9.427
			3	-8.931	4.591	.221	-21.539	3.677
		3	1	-14.943	4.193	.010	-26.458	-3.429
			2	8.931	4.591	.221	-3.677	21.539
	2	1	2	36.471	6.286	.000	19.209	53.733
			3	20.475	6.041	.015	3.886	37.064
		2	1	-36.471	6.286	.000	-53.733	-19.209
			3	-15.997	7.381	.148	-36.264	4.271
		3	1	-20.475	6.041	.015	-37.064	-3.886
			2	15.997	7.381	.148	-4.271	36.264
	3	1	2	54.267	11.315	.001	23.196	85.337
			3	29.990	8.898	.015	5.555	54.425
		2	1	-54.267	11.315	.001	-85.337	-23.196
			3	-24.277	11.909	.187	-56.979	8.426
		3	1	-29.990	8.898	.015	-54.425	-5.555
		Ū.	2	24.277	11.909	.187	-8.426	56.979
		tod morain		24.211	11.909	.107	-0.420	50.979

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AB: 20 & 80 HZ RAW TORQUE ANALYSIS

#### Tests of Within-Subjects Effects

		Type III Sum					Partial Eta	Noncent.	Observed
Source		of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Power <sup>a</sup>
req	Sphericity Assumed	25936.600	1	25936.600	125.991	.000	.913	125.991	1.00
	Greenhouse-Geisser	25936.600	1.000	25936.600	125.991	.000	.913	125.991	1.00
	Huynh-Feldt	25936.600	1.000	25936.600	125.991	.000	.913	125.991	1.00
	Lower-bound	25936.600	1.000	25936.600	125.991	.000	.913	125.991	1.00
req * Group2	Sphericity Assumed	11.123	1	11.123	.054	.820	.004	.054	.05
	Greenhouse-Geisser	11.123	1.000	11.123	.054	.820	.004	.054	.05
	Huynh-Feldt	11.123	1.000	11.123	.054	.820	.004	.054	.05
	Lower-bound	11.123	1.000	11.123	.054	.820	.004	.054	.05
rror(Freq)	Sphericity Assumed	2470.329	12	205.861					
	Greenhouse-Geisser	2470.329	12.000	205.861					
	Huynh-Feldt	2470.329	12.000	205.861					
	Lower-bound	2470.329	12.000	205.861					
luscle	Sphericity Assumed	4081.975	2	2040.987	5.021	.015	.295	10.041	.76
	Greenhouse-Geisser	4081.975	1.348	3028.606	5.021	.031	.295	6.767	.63
	Huynh-Feldt	4081.975	1.584	2577.582	5.021	.024	.295	7.951	.61
	Lower-bound	4081.975	1.000	4081.975	5.021	.045	.295	5.021	.54
luscle * Group2	Sphericity Assumed	197.122	2	98.561	.242	.787	.020	.485	.08
	Greenhouse-Geisser	197.122	1.348	146.254	.242	.700	.020	.327	.01
	Huynh-Feldt	197.122	1.584	124.474	.242	.736	.020	.384	30.
	Lower-bound	197.122	1.000	197.122	.242	.631	.020	.242	.07
rror(Muscle)	Sphericity Assumed	9756.675	24	406.528					
	Greenhouse-Geisser	9756.675	16.174	603.244					
	Huynh-Feldt	9756.675	19.004	513.408					
	Lower-bound	9756.675	12.000	813.056					
îme	Sphericity Assumed	223.515	2	111.758	8.437	.002	.413	16.874	.9
	Greenhouse-Geisser	223.515	1.959	114.094	8.437	.002	.413	16.529	.9:
	Huynh-Feldt	223.515	2.000	111.758	8.437	.002	.413	16.874	.9
	Lower-bound	223.515	1.000	223.515	8.437	.013	.413	8.437	.7
Time * Group2	Sphericity Assumed Greenhouse-Geisser	128.992	2	64.496	4.869	.017	.289	9.738	.7
		128.992	1.959	65.844	4.869	.018	.289	9.539	.7
	Huynh-Feldt	128.992	2.000	64.496	4.869	.017	.289	9.738	.74
rror(Time)	Lower-bound	128.992	1.000	128.992	4.869	.048	.289	4.869	.5:
rror(Time)	Sphericity Assumed	317.906	24	13.246					
	Greenhouse-Geisser	317.906	23.509	13.523					
	Huynh-Feldt	317.906	24.000	13.246					
	Lower-bound	317.906	12.000	26.492					
req * Muscle	Sphericity Assumed	881.982	2	440.991	5.615	.010	.319	11.229	.81
	Greenhouse-Geisser	881.982	1.259	700.587	5.615	.025	.319	7.068	.65
	Huynh-Feldt	881.982	1.455	606.306 881.982	5.615		.319	8.168	.70
	Lower-bound	881.982 51.821	1.000	25.911	5.615	.035	.319	5.615	.51
req * Muscle * Group2	Sphericity Assumed								
	Greenhouse-Geisser	51.821	1.259	41.163	.330	.625	.027	.415	0. 20.
	Huynh-Feldt	51.821	1.455	35.624 51.821	.330	.655	.027	.480	.05
Error(Freq*Muscle)	Lower-bound Sphericity Assumed	51.821 1885.000	24	78.542	.330	.570	.027	.330	.00
chor(Fred muscle)	Greenhouse-Geisser	1885.000	15.107	124,776					
	Huynh-Feldt	1885.000	17.456	107.985					
	Lower-bound	1885.000	12.000	157.083					
reg * Time	Sphericity Assumed	66.302	12.000	33.151	10.457	.001	.466	20.914	.97
req mile	Greenhouse-Geisser	66.302	1.358	48.822	10.457	.001	.466	14.201	.91
	Huynh-Feldt	66.302	1.599	41.475	10.457	.003	.466	16.716	.9
	Lower-bound	66.302	1.000	66.302	10.457	.002	.466	10.457	.8
req * Time * Group2		14.848	2	7.424	2.342	.007	.400	4.683	.0
req " Time " Group2	Sphericity Assumed Greenhouse-Geisser	14.040	1.358	10.933	2.342	.110	.163	4.683	.4
	Huynh-Feldt	14.848	1.599	9.288	2.342	.131	.163	3.743	.3
mor/FrogtTimes)	Lower-bound	14.848		14.848	2.342	.152	.163	2.342	.2
rror(Freq*Time)	Sphericity Assumed Greenhouse-Geisser	76.087 76.087	24 16.296	3.170					
	Huynh-Feldt	76.087	16.296	4.669					
	Lower-bound	76.087	12.000	6.341					
luscle * Time	Sphericity Assumed	38.844	12.000	9,711	2.184	.085	.154	8.737	.6
	Greenhouse-Geisser	38.844	3.117	9.711	2.184	.085	.154	6.808	.61
	Huynh-Feldt	38.844	4.000	9.711	2.184	.085	.154	8.737	.6
	Lower-bound	38.844	1.000	38.844	2.184	.085	.154	2.184	.0
luscle * Time * Group2	Sphericity Assumed	6.751	4	1.688	.380	.105	.031	1.519	.2
010002	Greenhouse-Geisser	6.751	3.117	2.166	.380	.822	.031	1.515	
	Huynh-Feldt	6.751	4.000	1.688	.380	.822	.031	1.519	.1:
	Lower-bound	6.751	1.000	6.751	.380	.549	.031	.380	.0
rror(Muscle*Time)	Sphericity Assumed	213.404	48	4.446					.0
	Greenhouse-Geisser	213.404	37.405	5.705					
	Huynh-Feldt	213.404	48.000	4.446					
	Lower-bound	213.404	12.000	17.784					
req * Muscle * Time	Sphericity Assumed	3.066	4	.766	.377	.824	.030	1.508	.1
	Greenhouse-Geisser	3.066	3.193	.960	.377	.782	.030	1.204	.1
	Huynh-Feldt	3.066	4.000	.766	.377	.824	.030	1.508	.10
	Lower-bound	3.066	1.000	3.066	.377	.551	.030	.377	.0
req * Muscle * Time *	Sphericity Assumed	10.920	4	2.730	1.343	.268	.101	5.372	.3
iroup2	Greenhouse-Geisser	10.920	3.193	3.420	1.343	.200	.101	4.288	.3
	Huynh-Feldt	10.920	4.000	2.730	1.343	.268	.101	5.372	.3
	Lower-bound	10.920	1.000	10.920	1.343	.269	.101	1.343	.0
rror(Freq*Muscle*Time)	Sphericity Assumed	97.565	48	2.033					
	Greenhouse-Geisser	97.565	38.311	2.547					
	Huynh-Feldt	97.565	48.000	2.033					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval for Difference <sup>b</sup>		
Group2	(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound	
Con	1	2	.825	.813	.991	-1.436	3.086	
		3	472	.916	1.000	-3.018	2.074	
	2	1	825	.813	.991	-3.086	1.436	
	3		-1.297	.841	.447	-3.634	1.040	
	3 1		.472	.916	1.000	-2.074	3.018	
		2	1.297	.841	.447	-1.040	3.634	
Inj	1	2	3.833	.704	.000	1.875	5.791	
		3	2.650	.793	.018	.445	4.855	
	2 <u>1</u> 3		-3.833	.704	.000	-5.791	-1.875	
			-1.183	.728	.390	-3.207	.841	
	3	1	-2.650	.793	.018	-4.855	445	
		2	1.183	.728	.390	841	3.207	

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

#### Pairwise Comparisons

Measur	re: MEA	SURE_1						
				Mean Difference (I-			95% Confiden Differ	ce Interval for ence <sup>b</sup>
Angle	Time	(I) Group	(J) Group	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1	1	Con	Inj	-1.714	14.081	.905	-32.133	28.705
		Inj	Con	1.714	14.081	.905	-28.705	32.133
	2	Con	Inj	24.746	12.562	.071	-2.393	51.885
		Inj	Con	-24.746	12.562	.071	-51.885	2.393
	3	Con	Inj	20.337	14.089	.173	-10.102	50.775
		Inj	Con	-20.337	14.089	.173	-50.775	10.102
2	1	Con	Inj	.572	21.603	.979	-46.098	47.243
		Inj	Con	572	21.603	.979	-47.243	46.098
		Con	Inj	43.016	21.514	.067	-3.462	89.493
		Inj	Con	-43.016	21.514	.067	-89.493	3.462
	3	Con	Inj	39.179	25.621	.150	-16.173	94.530
		Inj	Con	-39.179	25.621	.150	-94.530	16.173
3	1	Con	Inj	20.189	31.398	.531	-47.642	88.021
		Inj	Con	-20.189	31.398	.531	-88.021	47.642
	2	Con	Inj	79.227*	33.929	.036	5.927	152.527
		Inj	Con	-79.227*	33.929	.036	-152.527	-5.927
	3	Con	Inj	50.154	32.797	.150	-20.699	121.006
		Inj	Con	-50.154	32.797	.150	-121.006	20.699

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AC: 20 & 80 HZ NORMALIZED TORQUE ANALYSIS

#### Tests of Within-Subjects Effects

	T	pe III Sum					Partial Eta	Noncent.	Observed
Source	C	f Squares	df	Mean Square	F	Sig.	Squared	Parameter	Power <sup>a</sup>
luscle	Sphericity Assumed	.054	2	.027	1.898	.172	.137	3.797	.35
	Greenhouse-Geisser	.054	1.935	.028	1.898	.173	.137	3.674	.34
	Huynh-Feldt	.054	2.000	.027	1.898	.172	.137	3.797	.35
	Lower-bound	.054	1.000	.054	1.898	.193	.137	1.898	.24
/uscle * Group2	Sphericity Assumed	.029	2	.014	1.009	.380	.078	2.018	.20
	Greenhouse-Geisser	.029	1.935	.015	1.009	.378	.078	1.952	.20
	Huynh-Feldt Lower-bound	.029	2.000	.014	1.009	.380	.078	2.018	.20
Error(Muscle)	Sphericity Assumed	.029	24	.029	1.009	.335	.078	1.009	.15
citor(wuscie)	Greenhouse-Geisser	.341	23.224	.014					
	Huynh-Feldt	.341	24.000	.014					
	Lower-bound	.341	12.000	.028					
Freq	Sphericity Assumed	.052	1	.052	2.186	.165	.154	2.186	.2
	Greenhouse-Geisser	.052	1.000	.052	2.186	.165	.154	2.186	.2
	Huynh-Feldt	.052	1.000	.052	2.186	.165	.154	2.186	.2
	Lower-bound	.052	1.000	.052	2.186	.165	.154	2.186	.2
req * Group2	Sphericity Assumed	.097	1	.097	4.092	.066	.254	4.092	.46
	Greenhouse-Geisser	.097	1.000	.097	4.092	.066	.254	4.092	.46
	Huynh-Feldt	.097	1.000	.097	4.092	.066	.254	4.092	.46
	Lower-bound	.097	1.000	.097	4.092	.066	.254	4.092	.41
Error(Freq)	Sphericity Assumed	.284	12	.024					
	Greenhouse-Geisser	.284	12.000	.024					
	Huynh-Feldt	.284	12.000	.024					
	Lower-bound	.284	12.000	.024					
Fime	Sphericity Assumed	.199	1	.199	12.294	.004	.506	12.294	.89
	Greenhouse-Geisser	.199	1.000	.199	12.294	.004	.506	12.294	.8
	Huynh-Feldt	.199	1.000	.199	12.294	.004	.506	12.294	.8
	Lower-bound	.199	1.000	.199	12.294	.004	.506	12.294	.8
Time * Group2	Sphericity Assumed	.003	1	.003	.204	.660	.017	.204	.0
	Greenhouse-Geisser	.003	1.000	.003	.204	.660	.017	.204	.0
	Huynh-Feldt	.003	1.000	.003	.204	.660	.017	.204	.0
	Lower-bound Sphericity Assumed	.003	1.000	.003	.204	.660	.017	.204	.0
Error(Time)	Greenhouse-Geisser	.195	12	.016					
	Huynh-Feldt		12.000	.016					
	Lower-bound	.195	12.000	.016					
/uscle * Freq	Sphericity Assumed	.007	12.000	.010	.515	.604	.041	1.030	.1:
Nuscie - Freq	Greenhouse-Geisser	.007	1.458	.004	.515	.550	.041	.751	
	Huynh-Feldt	.007	1.747	.003	.515	.581	.041	.900	
	Lower-bound	.007	1.000	.007	.515	.487	.041	.515	.1
Muscle * Freq * Group2	Sphericity Assumed	.027	2	.014	1.953	.164	.140	3.906	.3
	Greenhouse-Geisser	.027	1.458	.019	1.953	.178	.140	2.848	.3
	Huynh-Feldt	.027	1.747	.016	1.953	.170	.140	3.412	.3
	Lower-bound	.027	1.000	.027	1.953	.188	.140	1.953	.25
Error(Muscle*Freq)	Sphericity Assumed	.168	24	.007					
	Greenhouse-Geisser	.168	17.501	.010					
	Huynh-Feldt	.168	20.966	.008					
	Lower-bound	.168	12.000	.014					
duscle * Time	Sphericity Assumed	.024	2	.012	3.198	.059	.210	6.396	.5
	Greenhouse-Geisser	.024	1.859	.013	3.198	.063	.210	5.945	.5:
	Huynh-Feldt	.024	2.000	.012	3.198	.059	.210	6.396	.5
	Lower-bound	.024	1.000	.024	3.198	.099	.210	3.198	.3
/luscle * Time * Group2	Sphericity Assumed	.007	2	.004	.946	.402	.073	1.892	.11
	Greenhouse-Geisser	.007	1.859	.004	.946	.397	.073	1.759	.18
	Huynh-Feldt	.007	2.000	.004	.946	.402	.073	1.892	.13
	Lower-bound	.007	1.000	.007	.946	.350	.073	.946	.1
Error(Muscle*Time)	Sphericity Assumed	.089	24	.004					
	Greenhouse-Geisser	.089	22.308	.004					
	Huynh-Feldt	.089	24.000	.004					
and the second	Lower-bound	.089	12.000	.007	44.075		Ber		
req * Time	Sphericity Assumed	.204	1	.204	44.375	.000	.787	44.375	1.0
	Greenhouse-Geisser	.204	1.000	.204	44.375	.000	.787	44.375	1.0
	Huynh-Feldt	.204	1.000	.204	44.375	.000	.787	44.375	1.0
reg * Time * Group2	Lower-bound	.204	1.000	.204	44.375 2.460	.000	.787	44.375 2.460	1.0
reg - Time - Group2	Sphericity Assumed Greenhouse-Geisser	.011	1.000	.011	2.460	.143	.170	2.460	.3
	Greenhouse-Geisser Huynh-Feldt	.011	1.000	.011	2.460	.143	.170	2.460	.3
	Lower-bound	.011	1.000	.011	2.460	.143	.170	2.460	.3
Error(Freq*Time)	Sphericity Assumed	.011	1.000	.011	2.400	.143	.170	2.400	.3
(ried rille)	Greenhouse-Geisser	.055	12.000	.005					
	Huynh-Feldt	.055	12.000	.005					
	Lower-bound	.055	12.000	.005					
/uscle * Freq * Time	Sphericity Assumed	.003	12.000	.005	.742	.487	.058	1.484	.1
and a story time	Greenhouse-Geisser	.003	1.998	.001	.742	.487	.058	1.484	.1
	Huynh-Feldt	.003	2.000	.001	.742	.487	.058	1.482	.1
	Lower-bound	.003	1.000	.001	.742	.487	.058	.742	.1:
luscle * Freq * Time *	Sphericity Assumed	.003	2	.005	2.596	.406	.058	5.193	.1.
Group2	Greenhouse-Geisser	.009	1.998	.005	2.596	.095	.178	5.193	.4
	Huvnh-Feldt	.009	2.000	.005	2.596	.095	.178	5.188	.4
	Lower-bound	.009	1.000	.005	2.596	.095	.178	2.596	.4
Error(Muscle*Freq*Time)	Sphericity Assumed	.009	24	.009	2.000	.133	.170	2.580	
nuscle rieq (ime)	Greenhouse-Geisser	.043		.002					
	Huynh-Feldt	.043	23.978	.002					

Measure:	MEASURE_1
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		Mean Difference (I-			95% Confiden Differ	
(I) Group2	(J) Group2	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	Inj	.139	.040	.005	.051	.227
Inj	Con	139	.040	.005	227	051

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

### **Pairwise Comparisons**

Measure: MEASURE\_1

					95% Confidence Interval for			
		Mean Difference			Differ	ence <sup>a</sup>		
(I) Muscle	(J) Muscle	(I-J)	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound		
1	2	.022	.024	1.000	045	.090		
	3	.044	.021	.160	013	.102		
2	1	022	.024	1.000	090	.045		
	3	.022	.023	1.000	042	.086		
3	1	044	.021	.160	102	.013		
	2	022	.023	1.000	086	.042		

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

### **Pairwise Comparisons**

Measure:	MEASURE	_1				
					95% Confiden	ice Interval for
		Mean Difference			Differ	ence <sup>a</sup>
(I) Freq	(J) Freq	(I-J)	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound
1	2	035	.024	.165	088	.017
2	1	.035	.024	.165	017	.088

Based on estimated marginal means

Measure: MEASURE\_1

		Mean Difference (I-			95% Confiden Differ	
(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1	2	070*	.020	.004	113	026
2	1	.070*	.020	.004	.026	.113

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AD: 20/80 HZ TORQUE RATIO ANALYSIS

#### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Muscle	Sphericity Assumed	.036	2	.018	3.187	.059	.210	6.375	.555
	Greenhouse-Geisser	.036	1.520	.024	3.187	.076	.210	4.845	.475
	Huynh-Feldt	.036	1.840	.019	3.187	.064	.210	5.865	.529
	Lower-bound	.036	1.000	.036	3.187	.099	.210	3.187	.376
Muscle * Group	Sphericity Assumed	.007	2	.004	.649	.532	.051	1.297	.146
	Greenhouse-Geisser	.007	1.520	.005	.649	.494	.051	.986	.132
	Huynh-Feldt	.007	1.840	.004	.649	.520	.051	1.194	.141
	Lower-bound	.007	1.000	.007	.649	.436	.051	.649	.115
Error(Muscle)	Sphericity Assumed	.135	24	.006					
	Greenhouse-Geisser	.135	18.243	.007					
	Huynh-Feldt	.135	22.080	.006					
	Lower-bound	.135	12.000	.011					
Time	Sphericity Assumed	.122	2	.061	19.669	.000	.621	39.338	1.000
	Greenhouse-Geisser	.122	1.602	.076	19.669	.000	.621	31.518	.999
	Huynh-Feldt	.122	1.965	.062	19.669	.000	.621	38.654	1.000
	Lower-bound	.122	1.000	.122	19.669	.001	.621	19.669	.982
Time * Group	Sphericity Assumed	.032	2	.016	5.162	.014	.301	10.324	.775
	Greenhouse-Geisser	.032	1.602	.020	5.162	.021	.301	8.272	.702
	Huynh-Feldt	.032	1.965	.016	5.162	.014	.301	10.145	.769
	Lower-bound	.032	1.000	.032	5.162	.042	.301	5.162	.551
Error(Time)	Sphericity Assumed	.074	24	.003					
	Greenhouse-Geisser	.074	19.229	.004					
	Huynh-Feldt	.074	23.583	.003					
	Lower-bound	.074	12.000	.006					
Muscle * Time	Sphericity Assumed	.009	4	.002	1.583	.194	.117	6.331	.451
	Greenhouse-Geisser	.009	2.325	.004	1.583	.221	.117	3.680	.328
	Huynh-Feldt	.009	3.158	.003	1.583	.208	.117	4.998	.392
	Lower-bound	.009	1.000	.009	1.583	.232	.117	1.583	.212
Muscle * Time * Group	Sphericity Assumed	.012	4	.003	1.984	.112	.142	7.937	.553
	Greenhouse-Geisser	.012	2.325	.005	1.984	.151	.142	4.614	.403
	Huynh-Feldt	.012	3.158	.004	1.984	.130	.142	6.266	.482
	Lower-bound	.012	1.000	.012	1.984	.184	.142	1.984	.254
Error(Muscle*Time)	Sphericity Assumed	.070	48	.001					
	Greenhouse-Geisser	.070	27.901	.003					
	Huynh-Feldt	.070	37.893	.002					
	Lower-bound	.070	12.000	.006					

a. Computed using alpha = .05

#### Pairwise Comparisons

#### Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differe	
Time	(I) Group	(J) Group	J)	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound
1	1.00	2.00	028	.043	.533	123	.067
	2.00	1.00	.028	.043	.533	067	.123
2	1.00	2.00	.049	.045	.294	048	.146
	2.00	1.00	049	.045	.294	146	.048
3	1.00	2.00	005	.048	.918	109	.099
	2.00	1.00	.005	.048	.918	099	.109

Based on estimated marginal means

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval for Difference <sup>b</sup>		
Group	(I) Time	(J) Time	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound	
1.00	1	2	.018	.022	1.000	044	.079	
		3	029	.019	.424	081	.022	
	2	1	018	.022	1.000	079	.044	
		3	047*	.014	.017	086	008	
	3	1	.029	.019	.424	022	.081	
		2	.047*	.014	.017	.008	.086	
2.00	1	2	.094	.019	.001	.041	.148	
		3	006	.016	1.000	051	.038	
	2	1	094	.019	.001	148	041	
		3	101	.012	.000	134	067	
	3	1	.006	.016	1.000	038	.051	
		2	.101 <sup>*</sup>	.012	.000	.067	.134	

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AE: GLOBAL QUADRICEP SORENESS ANALYSIS

#### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Time	Sphericity Assumed	827.740	2	413.870	8.145	.002	.385	16.290	.936
	Greenhouse-Geisser	827.740	1.319	627.560	8.145	.007	.385	10.743	.836
	Huynh-Feldt	827.740	1.523	543.660	8.145	.005	.385	12.401	.875
	Lower-bound	827.740	1.000	827.740	8.145	.014	.385	8.145	.751
Time * Group	Sphericity Assumed	720.987	2	360.493	7.095	.003	.353	14.189	.899
	Greenhouse-Geisser	720.987	1.319	546.624	7.095	.011	.353	9.358	.782
	Huynh-Feldt	720.987	1.523	473.544	7.095	.008	.353	10.802	.826
	Lower-bound	720.987	1.000	720.987	7.095	.020	.353	7.095	.693
Error(Time)	Sphericity Assumed	1321.128	26	50.813					
	Greenhouse-Geisser	1321.128	17.147	77.048					
	Huynh-Feldt	1321.128	19.793	66.748					
	Lower-bound	1321.128	13.000	101.625					
Leg	Sphericity Assumed	17.580	1	17.580	.532	.479	.039	.532	.104
	Greenhouse-Geisser	17.580	1.000	17.580	.532	.479	.039	.532	.104
	Huynh-Feldt	17.580	1.000	17.580	.532	.479	.039	.532	.104
	Lower-bound	17.580	1.000	17.580	.532	.479	.039	.532	.104
Leg * Group	Sphericity Assumed	29.552	1	29.552	.895	.361	.064	.895	.142
	Greenhouse-Geisser	29.552	1.000	29.552	.895	.361	.064	.895	.142
	Huynh-Feldt	29.552	1.000	29.552	.895	.361	.064	.895	.142
	Lower-bound	29.552	1.000	29.552	.895	.361	.064	.895	.142
Error(Leg)	Sphericity Assumed	429.420	13	33.032					
	Greenhouse-Geisser	429.420	13.000	33.032					
	Huynh-Feldt	429.420	13.000	33.032					
	Lower-bound	429.420	13.000	33.032					
Time * Leg	Sphericity Assumed	5.265	2	2.633	.083	.920	.006	.166	.061
	Greenhouse-Geisser	5.265	1.094	4.813	.083	.799	.006	.091	.059
	Huynh-Feldt	5.265	1.210	4.350	.083	.823	.006	.101	.059
	Lower-bound	5.265	1.000	5.265	.083	.778	.006	.083	.058
Time * Leg * Group	Sphericity Assumed	5.075	2	2.538	.080	.923	.006	.160	.061
	Greenhouse-Geisser	5.075	1.094	4.639	.080	.803	.006	.088	.058
	Huynh-Feldt	5.075	1.210	4.194	.080	.827	.006	.097	.059
	Lower-bound	5.075	1.000	5.075	.080	.781	.006	.080	.058
Error(Time*Leg)	Sphericity Assumed	822.728	26	31.643					
	Greenhouse-Geisser	822.728	14.222	57.851					
	Huynh-Feldt	822.728	15.733	52.292					
	Lower-bound	822.728	13.000	63.287					

a. Computed using alpha = .05

Measure:	MEASURE_1
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			Mean Difference (I			95% Confidence Interval for Difference <sup>b</sup>	
Time	(I) Group	(I) Group (J) Group	Difference (I- J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1	1.00	2.00	198	.308	.532	863	.467
	2.00	1.00	.198	.308	.532	467	.863
2	1.00	2.00	-7.397	4.523	.126	-17.170	2.375
	2.00	1.00	7.397	4.523	.126	-2.375	17.170
3	1.00	2.00	-14.092	1.942	.000	-18.288	-9.895
	2.00	1.00	14.092	1.942	.000	9.895	18.288

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

### Pairwise Comparisons

			Mean Difference (I-			95% Confidence Interval for Difference <sup>b</sup>	
Group	(I) Time	(J) Time	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1.00	1	2	988	3.136	1.000	-9.600	7.624
		3	425	1.429	1.000	-4.350	3.500
	2	1	.988	3.136	1.000	-7.624	9.600
		3	.563	3.146	1.000	-8.076	9.202
	3	1	.425	1.429	1.000	-3.500	4.350
		2	563	3.146	1.000	-9.202	8.076
2.00	1	2	-8.187*	2.934	.046	-16.243	132
		3	-14.319	1.337	.000	-17.990	-10.647
	2	1	8.188	2.934	.046	.132	16.243
		3	-6.131	2.943	.173	-14.212	1.950
	3	1	14.319	1.337	.000	10.647	17.990
		2	6.131	2.943	.173	-1.950	14.212

Measure: MEASURE\_1

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AF: KNEE PAIN ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Time	Sphericity Assumed	141.794	2	70.897	.995	.383	.071	1.989	.204
	Greenhouse-Geisser	141.794	1.328	106.746	.995	.357	.071	1.321	.170
	Huynh-Feldt	141.794	1.536	92.328	.995	.367	.071	1.528	.181
	Lower-bound	141.794	1.000	141.794	.995	.337	.071	.995	.152
Time * Group	Sphericity Assumed	41.253	2	20.626	.289	.751	.022	.579	.091
	Greenhouse-Geisser	41.253	1.328	31.056	.289	.662	.022	.384	.083
	Huynh-Feldt	41.253	1.536	26.861	.289	.694	.022	.444	.086
	Lower-bound	41.253	1.000	41.253	.289	.600	.022	.289	.079
Error(Time)	Sphericity Assumed	1853.164	26	71.276					
	Greenhouse-Geisser	1853.164	17.268	107.316					
	Huynh-Feldt	1853.164	19.965	92.820					
	Lower-bound	1853.164	13.000	142.551					
Leg	Sphericity Assumed	40.058	1	40.058	1.593	.229	.109	1.593	.216
	Greenhouse-Geisser	40.058	1.000	40.058	1.593	.229	.109	1.593	.216
	Huynh-Feldt	40.058	1.000	40.058	1.593	.229	.109	1.593	.216
	Lower-bound	40.058	1.000	40.058	1.593	.229	.109	1.593	.216
Leg * Group	Sphericity Assumed	.068	1	.068	.003	.959	.000	.003	.050
	Greenhouse-Geisser	.068	1.000	.068	.003	.959	.000	.003	.050
	Huynh-Feldt	.068	1.000	.068	.003	.959	.000	.003	.050
	Lower-bound	.068	1.000	.068	.003	.959	.000	.003	.050
Error(Leg)	Sphericity Assumed	326.806	13	25.139					
	Greenhouse-Geisser	326.806	13.000	25.139					
	Huynh-Feldt	326.806	13.000	25.139					
	Lower-bound	326.806	13.000	25.139					
Time * Leg	Sphericity Assumed	33.206	2	16.603	.915	.413	.066	1.830	.191
	Greenhouse-Geisser	33.206	1.092	30.417	.915	.364	.066	.999	.149
	Huynh-Feldt	33.206	1.207	27.507	.915	.373	.066	1.104	.154
	Lower-bound	33.206	1.000	33.206	.915	.356	.066	.915	.144
Time * Leg * Group	Sphericity Assumed	53.538	2	26.769	1.475	.247	.102	2.950	.286
	Greenhouse-Geisser	53.538	1.092	49.041	1.475	.248	.102	1.610	.211
	Huynh-Feldt	53.538	1.207	44.350	1.475	.249	.102	1.781	.222
	Lower-bound	53.538	1.000	53.538	1.475	.246	.102	1.475	.203
Error(Time*Leg)	Sphericity Assumed	471.876	26	18.149					
/	Greenhouse-Geisser	471.876	14.192	33.249					
	Huynh-Feldt	471.876	15.693	30.069					
	Lower-bound	471.876	13.000	36.298					

a. Computed using alpha = .05

### Tests of Between-Subjects Effects

### Measure: MEASURE\_1 Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Intercept	398.535	1	398.535	4.581	.052	.261	4.581	.509
Group	71.368	1	71.368	.820	.382	.059	.820	.134
Error	1130.919	13	86.994					

# APPENDIX AG: INDIVIDUAL QUADRICEP MUSCLE SORENESS

Measure: MEASURE_1									
Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Time	Sphericity Assumed	604.399	2	302.199	19.938	.000	.605	39.877	1.000
	Greenhouse-Geisser	604.399	1.246	485.147	19.938	.000	.605	24.839	.995
	Huynh-Feldt Lower-bound	604.399	1.420	425.731	19.938	.000	.605	28.306	.998
Time t Oreun		604.399	1.000	604.399	19.938	.001	.605	19.938	.984
Time * Group	Sphericity Assumed Greenhouse-Geisser	662.422 662.422	2 1.246	331.211 531.722	21.852 21.852	.000	.627	43.705 27.224	1.000
	Huynh-Feldt	662.422	1.420	466.603	21.852	.000	.627	31.023	.997
	Lower-bound	662.422	1.420	662.422	21.852	.000	.627	21.852	.999
Error(Time)	Sphericity Assumed	394.075	26	15.157	21.032	.000	.027	21.032	.001
Litor(Time)	Greenhouse-Geisser	394.075	16.195	24.332					
	Huynh-Feldt	394.075	18.456	21.352					
	Lower-bound	394.075	13.000	30.313					
Lea	Sphericity Assumed	5.673	1	5.673	1.108	.312	.079	1.108	.164
	Greenhouse-Geisser	5.673	1.000	5.673	1.108	.312	.079	1.108	.164
	Huynh-Feldt	5.673	1.000	5.673	1.108	.312	.079	1.108	.164
	Lower-bound	5.673	1.000	5.673	1.108	.312	.079	1.108	.164
Leg * Group	Sphericity Assumed	3.354	1	3.354	.655	.433	.048	.655	.117
	Greenhouse-Geisser	3.354	1.000	3.354	.655	.433	.048	.655	.117
	Huynh-Feldt	3.354	1.000	3.354	.655	.433	.048	.655	.117
	Lower-bound	3.354	1.000	3.354	.655	.433	.048	.655	.117
Error(Leg)	Sphericity Assumed	66.540	13	5.118					
	Greenhouse-Geisser	66,540	13.000	5,118					
	Huynh-Feldt	66.540	13.000	5.118					
	Lower-bound	66.540	13.000	5.118					
Muscle	Sphericity Assumed	24.557	2	12.278	3.503	.045	.212	7.005	.602
	Greenhouse-Geisser	24.557	1.458	16.844	3.503	.063	.212	5.107	.506
	Huynh-Feldt	24.557	1.721	14.266	3.503	.054	.212	6.030	.555
	Lower-bound	24.557	1.000	24.557	3.503	.084	.212	3.503	.410
Muscle * Group	Sphericity Assumed	26.671	2	13.336	3.804	.036	.226	7.609	.640
	Greenhouse-Geisser	26.671	1.458	18.294	3.804	.053	.226	5.546	.540
	Huynh-Feldt	26.671	1.721	15.494	3.804	.043	.226	6.549	.591
	Lower-bound	26.671	1.000	26.671	3.804	.073	.226	3.804	.439
Error(Muscle)	Sphericity Assumed	91.140	26	3.505					
	Greenhouse-Geisser	91.140	18.953	4.809					
	Huynh-Feldt	91.140	22.378	4.073					
	Lower-bound	91.140	13.000	7.011					
Time * Leg	Sphericity Assumed	13.687	2	6.843	1.060	.361	.075	2.120	.215
	Greenhouse-Geisser	13.687	1.193	11.473	1.060	.333	.075	1.264	.171
	Huynh-Feldt	13.687	1.346	10.167	1.060	.340	.075	1.427	.180
	Lower-bound	13.687	1.000	13.687	1.060	.322	.075	1.060	.159
Time * Leg * Group	Sphericity Assumed	29.239	2	14.619	2.264	.124	.148	4.528	.418
	Greenhouse-Geisser	29.239	1.193	24.509	2.264	.150	.148	2.701	.314
	Huynh-Feldt	29.239	1.346	21.719	2.264	.145	.148	3.048	.335
	Lower-bound	29.239	1.000	29.239	2.264	.156	.148	2.264	.286
Error(Time*Leg)	Sphericity Assumed	167.897	26	6.458					
	Greenhouse-Geisser	167.897	15.509	10.826					
	Huynh-Feldt	167.897	17.501	9.594					
	Lower-bound	167.897	13.000	12.915					
Time * Muscle	Sphericity Assumed	88.240	4	22.060	7.297	.000	.360	29.189	.993
	Greenhouse-Geisser	88.240	2.319	38.044	7.297	.002	.360	16.926	.937
	Huynh-Feldt	88.240	3.070	28.741	7.297	.000	.360	22.404	.976
	Lower-bound	88.240	1.000	88.240	7.297	.018	.360	7.297	.705
Time * Muscle * Group	Sphericity Assumed	56.105	4	14.026	4.640	.003	.263	18.559	.928
	Greenhouse-Geisser	56.105	2.319	24.189	4.640	.014	.263	10.762	.781
	Huynh-Feldt	56.105	3.070	18.274	4.640	.007	.263	14.245	.865
	Lower-bound	56.105	1.000	56.105	4.640	.051	.263	4.640	.514
Error(Time*Muscle)	Sphericity Assumed	157.196	52	3.023					
	Greenhouse-Geisser	157.196	30.152	5.213					
	Huynh-Feldt	157.196	39.912	3.939					
	Lower-bound	157.196	13.000	12.092					
Leg * Muscle	Sphericity Assumed	8.719	2	4.360	1.853	.177	.125	3.705	.350
	Greenhouse-Geisser	8.719	1.824	4.781	1.853	.181	.125	3.379	.333
	Huynh-Feldt	8.719	2.000	4.360	1.853	.177	.125	3.705	.350
	Lower-bound	8.719	1.000	8.719	1.853	.197	.125	1.853	.243
Leg * Muscle * Group	Sphericity Assumed	15.634	2	7.817	3.322	.052	.204	6.644	.577
	Greenhouse-Geisser	15.634	1.824	8.573	3.322	.057	.204	6.058	.549
	Huynh-Feldt	15.634	2.000	7.817	3.322	.052	.204	6.644	.577
	Lower-bound	15.634	1.000	15.634	3.322	.091	.204	3.322	.393
Error(Leg*Muscle)	Sphericity Assumed	61.186	26	2.353					
	Greenhouse-Geisser	61.186	23.709	2.581					
	Huynh-Feldt	61.186	26.000	2.353					
	Lower-bound	61.186	13.000	4.707					
Time * Leg * Muscle	Sphericity Assumed	11.481	4	2.870	1.260	.298	.088	5.041	.366
	Greenhouse-Geisser	11.481	2.491	4.609	1.260	.302	.088	3.139	.280
	Huynh-Feldt	11.481	3.366	3.411	1.260	.300	.088	4.241	.331
	Lower-bound	11.481	1.000	11.481	1.260	.282	.088	1.260	.180
Time * Leg * Muscle *	Sphericity Assumed	12.671	4	3.168	1.391	.250	.097	5.563	.403
Group	Greenhouse-Geisser	12.671	2.491	5.086	1.391	.264	.097	3.465	.306
	Huynh-Feldt	12.671	3.366	3.765	1.391	.257	.097	4.681	.364
	Lower-bound	12.671	1.000	12.671	1.391	.259	.097	1.391	.194
Error(Time*Leg*Muscle)	Sphericity Assumed	118.443	52	2.278					
( 10g massis)	Greenhouse-Geisser	118.443	32.386	3.657					
	Huynh-Feldt	118.443	43.754	2.707					
	Lower-bound	118.443	13.000	9.111					

#### Tests of Within-Subjects Effects

Measure: MEASURE\_1

				Mean Difference (I-			95% Confiden Differe	
Time	Muscle	(I) Group	(J) Group	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1	1	1.00	2.00	.087	.159	.592	255	.430
		2.00	1.00	087	.159	.592	430	.255
	2	1.00	2.00	194	.190	.325	605	.216
		2.00	1.00	.194	.190	.325	216	.605
	3	1.00	2.00	246	.132	.086	532	.040
		2.00	1.00	.246	.132	.086	040	.532
2	1	1.00	2.00	-1.253	1.256	.337	-3.966	1.461
		2.00	1.00	1.253	1.256	.337	-1.461	3.966
	2	1.00	2.00	.260	.650	.695	-1.143	1.663
		2.00	1.00	260	.650	.695	-1.663	1.143
	3	1.00	2.00	320	.470	.507	-1.335	.695
		2.00	1.00	.320	.470	.507	695	1.335
3	1	1.00	2.00	-5.658	2.021	.015	-10.024	-1.292
		2.00	1.00	5.658	2.021	.015	1.292	10.024
	2	1.00	2.00	-5.643	1.292	.001	-8.435	-2.851
		2.00	1.00	5.643	1.292	.001	2.851	8.435
	3	1.00	2.00	-9.496	1.050	.000	-11.765	-7.226
		2.00	1.00	9.496	1.050	.000	7.226	11.765

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

Measure: MEASURE\_1

		-		Mean			95% Confiden Differ	ce Interval for
				Difference (I-		e: h		
Group	Muscle	(I) Time	(J) Time	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1.00	1	1	2	911	.835	.886	-3.204	1.383
			3	.113	1.479	1.000	-3.948	4.174
		2	1	.911	.835	.886	-1.383	3.204
			3	1.024	1.614	1.000	-3.407	5.455
		3	1	113	1.479	1.000	-4.174	3.948
			2	-1.024	1.614	1.000	-5.455	3.407
	2	1	2	759	.451	.350	-1.999	.481
			3	179	.922	1.000	-2.712	2.355
		2	1	.759	.451	.350	481	1.999
			3	.580	1.054	1.000	-2.314	3.475
		3	1	.179	.922	1.000	-2.355	2.712
			2	580	1.054	1.000	-3.475	2.314
	3	1	2	527	.324	.384	-1.417	.363
			3	308	.756	1.000	-2.384	1.768
		2	1	.527	.324	.384	363	1.417
			3	.219	.869	1.000	-2.168	2.606
	3	1	.308	.756	1.000	-1.768	2.384	
		2	219	.869	1.000	-2.606	2.168	
2.00	1	1	2	-2.250	.781	.039	-4.396	105
			3	-5.632	1.383	.004	-9.431	-1.833
		2	1	2.250	.781	.039	.105	4.396
			3	-3.381	1.509	.130	-7.526	.763
		3	1	5.632	1.383	.004	1.833	9.431
			2	3.381	1.509	.130	763	7.526
	2	1	2	305	.422	1.000	-1.464	.855
			3	-5.627*	.863	.000	-7.997	-3.258
		2	1	.305	.422	1.000	855	1.464
			3	-5.323	.986	.000	-8.030	-2.615
		3	1	5.627	.863	.000	3.258	7.997
			2	5.323	.986	.000	2.615	8.030
	3	1	2	602	.303	.206	-1.434	.231
			3	-9.558	.707	.000	-11.500	-7.616
		2	1	.602	.303	.206	231	1.434
			3	-8.956	.813	.000	-11.189	-6.724
		3	1	9.558	.707	.000	7.616	11.500
			2	8.956	.813	.000	6.724	11.189

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

Measure: MEASURE\_1

				Mean Difference (l-			95% Confiden Differ	ce Interval for ence <sup>b</sup>
Group	Time	(I) Muscle	(J) Muscle	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1.00	1	1	2	.188	.115	.377	127	.502
			3	.161	.103	.428	122	.444
		2	1	187	.115	.377	502	.127
			3	027	.134	1.000	393	.340
		3	1	161	.103	.428	444	.122
			2	.027	.134	1.000	340	.393
	2	1	2	.339	.847	1.000	-1.988	2.666
			3	.545	.888	1.000	-1.894	2.983
		2	1	339	.847	1.000	-2.666	1.988
			3	.205	.222	1.000	404	.814
		3	1	545	.888	1.000	-2.983	1.894
			2	205	.222	1.000	814	.404
	3	1	2	104	.876	1.000	-2.510	2.302
			3	260	1.082	1.000	-3.233	2.712
		2	1	.104	.876	1.000	-2.302	2.510
			3	156	.746	1.000	-2.205	1.893
		3	1	.260	1.082	1.000	-2.712	3.233
			2	.156	.746	1.000	-1.893	2.205
2.00	1	1	2	094	.107	1.000	388	.201
			3	172	.096	.293	436	.093
		2	1	.094	.107	1.000	201	.388
			3	078	.125	1.000	421	.265
		3	1	.172	.096	.293	093	.436
			2	.078	.125	1.000	265	.421
	2	1	2	1.852	.793	.108	325	4.029
			3	1.477	.831	.296	804	3.758
		2	1	-1.852	.793	.108	-4.029	.325
			3	375	.207	.281	945	.195
		3	1	-1.477	.831	.296	-3.758	.804
			2	.375	.207	.281	195	.945
	3	1	2	089	.820	1.000	-2.340	2.161
			3	-4.098	1.013	.004	-6.879	-1.318
		2	1	.089	.820	1.000	-2.161	2.340
			3	-4.009	.698	.000	-5.925	-2.092
		3	1	4.098	1.013	.004	1.318	6.879
			2	4.009	.698	.000	2.092	5.925

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AH: KNEE EXTENSOR MVC EMG RMS ANALYSIS

#### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sia.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Time	Sphericity Assumed	178.121	2	89.061	.058	.944	.004	.116	.05
	Greenhouse-Geisser	178.121	1.614	110.393	.058	.912	.004	.093	.05
	Huynh-Feldt	178.121	1.950	91.348	.058	.941	.004	.113	.05
	Lower-bound	178.121	1.000	178.121	.058	.814	.004	.058	.05
ime * Group	Sphericity Assumed	537.615	2	268.808	.175	.841	.013	.350	.07
inter ereap	Greenhouse-Geisser	537.615	1.614	333.195	.175	.795	.013	.282	.07
	Huynh-Feldt	537.615	1.950	275.711	.175	.835	.013	.341	.07
Irror(Time)  Auscle Auscle * Group  Fror(Muscle)  Ingle Fror(Angle)  Time * Muscle * Group  Fror(Time*Muscle)  Time * Angle Time * Angle * Group	Lower-bound	537.615	1.000	537.615	.175	.683	.013	.175	.06
rror(Time)	Sphericity Assumed	39987.136	26	1537.967					
	Greenhouse-Geisser	39987.136	20.976	1906.354					
	Huynh-Feldt	39987.136	25.349	1577.465					
	Lower-bound	39987.136	13.000	3075.934					
4		242270.621	2		15.495	.000	.544	30.991	.99
nuscie	Sphericity Assumed	242270.621	1.297	121135.311 186807.436	15.495	.000	.544	20.096	.95
	Greenhouse-Geisser								
	Huynh-Feldt	242270.621	1.491	162449.737 242270.621	15.495	.000	.544	23.109	.99
	Lower-bound	242270.621	1.000		15.495	.002	.544	15.495	.95
iuscie - Group	Sphericity Assumed	47122.371	2	23561.186	3.014	.066	.188	6.028	.5:
	Greenhouse-Geisser	47122.371	1.297	36334.613	3.014	.093	.188	3.909	.4
	Huynh-Feldt	47122.371	1.491	31596.967	3.014	.085	.188	4.495	.45
	Lower-bound	47122.371	1.000	47122.371	3.014	.106	.188	3.014	.36
rror(Muscle)	Sphericity Assumed	203256.615	26	7817.562					
	Greenhouse-Geisser	203256.615	16.860	12055.764					
	Huynh-Feldt	203256.615	19.388	10483.821					
	Lower-bound	203256.615	13.000	15635.124					
ngle	Sphericity Assumed	28828.878	2	14414.439	7.854	.002	.377	15.709	.9:
	Greenhouse-Geisser	28828.878	1.884	15300.025	7.854	.003	.377	14.799	.9
	Huynh-Feldt	28828.878	2.000	14414.439	7.854	.002	.377	15.709	.9
	Lower-bound	28828.878	1.000	28828.878	7.854	.015	.377	7.854	.7
ngle * Group	Sphericity Assumed	367.900	2	183.950	.100	.905	.008	.200	.0
	Greenhouse-Geisser	367.900	1.884	195.251	.100	.895	.008	.189	.0
	Huynh-Feldt	367.900	2.000	183.950	.100	.905	.008	.200	.0
	Lower-bound	367.900	1.000	367.900	.100	.757	.008	.100	.0
rror(Apgle)	Sphericity Assumed	47715.990	26	1835.230			.000		.0
(Aligie)		47715.990	24,495	1947.982					
	Greenhouse-Geisser		26.000						
	Huynh-Feldt	47715.990		1835.230					
	Lower-bound	47715.990	13.000	3670.461					
ime * Muscle	Sphericity Assumed	3762.388	4	940.597	1.404	.246	.097	5.616	.4
	Greenhouse-Geisser	3762.388	2.234	1684.235	1.404	.262	.097	3.136	.2
	Huynh-Feldt	3762.388	2.927	1285.576	1.404	.257	.097	4.109	.3
	Lower-bound	3762.388	1.000	3762.388	1.404	.257	.097	1.404	.13
îme * Muscle * Group	Sphericity Assumed	1188.991	4	297.248	.444	.776	.033	1.775	.1-
	Greenhouse-Geisser	1188.991	2.234	532.253	.444	.667	.033	.991	.1
	Huynh-Feldt	1188.991	2.927	406.268	.444	.718	.033	1.299	.1
	Lower-bound	1188.991	1.000	1188.991	.444	.517	.033	.444	.0
rror(Time*Muscle)	Sphericity Assumed	34835.714	52	669.918					
	Greenhouse-Geisser	34835.714	29.041	1199.556					
	Huynh-Feldt	34835,714	38.046	915.621					
	Lower-bound	34835,714	13.000	2679.670					
ime * Angle	Sphericity Assumed	4212,388	4	1053.097	3.847	.008	.228	15,387	.8
into rangio	Greenhouse-Geisser	4212,388	1.796	2345.660	3,847	.040	.228	6,908	.6
	Huynh-Feldt	4212.388	2.226	1892,598	3,847	.029	.228	8.562	.6
		4212.388		4212.388	3.847	.072			.4
	Lower-bound		1.000				.228	3.847	
The "Angle " Group	Sphericity Assumed	3457.717	4	864.429	3.158	.021	.195	12.631	.7
	Greenhouse-Geisser	3457.717	1.796	1925.423	3.158	.066	.195	5.671	.5
	Huynh-Feldt	3457.717	2.226	1553.529	3.158	.053	.195	7.028	.5
	Lower-bound	3457.717	1.000	3457.717	3.158	.099	.195	3.158	.3
rror(Time*Angle)	Sphericity Assumed	14235.395	52	273.758					
	Greenhouse-Geisser	14235.395	23.346	609.766					
	Huynh-Feldt	14235.395	28.934	491.990					
	Lower-bound	14235.395	13.000	1095.030					
uscle * Angle	Sphericity Assumed	9493.655	4	2373.414	3.169	.021	.196	12.675	.7
	Greenhouse-Geisser	9493.655	2.136	4445.312	3.169	.055	.196	6.767	.5
	Huynh-Feldt	9493.655	2.765	3434.091	3.169	.039	.196	8.760	.6
	Lower-bound	9493.655	1.000	9493.655	3.169	.098	.196	3.169	.3
luscle * Angle * Group	Sphericity Assumed	3417.573	4	854.393	1.141	.348	.081	4.563	.3
	Greenhouse-Geisser	3417.573	2.136	1600.246	1.141	.337	.081	2.436	.2
	Huynh-Feldt	3417.573	2.765	1236.221	1.141	.343	.081	3.154	.2
	Lower-bound	3417.573	1.000	3417.573	1.141	.305	.081	1.141	.1
ror(Muscle*Angle)	Sphericity Assumed	38947.644	52	748.993					
	Greenhouse-Geisser	38947.644	27.764	1402.835					
	Huvnh-Feldt	38947.644	35.939	1083 718					
	Lower-bound	38947.644	13 000	2995 973					
mo * Muncle * Americ	Sphericity Assumed	3062.012	13.000	382.752	3.195	.003	.197	25.560	.9
me * Muscle * Angle			2.048		3.195	.003		25.560	
	Greenhouse-Geisser	3062.012		1494.972			.197		.5
	Huynh-Feldt	3062.012	2.623	1167.510	3.195	.041	.197	8.379	.6
	Lower-bound	3062.012	1.000	3062.012	3.195	.097	.197	3.195	.3
me * Muscle * Angle * roup	Sphericity Assumed	830.229	8	103.779	.866	.547	.062	6.930	.3
.oup	Greenhouse-Geisser	830.229	2.048	405.344	.866	.434	.062	1.774	.1
	Huynh-Feldt	830.229	2.623	316.557	.866	.455	.062	2.272	.2
	Lower-bound	830.229	1.000	830.229	.866	.369	.062	.866	.1
rror	Sphericity Assumed	12458.896	104	119.797					
"ime*Muscle*Angle)	Greenhouse-Geisser	12458.896	26.627	467.910					
	Huynh-Feldt	12458.896	34.095	365.418					
	Lower-bound	12458.896	13.000	958.377					

Measure: MEASURE\_1

				Mean Difference (I-			95% Confiden Differ	
Time	Angle	(I) Group	(J) Group	J) J	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound
1	1	Con	Inj	-25.640	31.611	.432	-93.931	42.650
		Inj	Con	25.640	31.611	.432	-42.650	93.931
	2	Con	Inj	-6.480	24.225	.793	-58.816	45.856
		Inj	Con	6.480	24.225	.793	-45.856	58.816
	3	Con	Inj	-14.356	21.691	.520	-61.217	32.505
		Inj	Con	14.356	21.691	.520	-32.505	61.217
2	1	Con	Inj	-6.117	31.341	.848	-73.826	61.591
		Inj	Con	6.117	31.341	.848	-61.591	73.826
	2	Con	Inj	-18.869	36.479	.614	-97.676	59.939
		Inj	Con	18.869	36.479	.614	-59.939	97.676
	3	Con	Inj	-17.134	26.685	.532	-74.785	40.516
		Inj	Con	17.134	26.685	.532	-40.516	74.785
3	1	Con	Inj	-5.217	24.107	.832	-57.296	46.862
		Inj	Con	5.217	24.107	.832	-46.862	57.296
	2	Con	Inj	-8.858	27.228	.750	-67.679	49.964
		Inj	Con	8.858	27.228	.750	-49.964	67.679
	3	Con	Inj	-16.019	24.064	.517	-68.007	35.969
		Inj	Con	16.019	24.064	.517	-35.969	68.007

Based on estimated marginal means

Measure: MEASURE\_1

				Mean Difference (I-			95% Confiden Differe	
Group	Angle	(I) Time	(J) Time	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	1	2	.700	7.494	1.000	-19.878	21.279
			3	242	6.910	1.000	-19.216	18.732
		2	1	700	7.494	1.000	-21.279	19.878
			3	942	9.575	1.000	-27.234	25.349
		3	1	.242	6.910	1.000	-18.732	19.216
			2	.942	9.575	1.000	-25.349	27.234
	2	1	2	-1.144	11.885	1.000	-33.780	31.492
			3	-2.603	5.642	1.000	-18.095	12.890
		2	1	1.144	11.885	1.000	-31.492	33.780
			3	-1.459	9.879	1.000	-28.587	25.669
		3	1	2.603	5.642	1.000	-12.890	18.095
			2	1.459	9.879	1.000	-25.669	28.587
	3	1	2	100	7.624	1.000	-21.036	20.837
			3	543	6.956	1.000	-19.645	18.558
		2	1	.100	7.624	1.000	-20.837	21.036
			3	444	4.802	1.000	-13.629	12.741
		3	1	.543	6.956	1.000	-18.558	19.645
			2	.444	4.802	1.000	-12.741	13.629
Inj	1	1	2	20.223	7.010	.038	.974	39.473
			3	20.182	6.463	.024	2.433	37.930
		2	1	-20.223	7.010	.038	-39.473	974
			3	042	8.956	1.000	-24.635	24.552
		3	1	-20.182	6.463	.024	-37.930	-2.433
			2	.042	8.956	1.000	-24.552	24.635
	2	1	2	-13.532	11.118	.735	-44.061	16.996
			3	-4.980	5.278	1.000	-19.473	9.512
		2	1	13.532	11.118	.735	-16.996	44.061
			3	8.552	9.241	1.000	-16.824	33.928
		3	1	4.980	5.278	1.000	-9.512	19.473
			2	-8.552	9.241	1.000	-33.928	16.824
	3	1	2	-2.878	7.132	1.000	-22.463	16.706
			3	-2.207	6.507	1.000	-20.074	15.661
		2	1	2.878	7.132	1.000	-16.706	22.463
			3	.672	4.491	1.000	-11.662	13.005
		3	1	2.207	6.507	1.000	-15.661	20.074
			2	672	4.491	1.000	-13.005	11.662

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

Measure: MEASURE\_1

				Mean Difference (l-			95% Confiden Differ	ice Interval for ence <sup>b</sup>
Group	Time	(I) Angle	(J) Angle	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	1	2	18.079	9.774	.262	-8.759	44.917
			3	20.764	9.834	.164	-6.239	47.768
		2	1	-18.079	9.774	.262	-44.917	8.759
			3	2.686	6.735	1.000	-15.809	21.180
		3	1	-20.764	9.834	.164	-47.768	6.239
			2	-2.686	6.735	1.000	-21.180	15.809
	2	1	2	16.235	7.883	.180	-5.411	37.881
			3	19.964	8.072	.084	-2.201	42.130
		2	1	-16.235	7.883	.180	-37.881	5.411
			3	3.730	8.758	1.000	-20.320	27.779
		3	1	-19.964	8.072	.084	-42.130	2.201
			2	-3.730	8.758	1.000	-27.779	20.320
	3	1	2	15.718	10.021	.422	-11.799	43.236
			3	20.463	9.348	.142	-5.205	46.131
		2	1	-15.718	10.021	.422	-43.236	11.799
			3	4.745	7.168	1.000	-14.938	24.428
		3	1	-20.463	9.348	.142	-46.131	5.205
			2	-4.745	7.168	1.000	-24.428	14.938
Inj	1	1	2	37.239	9.142	.004	12.135	62.344
			3	32.049	9.199	.012	6.790	57.308
		2	1	-37.239	9.142	.004	-62.344	-12.135
			3	-5.190	6.300	1.000	-22.491	12.110
		3	1	-32.049	9.199	.012	-57.308	-6.790
			2	5.190	6.300	1.000	-12.110	22.491
	2	1	2	3.484	7.374	1.000	-16.764	23.732
			3	8.947	7.551	.772	-11.786	29.681
		2	1	-3.484	7.374	1.000	-23.732	16.764
			3	5.464	8.193	1.000	-17.033	27.960
		3	1	-8.947	7.551	.772	-29.681	11.786
			2	-5.464	8.193	1.000	-27.960	17.033
	3	1	2	12.077	9.374	.660	-13.663	37.818
			3	9.661	8.744	.868	-14.349	33.671
		2	1	-12.077	9.374	.660	-37.818	13.663
			3	-2.417	6.705	1.000	-20.828	15.995
		3	1	-9.661	8.744	.868	-33.671	14.349
			2	2.417	6.705	1.000	-15.995	20.828

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AI: KNEE EXTENSOR MVC EMG MF ANALYSIS

#### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Fime	Sphericity Assumed	954.664	2	477.332	2.006	.155	.134	4.013	.37
	Greenhouse-Geisser	954.664	1.846	517.034	2.006	.159	.134	3.705	.35
	Huynh-Feldt	954.664	2.000	477.332	2.006	.155	.134	4.013	.37
	Lower-bound	954.664	1.000	954.664	2.006	.180	.134	2.006	.25
ime * Group	Sphericity Assumed	1909.872	2	954.936	4.014	.030	.236	8.028	.66
	Greenhouse-Geisser	1909.872	1.846	1034.363	4.014	.034	.236	7.412	.63
	Huynh-Feldt	1909.872	2.000	954.936	4.014	.030	.236	8.028	.66
	Lower-bound	1909.872	1.000	1909.872	4.014	.066	.236	4.014	.451
rror(Time)	Sphericity Assumed	6185.248	26	237.894					
	Greenhouse-Geisser	6185.248	24.004	257.681					
	Huynh-Feldt	6185.248	26.000	237.894					
	Lower-bound	6185.248	13.000	475.788					
luscle	Sphericity Assumed	163491.417	2	81745.709	38.409	.000	.747	76.818	1.00
	Greenhouse-Geisser	163491.417	1.528	106965.605	38.409	.000	.747	58.706	1.00
	Huynh-Feldt	163491.417	1.824	89622.242	38.409	.000	.747	70.066	1.00
	Lower-bound	163491.417	1.000	163491.417	38.409	.000	.747	38.409	1.00
luscle * Group	Sphericity Assumed	20675.421	2	10337.710	4.857	.016	.272	9.714	.75
	Greenhouse-Geisser	20675.421	1.528	13527.064	4.857	.026	.272	7.424	.66:
	Huynh-Feldt	20675.421	1.824	11333.791	4.857	.019	.272	8.861	.72
	Lower-bound	20675.421	1.000	20675.421	4.857	.046	.272	4.857	.53
rror(Muscle)	Sphericity Assumed	55335.980	26	2128.307					
	Greenhouse-Geisser	55335.980	19.870	2784.925					
	Huynh-Feldt	55335.980	23.715	2333.378					
	Lower-bound	55335.980	13.000	4256.614					
ngle	Sphericity Assumed	41540,990	2	20770.495	151.003	.000	.921	302.005	1.00
ngio			1.804	23033.418	151.003	.000			
	Greenhouse-Geisser	41540.990					.921	272.335	1.00
	Huynh-Feldt	41540.990	2.000	20770.495	151.003	.000	.921	302.005	1.00
	Lower-bound	41540.990	1.000	41540.990	151.003	.000	.921	151.003	1.00
ngle * Group	Sphericity Assumed	108.114	2	54.057	.393	.679	.029	.786	.10
	Greenhouse-Geisser	108.114	1.804	59.946	.393	.658	.029	.709	.10
	Huynh-Feldt	108.114	2.000	54.057	.393	.679	.029	.786	.10
	Lower-bound	108.114	1.000	108.114	.393	.542	.029	.393	.09
rror(Angle)	Sphericity Assumed	3576.314	26	137.551					
	Greenhouse-Geisser	3576.314	23.446	152.537					
	Huynh-Feldt	3576.314	26.000	137.551					
	Lower-bound	3576.314	13.000	275.101					
Fime * Muscle	Sphericity Assumed	2685.662	4	671.416	1.935	.118	.130	7.741	.54
	Greenhouse-Geisser	2685.662	2.134	1258.218	1.935	.161	.130	4.131	.37
	Huynh-Feldt	2685.662	2.763	972.137	1.935	.145	.130	5.346	.43
	Lower-bound	2685.662	1.000	2685.662	1.935	.188	.130	1.935	.25
Fime * Muscle * Group	Sphericity Assumed	1363.857	4	340.964	.983	.425	.070	3.931	.28
	Greenhouse-Geisser	1363.857	2.134	638.960	.983	.392	.070	2.098	.20
	Huynh-Feldt	1363.857	2.763	493.679	.983	.406	.070	2.715	.23
	Lower-bound	1363.857	1.000	1363.857	.983	.340	.070	.983	.15
rror(Time*Muscle)	Sphericity Assumed	18041.171	52	346.946					
	Greenhouse-Geisser	18041.171	27.748	650.168					
	Huynh-Feldt	18041.171	35.914	502.340					
	Lower-bound	18041.171	13.000	1387.782					
ïme * Angle	Sphericity Assumed	914.153	4	228.538	6.380	.000	.329	25.520	.98
inte vingle	Greenhouse-Geisser	914.153	1.827	500.235	6.380	.007	.329	11.659	.83
	Huynh-Feldt	914.153	2.274	401.918	6.380	.004	.329	14.511	.89
	Lower-bound	914.153	1.000	914.153	6.380	.025	.329	6.380	.64
ime * Angle * Oreun			4						
ime * Angle * Group	Sphericity Assumed	138.642		34.661	.968	.433	.069	3.870	.28
	Greenhouse-Geisser	138.642	1.827	75.866	.968	.387	.069	1.768	.19
	Huynh-Feldt	138.642	2.274	60.956	.968	.401	.069	2.201	.21
	Lower-bound	138.642	1.000	138.642	.968	.343	.069	.968	.14
rror(Time*Angle)	Sphericity Assumed	1862.680	52	35.821					
	Greenhouse-Geisser	1862.680	23.757	78.406					
	Huynh-Feldt	1862.680	29.568	62.996					
	Lower-bound	1862.680	13.000	143.283					
uscle * Angle	Sphericity Assumed	10188.531	4	2547.133	21.249	.000	.620	84.994	1.00
	Greenhouse-Geisser	10188.531	2.507	4064.776	21.249	.000	.620	53.260	1.00
	Huynh-Feldt	10188.531	3.392	3003.330	21.249	.000	.620	72.084	1.00
	Lower-bound	10188.531	1.000	10188.531	21.249	.000	.620	21.249	.98
luscle * Angle * Group	Sphericity Assumed	134.201	4	33.550	.280	.890	.021	1.120	.10
	Greenhouse-Geisser	134.201	2.507	53.540	.280	.805	.021	.702	.09
	Huynh-Feldt	134.201	3.392	39.559	.280	.862	.021	.949	.10
	Lower-bound	134.201	1.000	134.201	.280	.606	.021	.280	.07
rror(Muscle*Angle)	Sphericity Assumed	6233.423	52	119.874					
	Greenhouse-Geisser	6233.423	32.585	191.297					
	Huynh-Feldt	6233.423	44.101	141.343					
	Lower-bound	6233.423	13.000	479,494					
ime * Muscle * Angle	Sphericity Assumed	687.899	8	85.987	1.479	.174	.102	11.831	.63
in a start of the	Greenhouse-Geisser	687.899	3.043	226.055	1.479	.235	.102	4.500	.05
	Huynh-Feldt	687.899	4.383	156.930	1.479	.235	.102	6.483	.30
	Huynn-Feldt		4.383	156.930			.102		
ime * Muscle * Angle *		687.899			1.479	.246		1.479	.20
ime * Muscle * Angle * iroup	Sphericity Assumed	220.629	8	27.579	.474	.872	.035	3.795	.21
	Greenhouse-Geisser	220.629	3.043	72.502	.474	.705	.035	1.443	.13
	Huynh-Feldt	220.629	4.383	50.332	.474	.771	.035	2.079	.15
	Lower-bound	220.629	1.000	220.629	.474	.503	.035	.474	.091
Fror	Sphericity Assumed	6047.009	104	58.144					
Time*Muscle*Angle)	Greenhouse-Geisser	6047.009	39.560	152.857					
	Huynh-Feldt	6047.009	56.985	106.116					
	Lower-bound	6047.009	13.000	465.155					

# APPENDIX AJ: KNEE EXTENSOR WALKING EMG RMS ANALYSIS

WEASURE. WEASORE_T									
Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
_eg	Sphericity Assumed	1.856	1	1.856	.159	.698	.014	.159	.06
	Greenhouse-Geisser	1.856	1.000	1.856	.159	.698	.014	.159	.06
	Huynh-Feldt	1.856	1.000	1.856	.159	.698	.014	.159	.06
	Lower-bound	1.856	1.000	1.856	.159	.698	.014	.159	.06
.eg * Group	Sphericity Assumed	29.955	1	29.955	2.565	.138	.189	2.565	.31
eg * Group rror(L=g) uscle uscle * Group rror(Muscle) ime me * Group rror(Time) eg * Muscle * Group rror(Leg*Muscle) eg * Time eg * Time	Greenhouse-Geisser	29.955	1.000	29.955	2.565	.138	.189	2.565	.31
	Huynh-Feldt	29.955	1.000	29.955	2.565	.138	.189	2.565	.31
	Lower-bound	29.955	1.000	29.955	2.565	.138	.189	2.565	.31
rror(Leg)	Sphericity Assumed	128.457	11	11.678					
	Greenhouse-Geisser	128.457	11.000	11.678					
	Huynh-Feldt Lower-bound	128.457	11.000	11.678					
turnela		128.457 893,184	2	11.678 446.592	52.551	.000	.827	105.102	1.00
nuscie	Sphericity Assumed Greenhouse-Geisser	893.184	1.706	523.623	52.551	.000	.827	89.640	1.00
	Huynh-Feldt	893,184	2.000	446.592	52.551	.000	.827	105.102	1.00
	Lower-bound	893.184	1.000	893.184	52.551	.000	.827	52.551	1.00
luscle * Group	Sphericity Assumed	31.627	2	15.814	1.861	.179	.145	3.722	.34
acces croup	Greenhouse-Geisser	31.627	1.706	18.541	1.861	.186	.145	3.174	.31
	Huynh-Feldt	31.627	2.000	15.814	1.861	.179	.145	3.722	.34
	Lower-bound	31.627	1.000	31.627	1.861	.200	.145	1.861	.23
rror(Muscle)	Sphericity Assumed	186.962	22	8,498					.20
	Greenhouse-Geisser	186.962	18.764	9.964					
	Huynh-Feldt	186.962	22.000	8.498					
	Lower-bound	186.962	11.000	16.997					
îme	Sphericity Assumed	32.976	2	16.488	.978	.392	.082	1.956	.19
	Greenhouse-Geisser	32.976	1.313	25.122	.978	.364	.082	1.283	.16
	Huynh-Feldt	32.976	1.555	21.206	.978	.376	.082	1.521	.17
	Lower-bound	32.976	1.000	32.976	.978	.344	.082	.978	.14
"ime * Group	Sphericity Assumed	43.675	2	21.838	1.295	.294	.105	2.590	.25
	Greenhouse-Geisser	43.675	1.313	33.273	1.295	.288	.105	1.700	.20
	Huynh-Feldt	43.675	1.555	28.087	1.295	.291	.105	2.014	.22
	Lower-bound	43.675	1.000	43.675	1.295	.279	.105	1.295	.18
rror(Time)	Sphericity Assumed	370.975	22	16.862					
	Greenhouse-Geisser	370.975	14.439	25.693					
	Huynh-Feldt	370.975	17.105	21.688					
	Lower-bound	370.975	11.000	33.725					
eg * Muscle	Sphericity Assumed	13.245	2	6.623	.677	.518	.058	1.354	.14
	Greenhouse-Geisser	13.245	1.978	6.697	.677	.517	.058	1.339	.14
	Huynh-Feldt	13.245	2.000	6.623	.677	.518	.058	1.354	.14
	Lower-bound	13.245	1.000	13.245	.677	.428	.058	.677	.11
eg * Muscle * Group	Sphericity Assumed	26.117	2	13.058	1.335	.284	.108	2.670	.25
	Greenhouse-Geisser	26.117	1.978	13.205	1.335	.284	.108	2.640	.25
	Huynh-Feldt	26.117	2.000	13.058	1.335	.284	.108	2.670	.25
	Lower-bound	26.117	1.000	26.117	1.335	.272	.108	1.335	.18
rror(Leg*Muscle)	Sphericity Assumed	215.225	22	9.783					
	Greenhouse-Geisser	215.225	21.756	9.893					
	Huynh-Feldt	215.225	22.000	9.783					
	Lower-bound	215.225	11.000	19.566					
.eg * Time	Sphericity Assumed	.861	2	.431	.208	.814	.019	.416	.07
	Greenhouse-Geisser	.861	1.325	.650	.208	.723	.019	.276	.07
	Huynh-Feldt	.861	1.574	.547	.208	.762	.019	.327	.07
	Lower-bound	.861	1.000	.861	.208	.657	.019	.208	.07
eg * Time * Group	Sphericity Assumed	1.540	2	.770	.372	.694	.033	.743	.10
	Greenhouse-Geisser	1.540	1.325	1.162	.372	.611	.033	.493	.09
	Huynh-Feldt	1.540	1.574	.978	.372	.645	.033	.585	.09
	Lower-bound	1.540	1.000	1.540	.372	.554	.033	.372	.08
rror(Leg*Time)	Sphericity Assumed	45.569	22	2.071					
	Greenhouse-Geisser	45.569	14.579	3.126					
	Huynh-Feldt	45.569	17.317	2.631					
to an a fact the Third of the second s	Lower-bound	45.569	11.000	4.143					
iuscle * Time	Sphericity Assumed	9.774	4	2.443	1.067	.384	.088	4.270	.30
	Greenhouse-Geisser	9.774	2.261	4.322	1.067	.366	.088	2.414	.22
	Huynh-Feldt	9.774	3.135	3.118	1.067	.378	.088	3.346	.26
luncio * Time * C	Lower-bound	9.774	1.000	9.774	1.067	.324	.088	1.067	.15
uscle - Time - Group	Sphericity Assumed	10.204	4	2.551	1.114	.362	.092	4.458	.32
	Greenhouse-Geisser Huynh-Feldt	10.204	2.261 3.135	4.513 3.255	1.114	.350	.092	2.520 3.494	.23
			3.135	3.255		.358	.092		.27
irror(Muscle*Time)	Lower-bound Sphericity Assumed	10.204 100.718	1.000	2.289	1.114	.314	.092	1.114	.16
	Greenhouse-Geisser	100.718	24.872	4.049					
	Huynh-Feldt	100.718	34.483	2.921					
	Lower-bound	100.718	11.000	9.156					
ea * Muscle * Time	Sphericity Assumed	7.404	4	1.851	.798	.533	.068	3.191	.23
and the second s	Greenhouse-Geisser	7.404	1.419	5.218	.798	.428	.068	1.132	.14
	Huvnh-Feldt	7.404	1.717	4.313	.798	.420	.068	1.369	.15
	Lower-bound	7.404	1.000	7.404	.798	.391	.068	.798	.10
eg * Muscle * Time *	Sphericity Assumed	9.175	4	2.294	.988	.424	.082	3.954	.28
Froup	Greenhouse-Geisser	9.175	1.419	6.466	.988	.424	.082	1.403	.26
	Huynh-Feldt	9.175	1.717	5.345	.988	.379	.082	1.697	.18
	Lower-bound	9.175	1.000	9,175	.988	.341	.082	.988	.14
Error(Leg*Muscle*Time)	Sphericity Assumed	102.102	44	2.320			.002	.000	
(	Greenhouse-Geisser	102.102	15.608	6.542					
	Huynh-Feldt	102.102	18.882	5.407					

#### Tests of Within-Subjects Effects

# APPENDIX AK: TIBIALIS ANTERIOR WALKING EMG RMS ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Leg	Sphericity Assumed	3.799	1	3.799	.088	.771	.007	.088	.059
	Greenhouse-Geisser	3.799	1.000	3.799	.088	.771	.007	.088	.059
	Huynh-Feldt	3.799	1.000	3.799	.088	.771	.007	.088	.059
	Lower-bound	3.799	1.000	3.799	.088	.771	.007	.088	.059
Leg * Group	Sphericity Assumed	.778	1	.778	.018	.895	.001	.018	.052
	Greenhouse-Geisser	.778	1.000	.778	.018	.895	.001	.018	.052
	Huynh-Feldt	.778	1.000	.778	.018	.895	.001	.018	.052
	Lower-bound	.778	1.000	.778	.018	.895	.001	.018	.052
Error(Leg)	Sphericity Assumed	559.709	13	43.055					
	Greenhouse-Geisser	559.709	13.000	43.055					
	Huynh-Feldt	559.709	13.000	43.055					
	Lower-bound	559.709	13.000	43.055					
Time	Sphericity Assumed	109.893	2	54.947	7.757	.002	.374	15.514	.924
	Greenhouse-Geisser	109.893	1.669	65.861	7.757	.004	.374	12.943	.883
	Huynh-Feldt	109.893	2.000	54.947	7.757	.002	.374	15.514	.924
	Lower-bound	109.893	1.000	109.893	7.757	.015	.374	7.757	.731
Time * Group	Sphericity Assumed	133.483	2	66.742	9.422	.001	.420	18.845	.964
	Greenhouse-Geisser	133.483	1.669	79.999	9.422	.002	.420	15.722	.936
	Huynh-Feldt	133.483	2.000	66.742	9.422	.001	.420	18.845	.964
	Lower-bound	133.483	1.000	133.483	9.422	.009	.420	9.422	.810
Error(Time)	Sphericity Assumed	184.168	26	7.083					
	Greenhouse-Geisser	184.168	21.691	8.490					
	Huynh-Feldt	184.168	26.000	7.083					
	Lower-bound	184.168	13.000	14.167					
Leg * Time	Sphericity Assumed	12.874	2	6.437	.339	.715	.025	.679	.098
	Greenhouse-Geisser	12.874	1.433	8.985	.339	.646	.025	.486	.091
	Huynh-Feldt	12.874	1.685	7.640	.339	.680	.025	.572	.094
	Lower-bound	12.874	1.000	12.874	.339	.570	.025	.339	.084
Leg * Time * Group	Sphericity Assumed	17.582	2	8.791	.463	.634	.034	.927	.117
	Greenhouse-Geisser	17.582	1.433	12.271	.463	.573	.034	.664	.106
	Huynh-Feldt	17.582	1.685	10.434	.463	.603	.034	.781	.111
	Lower-bound	17.582	1.000	17.582	.463	.508	.034	.463	.097
Error(Leg*Time)	Sphericity Assumed	493.316	26	18.974					
	Greenhouse-Geisser	493.316	18.626	26.485					
	Huynh-Feldt	493.316	21.906	22.519					
	Lower-bound	493.316	13.000	37.947					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval for Difference <sup>a</sup>			
Time	(I) Group	(J) Group	J) J	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound		
1	Con	Inj	6.242	3.459	.094	-1.231	13.714		
	Inj	Con	-6.242	3.459	.094	-13.714	1.231		
2	Con	Inj	.274	3.807	.944	-7.951	8.498		
	Inj	Con	274	3.807	.944	-8.498	7.951		
3	Con	Inj	3.580	3.336	.303	-3.626	10.786		
	Inj	Con	-3.580	3.336	.303	-10.786	3.626		

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

### Pairwise Comparisons

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval fo Difference <sup>b</sup>				
Group	(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound			
Con	1	2	.406	1.209	1.000	-2.914	3.725			
		3	.772	.869	1.000	-1.614	3.159			
	2	1	406	1.209	1.000	-3.725	2.914			
		3	.366	.905	1.000	-2.118	2.851			
	3	1	772	.869	1.000	-3.159	1.614			
		2	366	.905	1.000	-2.851	2.118			
Inj	1	2	-5.562	1.131	.001	-8.667	-2.457			
		3	-1.889	.813	.111	-4.122	.343			
	2	1	5.562	1.131	.001	2.457	8.667			
		3	3.673	.846	.002	1.349	5.997			
	3	1	1.889	.813	.111	343	4.122			
		2	-3.673	.846	.002	-5.997	-1.349			

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AL KNEE EXTENSOR WALKING EMG MF ANALYSIS

#### Tests of Within-Subjects Effects

		Type III Sum					Partial Eta	Noncent.	Observed
Source		of Squares	df	Mean Square	F	Sig.	Squared	Parameter	Power <sup>a</sup>
Leg	Sphericity Assumed	4711.186	1	4711.186	16.728	.002	.603	16.728	.96
	Greenhouse-Geisser	4711.186	1.000	4711.186	16.728	.002	.603	16.728	.96
	Huynh-Feldt	4711.186	1.000	4711.186	16.728	.002	.603		.96
	Lower-bound	4711.186	1.000	4711.186	16.728	.002	.603		.96
_eg * Group	Sphericity Assumed	44.412	1	44.412	.158	.699	.014		.06
	Greenhouse-Geisser	44.412	1.000	44.412	.158	.699	.014	Parameter           16.728           16.728           16.728           16.728           16.728           16.728           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.158           1.175           1.175           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.13.352           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457           1.1457	.06
	Huynh-Feldt	44.412	1.000	44.412	.158	.699	.014		.06
	Lower-bound	44.412	1.000	44.412	.158	.699	.014	.158	.06
Error(Leg)	Sphericity Assumed	3097.908	11	281.628					
	Greenhouse-Geisser	3097.908	11.000	281.628					
	Huynh-Feldt	3097.908	11.000	281.628 281.628					
Muscle	Lower-bound	3097.908 72126.230	2		46.153	.000	.808	02.207	1.00
viuscie	Sphericity Assumed Greenhouse-Geisser	72126.230	1.753	36063.115 41137.943	46.153	.000	.808		1.00
	Huynh-Feldt	72126.230	2.000	36063.115	46.153	.000	.808		1.00
	Lower-bound	72126.230	1.000	72126,230	46.153	.000	.808		1.00
Muscle * Group	Sphericity Assumed	10432.987	2	5216.494	6.676	.005	.378		.87
addic broup	Greenhouse-Geisser	10432.987	1.753	5950.563	6.676	.008	.378		.83
	Huynh-Feldt	10432.987	2.000	5216.494	6.676	.005	.378		.87
	Lower-bound	10432.987	1.000	10432.987	6.676	.025	.378		.65
Error(Muscle)	Sphericity Assumed	17190.233	22	781.374	0.070	.020	.570	0.070	.00
	Greenhouse-Geisser	17190.233	19.286	891.330					
	Huvnh-Feldt	17190.233	22.000	781.374					
	Lower-bound	17190.233	11.000	1562.748					
Гime	Sphericity Assumed	1006.344	2	503.172	2.160	.139	.164	4 31 9	.39
	Greenhouse-Geisser	1006.344	1.683	598.098	2.160	.139	.164		.35
	Huynh-Feldt	1006.344	2.000	598.098	2.160	.149	.164		.3
	Lower-bound	1006.344	1.000	1006.344	2.160	.139	.164		.20
Fime * Group	Sphericity Assumed	678.966	1.000	339.483	1.457	.170	.164		.20
and oroup	Greenhouse-Geisser	678.966	1.683	403.528	1.457	.255	.117		.2
	Greenhouse-Geisser Huynh-Feldt	678.966	2.000	403.528 339.483	1.457	.256	.117		.21
	Lower-bound	678.966	1.000	678,966	1.457	.253	.117		.19
		5125.887	22		1.457	.253	.117	1.457	.13
Error(Time)	Sphericity Assumed	5125.887		232.995					
	Greenhouse-Geisser Huynh-Feldt	5125.887	18.508	276.950 232.995					
		5125.887							
_eg * Muscle	Lower-bound Sphericity Assumed	341.478	11.000	465.990 170.739	.329	.723	.029	650	.09
Leg Muscle		341.478		233.654	.329	.658	.029		.08
	Greenhouse-Geisser		1.461						
	Huynh-Feldt Lower-bound	341.478 341.478	1.782	191.610 341.478	.329	.699 .578	.029		20. 30.
			2						.00
_eg * Muscle * Group	Sphericity Assumed	1506.732 1506.732	1.461	753.366	1.451	.256	.117		.2
	Greenhouse-Geisser Huvnh-Feldt	1506.732	1.782	845.456	1.451	.258	.117		.2
Frror(Leg*Muscle)	Lower-bound	1506.732	1.000	1506.732	1.451	.254	.117	1.451	.19
Enor(Leg-Muscle)	Sphericity Assumed	11422.811		519.219					
	Greenhouse-Geisser	11422.811	16.076	710.543					
	Huynh-Feldt	11422.811	19.604	582.687					
a a # Wine a	Lower-bound	11422.811	11.000	1038.437	0.26	.974	002	052	
_eg * Time	Sphericity Assumed	8.491	2	4.245	.026		.002		.0
	Greenhouse-Geisser	8.491	1.369	6.200	.026	.931	.002		.05
	Huynh-Feldt	8.491	1.641	5.175	.026	.955	.002		.05
	Lower-bound	8.491	1.000	8.491	.026	.874	.002		.05
_eg * Time * Group	Sphericity Assumed	96.078	2	48.039	.299	.744	.026		.09
	Greenhouse-Geisser	96.078	1.369	70.162	.299	.663	.026		.08
	Huynh-Feldt	96.078	1.641	58.556	.299	.702	.026		.0
	Lower-bound	96.078	1.000	96.078	.299	.595	.026	.299	.0
Error(Leg*Time)	Sphericity Assumed	3532.516	22	160.569					
	Greenhouse-Geisser	3532.516	15.063	234.513					
	Huynh-Feldt	3532.516	18.049	195.720					
	Lower-bound	3532.516	11.000	321.138					
duscle * Time	Sphericity Assumed	1451.241	4	362.810	2.129	.093	.162		.58
	Greenhouse-Geisser	1451.241	2.736	530.350	2.129	.122	.162		.40
	Huynh-Feldt	1451.241	4.000	362.810	2.129	.093	.162		.58
	Lower-bound	1451.241	1.000	1451.241	2.129	.172	.162		.20
duscle * Time * Group	Sphericity Assumed	914.323	4	228.581	1.341	.270	.109		.38
	Greenhouse-Geisser	914.323	2.736	334.135	1.341	.279	.109		.30
	Huynh-Feldt	914.323	4.000	228.581	1.341	.270	.109		.3
	Lower-bound	914.323	1.000	914.323	1.341	.271	.109	1.341	.18
Error(Muscle*Time)	Sphericity Assumed	7497.449	44	170.397					
	Greenhouse-Geisser	7497.449	30.100	249.083					
	Huynh-Feldt	7497.449	44.000	170.397					
	Lower-bound	7497.449	11.000	681.586					
_eg * Muscle * Time	Sphericity Assumed	356.807	4	89.202	.729	.577	.062		.2
	Greenhouse-Geisser	356.807	2.407	148.260	.729	.516	.062		.15
	Huynh-Feldt	356.807	3.408	104.697	.729	.558	.062		.2
	Lower-bound	356.807	1.000	356.807	.729	.412	.062	.729	.13
eg * Muscle * Time *	Sphericity Assumed	767.021	4	191.755	1.566	.200	.125	6.265	.4
Group	Greenhouse-Geisser	767.021	2.407	318.712	1.566	.225	.125	3.770	.32
	Huynh-Feldt	767.021	3.408	225.065	1.566	.209	.125	5.338	.40
	Lower-bound	767.021	1.000	767.021	1.566	.237	.125	1.566	.20
Error(Leg*Muscle*Time)	Sphericity Assumed	5386.546	44	122.421					
-	Greenhouse-Geisser	5386.546	26.473	203.474					
	Huynh-Feldt	5386.546	37.488	143.687					
			11.000	489.686					

# APPENDIX AM: TIBIALIS ANTERIOR WALKING EMG MF ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Leg	Sphericity Assumed	238.989	1	238.989	.574	.462	.042	.574	.108
	Greenhouse-Geisser	238.989	1.000	238.989	.574	.462	.042	.574	.108
	Huynh-Feldt	238.989	1.000	238.989	.574	.462	.042	.574	.108
	Lower-bound	238.989	1.000	238.989	.574	.462	.042	.574	.108
Leg * Group	Sphericity Assumed	74.039	1	74.039	.178	.680	.013	.178	.068
	Greenhouse-Geisser	74.039	1.000	74.039	.178	.680	.013	.178	.068
	Huynh-Feldt	74.039	1.000	74.039	.178	.680	.013	.178	.068
	Lower-bound	74.039	1.000	74.039	.178	.680	.013	.178	.068
Error(Leg)	Sphericity Assumed	5414.252	13	416.481					
	Greenhouse-Geisser	5414.252	13.000	416.481					
	Huynh-Feldt	5414.252	13.000	416.481					
	Lower-bound	5414.252	13.000	416.481					
Time	Sphericity Assumed	616.420	2	308.210	5.797	.008	.308	11.593	.828
	Greenhouse-Geisser	616.420	1.691	364.451	5.797	.012	.308	9.804	.775
	Huynh-Feldt	616.420	2.000	308.210	5.797	.008	.308	11.593	.828
	Lower-bound	616.420	1.000	616.420	5.797	.032	.308	5.797	.606
Time * Group	Sphericity Assumed	1444.556	2	722.278	13.584	.000	.511	27.168	.995
	Greenhouse-Geisser	1444.556	1.691	854.078	13.584	.000	.511	22.976	.989
	Huynh-Feldt	1444.556	2.000	722.278	13.584	.000	.511	27.168	.995
	Lower-bound	1444.556	1.000	1444.556	13.584	.003	.511	13.584	.925
Error(Time)	Sphericity Assumed	1382.431	26	53.170					
	Greenhouse-Geisser	1382.431	21.988	62.873					
	Huynh-Feldt	1382.431	26.000	53.170					
	Lower-bound	1382.431	13.000	106.341					
Leg * Time	Sphericity Assumed	116.235	2	58.117	.838	.444	.061	1.677	.178
	Greenhouse-Geisser	116.235	1.632	71.232	.838	.425	.061	1.368	.163
	Huynh-Feldt	116.235	1.977	58.789	.838	.443	.061	1.658	.177
	Lower-bound	116.235	1.000	116.235	.838	.377	.061	.838	.136
Leg * Time * Group	Sphericity Assumed	11.108	2	5.554	.080	.923	.006	.160	.061
	Greenhouse-Geisser	11.108	1.632	6.807	.080	.889	.006	.131	.060
	Huynh-Feldt	11.108	1.977	5.618	.080	.921	.006	.158	.061
	Lower-bound	11.108	1.000	11.108	.080	.782	.006	.080	.058
Error(Leg*Time)	Sphericity Assumed	1802.136	26	69.313					
	Greenhouse-Geisser	1802.136	21.213	84.954					
	Huynh-Feldt	1802.136	25.703	70.114					
	Lower-bound	1802.136	13.000	138.626					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval for Difference <sup>b</sup>		
Time	(I) Group	(J) Group	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound	
1	Con	Inj	-19.156	6.150	.008	-32.443	-5.870	
	Inj	Con	19.156	6.150	.008	5.870	32.443	
2	Con	Inj	-4.297	6.280	.506	-17.865	9.271	
	Inj	Con	4.297	6.280	.506	-9.271	17.865	
3	Con	Inj	-22.889	5.693	.001	-35.187	-10.590	
	Inj	Con	22.889	5.693	.001	10.590	35.187	

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

### Pairwise Comparisons

Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differe	
Group	(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	2	-1.055	2.222	1.000	-7.155	5.045
		3	4.362	2.705	.392	-3.066	11.790
	2	1	1.055	2.222	1.000	-5.045	7.155
		3	5.417	3.246	.357	-3.495	14.330
	3	1	-4.362	2.705	.392	-11.790	3.066
		2	-5.417	3.246	.357	-14.330	3.495
Inj	1	2	13.805	2.078	.000	8.098	19.511
		3	.630	2.530	1.000	-6.318	7.578
	2	1	-13.805	2.078	.000	-19.511	-8.098
		3	-13.175	3.036	.002	-21.512	-4.838
	3	1	630	2.530	1.000	-7.578	6.318
		2	13.175	3.036	.002	4.838	21.512

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AN: KNEE EXTENSOR RUNNING EMG RMS ANALYSIS

#### Tests of Within-Subjects Effects

		Type III Sum					Partial Eta	Noncent.	Observed
Source		ofSquares	df	Mean Square	F	Sig.	Squared	Parameter	Power <sup>a</sup>
eg	Sphericity Assumed	89.931	1	89.931	.745	.404	.054	.745	.10
	Greenhouse-Geisser	89.931	1.000	89.931	.745	.404	.054	.745	.13
	Huynh-Feldt	89.931	1.000	89.931	.745	.404	.054	.745	.10
	Lower-bound	89.931	1.000	89.931	.745	.404	.054	.745	.1:
eg * Group	Sphericity Assumed	247.681	1	247.681	2.053	.176	.136	2.053	.2
	Greenhouse-Geisser	247.681	1.000	247.681	2.053	.176	.136	2.053	.2
	Huynh-Feldt	247.681	1.000	247.681	2.053	.176	.136	.745 .745 .745 .745 .745 .2053 2.053 2.053 2.053 2.053 2.053 .2053 .2053 .2053 .2053 .2053 .2053 .2053 .2053 .2053 .1.671 1.176 1.378 .835 .284 .335 .2839 .2.639 .2.639 .2.639 .2.654 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.396 .2.659 .3.172 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.452 .3.379 .3.371 .3.311.331 .3.371	.2
	Lower-bound	247.681	1.000	247.681	2.053	.176	.136	8         2.053           9         63.391           9         44.631           9         52.284           9         31.696           0         1.671           0         1.176           0         1.378           0         1.378           0         3.355           9         3.717           9         4.355           9         2.639           2         7.871           2         5.543           2         6.494           2         3.935           0         .451           0         .530           0         .451           0         .530           0         .451           0         .2396           4         2.396           4         2.396           4         1.198           7         3.452	.2
rror(Leg)	Sphericity Assumed	1568.516	13	120.655					
	Greenhouse-Geisser	1568.516	13.000	120.655					
	Huynh-Feldt	1568.516	13.000	120.655					
	Lower-bound	1568.516	13.000	120.655					
luscle	Sphericity Assumed	19993.094	2	9996.547	31.696	.000	.709	63.391	1.0
	Greenhouse-Geisser	19993.094	1.408	14198.611	31.696	.000	.709	44.631	1.0
	Huynh-Feldt	19993.094	1.650	12120.258	31.696	.000	.709		1.0
	Lower-bound	19993.094	1.000	19993.094	31.696	.000	.709		.9
uscle * Group	Sphericity Assumed	526.887	2	263.444	.835	.445	.060		.1
	Greenhouse-Geisser	526,887	1,408	374,182	.835	.411	.060		.1
	Huvnh-Feldt	526.887	1.650	319,411	.835	.427	.060		.1
	Lower-bound	526.887	1.000	526.887	.835	.377	.060		.1
rror(Muscle)	Sphericity Assumed	8200.194	26	315.392	.035	.377	.000	.035	
non(wascie)	Greenhouse-Geisser	8200.194	18.305	447.968					
	Huynh-Feldt	8200.194	21.444	382.395					
	Lower-bound	8200.194	13.000	630.784					
me	Sphericity Assumed	185.945	2	92.972	2.639	.090	.169		.4
	Greenhouse-Geisser	185.945	1.409	132.011	2.639	.112	.169		.3
	Huynh-Feldt	185.945	1.650	112.679	2.639	.103	.169		.4
	Lower-bound	185.945	1.000	185.945	2.639	.128	.169		.3
me * Group	Sphericity Assumed	277.265	2	138.632	3.935	.032	.232		.6
	Greenhouse-Geisser	277.265	1.409	196.843	3.935	.050	.232		.5
	Huynh-Feldt	277.265	1.650	168.017	3.935	.042	.232	6.494	.6
	Lower-bound	277.265	1.000	277.265	3.935	.069	.232	3.935	.4
rror(Time)	Sphericity Assumed	915.934	26	35.228					
	Greenhouse-Geisser	915.934	18.311	50.020					
	Huynh-Feldt	915.934	21.453	42.695					
	Lower-bound	915.934	13.000	70.456					
eg * Muscle	Sphericity Assumed	92.070	2	46.035	.265	.769	.020	.530	.c
	Greenhouse-Geisser	92.070	1.705	54.009	.265	.735	.020	.451	.0
	Huynh-Feldt	92.070	2.000	46.035	.265	.769	.020	.530	.0
	Lower-bound	92.070	1.000	92.070	.265	.615	.020		.0
eg * Muscle * Group	Sphericity Assumed	416.546	2	208.273	1.198	.318	.084		.2
	Greenhouse-Geisser	416.546	1.705	244.351	1.198	.314	.084		.2
	Huynh-Feldt	416.546	2.000	208.273	1.198	.318	.084		.2
	Lower-bound	416.546	1.000	416.546	1.198	.294	.084		.1
rror(Leg*Muscle)	Sphericity Assumed	4519.440	26	173.825	1.100	.204	.004	1.155	
indication in a series of the	Greenhouse-Geisser	4519.440	22.161	203.935					
	Huynh-Feldt	4519.440	26.000	173.825					
	Lower-bound	4519.440	13.000	347.649					
eg * Time	Sphericity Assumed	18.499	2	9.249	1.726	.198	.117	2.452	.3
eg mine	Greenhouse-Geisser	18,499	1.958	9,449	1.726	.199	.117		.3
	Huynh-Feldt	18.499	2.000	9.249	1.726	.198	.117		.3
	Lower-bound	18.499	1.000	18.499	1.726	.212	.117		.2
eg * Time * Group	Sphericity Assumed	33.220	2	16.610	3.100	.062	.193		.5
	Greenhouse-Geisser	33.220	1.958	16.969	3.100	.063	.193		.5
	Huynh-Feldt	33.220	2.000	16.610	3.100	.062	.193		.5
	Lower-bound	33.220	1.000	33.220	3.100	.102	.193	3.100	.3
rror(Leg*Time)	Sphericity Assumed	139.323	26	5.359					
	Greenhouse-Geisser	139.323	25.450	5.474					
	Huynh-Feldt	139.323	26.000	5.359					
	Lower-bound	139.323	13.000	10.717					
uscle * Time	Sphericity Assumed	38.699	4	9.675	.660	.623	.048	2.639	.2
	Greenhouse-Geisser	38.699	2.140	18.083	.660	.535	.048	1.412	.1
	Huynh-Feldt	38.699	2.772	13.961	.660	.571	.048	1.829	.1
	Lower-bound	38.699	1.000	38.699	.660	.431	.048	.660	.1
uscle * Time * Group	Sphericity Assumed	38.156	4	9.539	.651	.629	.048	2.602	.1
	Greenhouse-Geisser	38.156	2.140	17.829	.651	.540	.048	1.392	.1
	Huynh-Feldt	38.156	2.772	13.765	.651	.576	.048		.1
	Lower-bound	38.156	1.000	38.156	.651	.434	.048		.1
ror(Muscle*Time)	Sphericity Assumed	762.424	52	14.662					
	Greenhouse-Geisser	762.424	27.822	27.404					
	Huynh-Feldt	762.424	36.034	21.158					
	Lower-bound	762.424	13.000	58.648					
eg * Muscle * Time	Sphericity Assumed	34.318	4	8.579	.768	.551	.056	2.072	.2
g waste time	Greenhouse-Geisser								
		34.318	1.732	19.808	.768	.458	.056		.1
	Huynh-Feldt	34.318	2.129	16.120	.768	.481	.056		.1
and the second second second	Lower-bound	34.318	1.000	34.318	.768	.397	.056		.1
eg * Muscle * Time * roup	Sphericity Assumed	19.290	4	4.823	.432	.785	.032	1.727	.1
	Greenhouse-Geisser	19.290	1.732	11.134	.432	.626	.032	.748	.1
	Huynh-Feldt	19.290	2.129	9.061	.432	.666	.032	.919	.1
	Lower-bound	19.290	1.000	19.290	.432	.523	.032	.432	.0
rror(Leg*Muscle*Time)	Sphericity Assumed	580.674	52	11.167					
	Greenhouse-Geisser	580.674	22.522	25.782					
	Huynh-Feldt	580.674	27.675	20.982					
	Lower-bound	580.674	13.000	44.667					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval for Difference <sup>a</sup>			
Time	(I) Group	(J) Group	J)	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound		
1	Con	Inj	-1.844	4.886	.712	-12.400	8.711		
	Inj	Con	1.844	4.886	.712	-8.711	12.400		
2	Con	Inj	-6.816	6.632	.323	-21.143	7.512		
	Inj	Con	6.816	6.632	.323	-7.512	21.143		
3	Con	Inj	-4.504	6.462	.498	-18.464	9.455		
	Inj	Con	4.504	6.462	.498	-9.455	18.464		

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

### Pairwise Comparisons

Measure: MEASURE\_1

			Mean Difference (I-			95% Confidence Interval Difference <sup>b</sup>				
Group	(I) Time	(J) Time	J)	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound			
Con	1	2	.707	1.486	1.000	-3.373	4.788			
		3	1.302	1.495	1.000	-2.802	5.406			
	2	1	707	1.486	1.000	-4.788	3.373			
		3	.594	.769	1.000	-1.516	2.705			
	3	1	-1.302	1.495	1.000	-5.406	2.802			
		2	594	.769	1.000	-2.705	1.516			
Inj	1	2	-4.264*	1.390	.027	-8.081	447			
		3	-1.358	1.398	1.000	-5.197	2.481			
	2	1	4.264	1.390	.027	.447	8.081			
		3	2.906	.719	.004	.932	4.880			
	3	1	1.358	1.398	1.000	-2.481	5.197			
		2	-2.906	.719	.004	-4.880	932			

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AO: KNEE EXTENSOR RUNNING EMG MF ANALYSIS

#### Tests of Within-Subjects Effects

		Type III Sum					Partial Eta	Noncent.	Observed
Source		of Squares	df	Mean Square	F	Sig.	Squared	Parameter	Power <sup>a</sup>
Leg	Sphericity Assumed	567.530	1	567.530	4.671	.050	.264		.51
	Greenhouse-Geisser	567.530	1.000	567.530	4.671	.050	.264		.51
	Huynh-Feldt	567.530	1.000	567.530	4.671	.050	.264		.51
	Lower-bound	567.530	1.000	567.530	4.671	.050	.264		.51
Leg * Group	Sphericity Assumed	111.600	1	111.600	.919	.355	.066		.14
	Greenhouse-Geisser Huvnh-Feldt	111.600 111.600	1.000	111.600 111.600	.919	.355	.066		.14
	Lower-bound	111.600	1.000	111.600	.919	.355	.066	Parameter           4.671           4.671           4.671           4.671           4.671           919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .919           .910           .108.400           .22.531           .14.117           .919           .3.189           .3.189           .3.189           .3.189           .3.189           .3.181           .3.871           .3.871           .3.811           .3.811           .3.811           .3.811           .3.811           .3.811 <td>.14</td>	.14
Error(Leg)	Sphericity Assumed	1579.414	13	121.493	.919	.355	.000	.919	.14
Litor(Log)	Greenhouse-Geisser	1579.414	13.000	121.493					
	Huynh-Feldt	1579.414	13.000	121.493					
	Lower-bound	1579.414	13,000	121,493					
Muscle	Sphericity Assumed	91618.002	2	45809.001	108.400	.000	.893	216.800	1.00
	Greenhouse-Geisser	91618.002	1.958	46785.124	108.400	.000	.893		1.00
	Huynh-Feldt	91618.002	2.000	45809.001	108.400	.000	.893	216.800	1.00
	Lower-bound	91618.002	1.000	91618.002	108.400	.000	.893	108.400	1.00
Muscle * Group	Sphericity Assumed	11931.231	2	5965.616	14.117	.000	.521	28.233	.99
	Greenhouse-Geisser	11931.231	1.958	6092.734	14.117	.000	.521	27.644	.99
	Huynh-Feldt	11931.231	2.000	5965.616	14.117	.000	.521	28.233	.99
	Lower-bound	11931.231	1.000	11931.231	14.117	.002	.521	14.117	.93
Error(Muscle)	Sphericity Assumed	10987.399	26	422.592					
	Greenhouse-Geisser	10987.399	25.458	431.597					
	Huynh-Feldt	10987.399	26.000	422.592					
	Lower-bound	10987.399	13.000	845.185					
Time	Sphericity Assumed	508.648	2	254.324	3.189	.058	.197		.55
	Greenhouse-Geisser	508.648	1.421	358.015	3.189	.078	.197		.46
	Huynh-Feldt	508.648	1.668	304.992	3.189	.069	.197		.50
	Lower-bound	508.648	1.000	508.648	3.189	.097	.197		.38
Time * Group	Sphericity Assumed	333.481	2	166.741	2.091	.144	.139		.39
	Greenhouse-Geisser	333.481	1.421	234.722	2.091	.161	.139		.32
	Huynh-Feldt	333.481	1.668	199.960	2.091	.154	.139		.35
	Lower-bound	333.481	1.000	333.481	2.091	.172	.139	2.091	.26
Error(Time)	Sphericity Assumed	2073.275	26	79.741					
	Greenhouse-Geisser	2073.275	18.470	112.253					
	Huynh-Feldt	2073.275	21.681	95.628					
	Lower-bound	2073.275	13.000	159.483					
_eg * Muscle	Sphericity Assumed	1732.279	2	866.139	1.755	.193	.119		.33
	Greenhouse-Geisser	1732.279	1.340	1292.456	1.755	.205	.119		.26
	Huynh-Feldt	1732.279	1.553	1115.626	1.755	.201	.119		.29
	Lower-bound	1732.279	1.000	1732.279	1.755	.208	.119		.23
Leg * Muscle * Group	Sphericity Assumed	884.459	2	442.230	.896	.420	.064		.18
	Greenhouse-Geisser	884.459	1.340	659.896	.896	.387	.064		.15
	Huynh-Feldt	884.459	1.553	569.611	.896	.400	.064		.16
	Lower-bound	884.459	1.000	884.459	.896	.361	.064	.896	.14
Error(Leg*Muscle)	Sphericity Assumed	12828.590	26	493.407					
	Greenhouse-Geisser	12828.590	17.424	736.264					
	Huynh-Feldt Lower-bound	12828.590 12828.590	20.186	635.530 986.815					
Leg * Time		33.828	13.000		.323	.727	.024	646	.09
Leg minie	Sphericity Assumed Greenhouse-Geisser	33.828	1.581	16.914 21.402	.323	.677	.024		.09
	Huynh-Feldt	33.828	1.901	17.795	.323	.716	.024		.09
	Lower-bound	33.828	1.000	33.828	.323	.579	.024		.09
_eg * Time * Group	Sphericity Assumed	1.046	2	.523	.010	.990	.001		.05
Leg mile oloup	Greenhouse-Geisser	1.046	1.581	.662	.010	.977	.001		.05
	Huynh-Feldt	1.046	1.901	.550	.010	.988	.001		.05
	Lower-bound	1.046	1.000	1.046	.010	.922	.001		.05
Error(Leg*Time)	Sphericity Assumed	1360.513	26	52.327	.010	.522	.001	.010	.00
	Greenhouse-Geisser	1360.513	20.548	66.212					
	Huynh-Feldt	1360.513	24.714	55.051					
	Lower-bound	1360.513	13.000	104.655					
Muscle * Time	Sphericity Assumed	968,156	4	242.039	3.060	.024	.191	12.240	.77
	Greenhouse-Geisser	968.156	2.547	380.049	3,060	.049	.191		.61
	Huynh-Feldt	968.156	3.464	279.459	3.060	.032	.191		.72
	Lower-bound	968.156	1.000	968.156	3.060	.104	.191		.36
Muscle * Time * Group	Sphericity Assumed	376.087	4	94.022	1.189	.327	.084		.34
	Greenhouse-Geisser	376.087	2.547	147,633	1.189	.325	.084		.26
	Huynh-Feldt	376.087	3.464	108.558	1.189	.327	.084		.31
	Lower-bound	376.087	1.000	376.087	1.189	.295	.084		.17
Error(Muscle*Time)	Sphericity Assumed	4112.990	52	79.096					
	Greenhouse-Geisser	4112.990	33.117	124.196					
	Huynh-Feldt	4112.990	45.037	91.324					
	Lower-bound	4112.990	13.000	316.384					
_eg * Muscle * Time	Sphericity Assumed	1037.573	4	259.393	3.713	.010	.222	14.850	.85
	Greenhouse-Geisser	1037.573	2.600	399.024	3.713	.025	.222		.71
	Huynh-Feldt	1037.573	3.558	291.601	3.713	.013	.222		.81
	Lower-bound	1037.573	1.000	1037.573	3.713	.076	.222		.43
_eg * Muscle * Time *	Sphericity Assumed	289.354	4	72.339	1.035	.398	.074		.30
Group	Greenhouse-Geisser	289.354	2.600	111.278	1.035	.382	.074		.24
	Huynh-Feldt	289.354	3.558	81.321	1.035	.394	.074		.28
	Lower-bound	289.354	1.000	289.354	1.035	.327	.074		.15
Error(Leg*Muscle*Time)	Sphericity Assumed	3633.225	52	69.870				1.000	.15
L(Log muscle fille)	Greenhouse-Geisser	3633.225	33.804	107.480					
	Huynh-Feldt	3633.225	46.256	78.545					
			40.200	/0.040					

# APPENDIX AP: TIBIALIS ANTERIOR RUNNING EMG MF ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Leg	Sphericity Assumed	302.953	1	302.953	.838	.377	.061	.838	.136
	Greenhouse-Geisser	302.953	1.000	302.953	.838	.377	.061	.838	.136
	Huynh-Feldt	302.953	1.000	302.953	.838	.377	.061	.838	.136
	Lower-bound	302.953	1.000	302.953	.838	.377	.061	.838	.136
Leg * Group	Sphericity Assumed	326.098	1	326.098	.902	.360	.065	.902	.143
	Greenhouse-Geisser	326.098	1.000	326.098	.902	.360	.065	.902	.143
	Huynh-Feldt	326.098	1.000	326.098	.902	.360	.065	.902	.143
	Lower-bound	326.098	1.000	326.098	.902	.360	.065	.902	.143
Error(Leg)	Sphericity Assumed	4702.452	13	361.727					
	Greenhouse-Geisser	4702.452	13.000	361.727					
	Huynh-Feldt	4702.452	13.000	361.727					
	Lower-bound	4702.452	13.000	361.727					
Time	Sphericity Assumed	124.075	2	62.037	1.178	.324	.083	2.355	.235
	Greenhouse-Geisser	124.075	1.938	64.034	1.178	.323	.083	2.282	.231
	Huynh-Feldt	124.075	2.000	62.037	1.178	.324	.083	2.355	.235
	Lower-bound	124.075	1.000	124.075	1.178	.298	.083	1.178	.172
Time * Group	Sphericity Assumed	985.474	2	492.737	9.353	.001	.418	18.707	.963
	Greenhouse-Geisser	985.474	1.938	508.592	9.353	.001	.418	18.123	.959
	Huynh-Feldt	985.474	2.000	492.737	9.353	.001	.418	18.707	.963
	Lower-bound	985.474	1.000	985.474	9.353	.009	.418	9.353	.807
Error(Time)	Sphericity Assumed	1369.698	26	52.681					
	Greenhouse-Geisser	1369.698	25.189	54.376					
	Huynh-Feldt	1369.698	26.000	52.681					
	Lower-bound	1369.698	13.000	105.361					
Leg * Time	Sphericity Assumed	456.722	2	228.361	3.646	.040	.219	7.291	.620
	Greenhouse-Geisser	456.722	1.334	342.382	3.646	.063	.219	4.863	.497
	Huynh-Feldt	456.722	1.544	295.854	3.646	.055	.219	5.628	.539
	Lower-bound	456.722	1.000	456.722	3.646	.079	.219	3.646	.424
Leg * Time * Group	Sphericity Assumed	36.605	2	18.303	.292	.749	.022	.584	.091
	Greenhouse-Geisser	36.605	1.334	27.441	.292	.661	.022	.390	.084
	Huynh-Feldt	36.605	1.544	23.712	.292	.693	.022	.451	.086
	Lower-bound	36.605	1.000	36.605	.292	.598	.022	.292	.079
Error(Leg*Time)	Sphericity Assumed	1628.675	26	62.641					
,	Greenhouse-Geisser	1628.675	17.341	93.918					
	Huynh-Feldt	1628.675	20.069	81.155					
	Lower-bound	1628.675	13.000	125.283					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differ	
Time	(I) Group	(J) Group	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
1	Con	Inj	-23.640*	6.425	.003	-37.520	-9.761
	Inj	Con	23.640*	6.425	.003	9.761	37.520
2	Con	Inj	-10.759	5.536	.074	-22.719	1.201
	Inj	Con	10.759	5.536	.074	-1.201	22.719
3	Con	Inj	-25.775	5.707	.001	-38.105	-13.446
	Inj	Con	25.775	5.707	.001	13.446	38.105

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

### Pairwise Comparisons

Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differ	
Group	(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	2	-3.685	2.963	.707	-11.821	4.451
		3	1.712	2.708	1.000	-5.724	9.147
	2	1	3.685	2.963	.707	-4.451	11.821
		3	5.397	2.543	.161	-1.585	12.379
	3	1	-1.712	2.708	1.000	-9.147	5.724
		2	-5.397	2.543	.161	-12.379	1.585
Inj	1	2	9.196	2.772	.017	1.585	16.807
		3	423	2.533	1.000	-7.379	6.532
	2	1	-9.196	2.772	.017	-16.807	-1.585
		3	-9.619	2.378	.004	-16.150	-3.088
	3	1	.423	2.533	1.000	-6.532	7.379
		2	9.619	2.378	.004	3.088	16.150

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

# APPENDIX AQ: COP AREA ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
System	Sphericity Assumed	345472.475	2	172736.237	12.318	.000	.528	24.636	.990
	Greenhouse-Geisser	345472.475	1.907	181205.689	12.318	.000	.528	23.484	.987
	Huynh-Feldt	345472.475	2.000	172736.237	12.318	.000	.528	24.636	.990
	Lower-bound	345472.475	1.000	345472.475	12.318	.005	.528	12.318	.891
System * Group	Sphericity Assumed	13905.789	2	6952.894	.496	.616	.043	.992	.121
	Greenhouse-Geisser	13905.789	1.907	7293.803	.496	.607	.043	.945	.119
	Huynh-Feldt	13905.789	2.000	6952.894	.496	.616	.043	.992	.121
	Lower-bound	13905.789	1.000	13905.789	.496	.496	.043	.496	.099
Error(System)	Sphericity Assumed	308512.011	22	14023.273					
	Greenhouse-Geisser	308512.011	20.972	14710.850					
	Huynh-Feldt	308512.011	22.000	14023.273					
	Lower-bound	308512.011	11.000	28046.546					
Time	Sphericity Assumed	81741.844	2	40870.922	1.415	.264	.114	2.829	.271
	Greenhouse-Geisser	81741.844	1.110	73610.255	1.415	.262	.114	1.571	.202
	Huynh-Feldt	81741.844	1.257	65003.456	1.415	.264	.114	1.779	.214
	Lower-bound	81741.844	1.000	81741.844	1.415	.259	.114	1.415	.193
Time * Group	Sphericity Assumed	103440.105	2	51720.053	1.790	.190	.140	3.580	.333
	Greenhouse-Geisser	103440.105	1.110	93149.997	1.790	.207	.140	1.988	.244
	Huynh-Feldt	103440.105	1.257	82258.535	1.790	.205	.140	2.251	.259
	Lower-bound	103440.105	1.000	103440.105	1.790	.208	.140	1.790	.231
Error(Time)	Sphericity Assumed	635581.123	22	28890.051					
	Greenhouse-Geisser	635581.123	12.215	52032.201					
	Huynh-Feldt	635581.123	13.832	45948.392					
	Lower-bound	635581.123	11.000	57780.102					
System * Time	Sphericity Assumed	53613.551	4	13403.388	1.336	.272	.108	5.346	.382
	Greenhouse-Geisser	53613.551	1.853	28927.661	1.336	.283	.108	2.477	.248
	Huynh-Feldt	53613.551	2.416	22195.565	1.336	.282	.108	3.228	.285
	Lower-bound	53613.551	1.000	53613.551	1.336	.272	.108	1.336	.185
System * Time * Group	Sphericity Assumed	39548.410	4	9887.103	.986	.425	.082	3.943	.286
	Greenhouse-Geisser	39548.410	1.853	21338.691	.986	.384	.082	1.827	.192
	Huynh-Feldt	39548.410	2.416	16372.714	.986	.400	.082	2.381	.218
	Lower-bound	39548.410	1.000	39548.410	.986	.342	.082	.986	.149
Error(System*Time)	Sphericity Assumed	441288.155	44	10029.276					
	Greenhouse-Geisser	441288.155	20.387	21645.536					
	Huynh-Feldt	441288.155	26.571	16608.149					
	Lower-bound	441288.155	11.000	40117.105					

# APPENDIX AR: COP LENGTH ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
System	Sphericity Assumed	1574351.704	2	787175.852	7.418	.003	.403	14.837	.906
	Greenhouse-Geisser	1574351.704	1.831	859947.308	7.418	.005	.403	13.581	.884
	Huynh-Feldt	1574351.704	2.000	787175.852	7.418	.003	.403	14.837	.906
	Lower-bound	1574351.704	1.000	1574351.704	7.418	.020	.403	7.418	.699
System * Group	Sphericity Assumed	156190.845	2	78095.423	.736	.490	.063	1.472	.159
	Greenhouse-Geisser	156190.845	1.831	85315.052	.736	.480	.063	1.347	.153
	Huynh-Feldt	156190.845	2.000	78095.423	.736	.490	.063	1.472	.159
	Lower-bound	156190.845	1.000	156190.845	.736	.409	.063	.736	.123
Error(System)	Sphericity Assumed	2334464.874	22	106112.040					
	Greenhouse-Geisser	2334464.874	20.138	115921.700					
	Huynh-Feldt	2334464.874	22.000	106112.040					
	Lower-bound	2334464.874	11.000	212224.079					
Time	Sphericity Assumed	7920412.461	2	3960206.231	6.487	.006	.371	12.973	.862
	Greenhouse-Geisser	7920412.461	1.073	7380035.853	6.487	.024	.371	6.962	.664
	Huynh-Feldt	7920412.461	1.204	6578395.375	6.487	.020	.371	7.810	.702
	Lower-bound	7920412.461	1.000	7920412.461	6.487	.027	.371	6.487	.641
Time * Group	Sphericity Assumed	7468020.870	2	3734010.435	6.116	.008	.357	12.232	.840
	Greenhouse-Geisser	7468020.870	1.073	6958509.047	6.116	.028	.357	6.564	.638
	Huynh-Feldt	7468020.870	1.204	6202656.010	6.116	.023	.357	7.364	.676
	Lower-bound	7468020.870	1.000	7468020.870	6.116	.031	.357	6.116	.616
Error(Time)	Sphericity Assumed	13431343.11	22	610515.596					
	Greenhouse-Geisser	13431343.11	11.805	1137725.342					
	Huynh-Feldt	13431343.11	13.244	1014142.380					
	Lower-bound	13431343.11	11.000	1221031.192					
System * Time	Sphericity Assumed	533540.490	4	133385.122	2.084	.099	.159	8.334	.573
	Greenhouse-Geisser	533540.490	2.691	198252.279	2.084	.129	.159	5.607	.454
	Huynh-Feldt	533540.490	3.970	134393.059	2.084	.100	.159	8.272	.570
	Lower-bound	533540.490	1.000	533540.490	2.084	.177	.159	2.084	.261
System * Time * Group	Sphericity Assumed	146887.917	4	36721.979	.574	.683	.050	2.294	.176
	Greenhouse-Geisser	146887.917	2.691	54580.420	.574	.619	.050	1.544	.149
	Huynh-Feldt	146887.917	3.970	36999.472	.574	.682	.050	2.277	.175
	Lower-bound	146887.917	1.000	146887.917	.574	.465	.050	.574	.107
Error(System*Time)	Sphericity Assumed	2816846.723	44	64019.244					
	Greenhouse-Geisser	2816846.723	29.603	95152.748					
	Huynh-Feldt	2816846.723	43.670	64503.011					
	Lower-bound	2816846.723	11.000	256076.975					

Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differ	
Time	(I) Group	(J) Group	J) J	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound
1	Con	Inj	-263.395	1462.504	.860	-3482.345	2955.555
	Inj	Con	263.395	1462.504	.860	-2955.555	3482.345
2	Con	Inj	-511.441	1492.603	.738	-3796.639	2773.757
	Inj	Con	511.441	1492.603	.738	-2773.757	3796.639
3	Con	Inj	-1467.885	1406.367	.319	-4563.277	1627.508
	Inj	Con	1467.885	1406.367	.319	-1627.508	4563.277

Based on estimated marginal means

a. Adjustment for multiple comparisons: Bonferroni.

## Pairwise Comparisons

Measure: MEASURE\_1

			Mean Difference (I-			95% Confiden Differ	
Group	(I) Time	(J) Time	J) J	Std. Error	Sig. <sup>b</sup>	Lower Bound	Upper Bound
Con	1	2	37.400	82.707	1.000	-195.836	270.636
		3	1121.196	328.081	.017	195.997	2046.395
	2	1	-37.400	82.707	1.000	-270.636	195.836
		3	1083.796	360.179	.036	68.079	2099.513
	3	1	-1121.196	328.081	.017	-2046.395	-195.997
		2	-1083.796	360.179	.036	-2099.513	-68.079
Inj	1	2	-210.646	65.385	.024	-395.035	-26.257
		3	-83.294	259.371	1.000	-814.728	648.140
	2	1	210.646	65.385	.024	26.257	395.035
		3	127.352	284.746	1.000	-675.643	930.347
	3	1	83.294	259.371	1.000	-648.140	814.728
		2	-127.352	284.746	1.000	-930.347	675.643

Based on estimated marginal means

\*. The mean difference is significant at the .05 level.

## APPENDIX AS: PROCIOCEPTION AND ROMBERG'S QUOTIENT ANALYSIS (AREA)

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
System	Sphericity Assumed	19.156	1	19.156	8.191	.015	.427	8.191	.741
	Greenhouse-Geisser	19.156	1.000	19.156	8.191	.015	.427	8.191	.741
	Huynh-Feldt	19.156	1.000	19.156	8.191	.015	.427	8.191	.741
	Lower-bound	19.156	1.000	19.156	8.191	.015	.427	8.191	.741
System * Group	Sphericity Assumed	.143	1	.143	.061	.810	.006	.061	.056
	Greenhouse-Geisser	.143	1.000	.143	.061	.810	.006	.061	.056
	Huynh-Feldt	.143	1.000	.143	.061	.810	.006	.061	.056
	Lower-bound	.143	1.000	.143	.061	.810	.006	.061	.056
Error(System)	Sphericity Assumed	25.727	11	2.339					
	Greenhouse-Geisser	25.727	11.000	2.339					
	Huynh-Feldt	25.727	11.000	2.339					
	Lower-bound	25.727	11.000	2.339					
Time	Sphericity Assumed	.180	2	.090	.019	.981	.002	.038	.052
	Greenhouse-Geisser	.180	1.719	.105	.019	.970	.002	.032	.052
	Huynh-Feldt	.180	2.000	.090	.019	.981	.002	.038	.052
	Lower-bound	.180	1.000	.180	.019	.893	.002	.019	.052
Time * Group	Sphericity Assumed	4.727	2	2.364	.496	.615	.043	.993	.121
	Greenhouse-Geisser	4.727	1.719	2.749	.496	.589	.043	.853	.115
	Huynh-Feldt	4.727	2.000	2.364	.496	.615	.043	.993	.121
	Lower-bound	4.727	1.000	4.727	.496	.496	.043	.496	.099
Error(Time)	Sphericity Assumed	104.756	22	4.762					
	Greenhouse-Geisser	104.756	18.912	5.539					
	Huynh-Feldt	104.756	22.000	4.762					
	Lower-bound	104.756	11.000	9.523					
System * Time	Sphericity Assumed	6.217	2	3.109	2.916	.075	.210	5.831	.511
	Greenhouse-Geisser	6.217	1.682	3.695	2.916	.087	.210	4.905	.462
	Huynh-Feldt	6.217	2.000	3.109	2.916	.075	.210	5.831	.511
	Lower-bound	6.217	1.000	6.217	2.916	.116	.210	2.916	.345
System * Time * Group	Sphericity Assumed	3.597	2	1.798	1.687	.208	.133	3.373	.316
	Greenhouse-Geisser	3.597	1.682	2.138	1.687	.214	.133	2.838	.287
	Huynh-Feldt	3.597	2.000	1.798	1.687	.208	.133	3.373	.316
	Lower-bound	3.597	1.000	3.597	1.687	.221	.133	1.687	.221
Error(System*Time)	Sphericity Assumed	23.456	22	1.066					
	Greenhouse-Geisser	23.456	18.506	1.268					
	Huynh-Feldt	23.456	22.000	1.066					
	Lower-bound	23.456	11.000	2.132					

# APPENDIX AS: PROCIOCEPTION AND ROMBERG'S QUOTIENT ANALYSIS (LENGTH)

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
System	Sphericity Assumed	.008	1	.008	6.233	.030	.362	6.233	.624
	Greenhouse-Geisser	.008	1.000	.008	6.233	.030	.362	6.233	.624
	Huynh-Feldt	.008	1.000	.008	6.233	.030	.362	6.233	.624
	Lower-bound	.008	1.000	.008	6.233	.030	.362	6.233	.624
System * Group	Sphericity Assumed	1.154E-7	1	1.154E-7	.000	.993	.000	.000	.050
	Greenhouse-Geisser	1.154E-7	1.000	1.154E-7	.000	.993	.000	.000	.050
	Huynh-Feldt	1.154E-7	1.000	1.154E-7	.000	.993	.000	.000	.050
	Lower-bound	1.154E-7	1.000	1.154E-7	.000	.993	.000	.000	.050
Error(System)	Sphericity Assumed	.015	11	.001					
	Greenhouse-Geisser	.015	11.000	.001					
	Huynh-Feldt	.015	11.000	.001					
	Lower-bound	.015	11.000	.001					
Time	Sphericity Assumed	.012	2	.006	3.428	.051	.238	6.855	.582
	Greenhouse-Geisser	.012	1.514	.008	3.428	.067	.238	5.188	.498
	Huynh-Feldt	.012	1.863	.006	3.428	.055	.238	6.387	.560
	Lower-bound	.012	1.000	.012	3.428	.091	.238	3.428	.394
Time * Group	Sphericity Assumed	.003	2	.001	.861	.437	.073	1.722	.179
	Greenhouse-Geisser	.003	1.514	.002	.861	.412	.073	1.303	.159
	Huynh-Feldt	.003	1.863	.002	.861	.430	.073	1.604	.173
	Lower-bound	.003	1.000	.003	.861	.373	.073	.861	.136
Error(Time)	Sphericity Assumed	.038	22	.002					
	Greenhouse-Geisser	.038	16.649	.002					
	Huynh-Feldt	.038	20.497	.002					
	Lower-bound	.038	11.000	.003					
System * Time	Sphericity Assumed	.002	2	.001	1.448	.257	.116	2.896	.276
	Greenhouse-Geisser	.002	1.934	.001	1.448	.257	.116	2.801	.271
	Huynh-Feldt	.002	2.000	.001	1.448	.257	.116	2.896	.276
	Lower-bound	.002	1.000	.002	1.448	.254	.116	1.448	.196
System * Time * Group	Sphericity Assumed	.001	2	.000	.478	.626	.042	.957	.118
	Greenhouse-Geisser	.001	1.934	.000	.478	.620	.042	.925	.117
	Huynh-Feldt	.001	2.000	.000	.478	.626	.042	.957	.118
	Lower-bound	.001	1.000	.001	.478	.504	.042	.478	.097
Error(System*Time)	Sphericity Assumed	.016	22	.001					
	Greenhouse-Geisser	.016	21.275	.001					
	Huynh-Feldt	.016	22.000	.001					
	Lower-bound	.016	11.000	.001					

### Tests of Within-Subjects Effects

# APPENDIX AT: THIGH CIRUMFERENCE ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Leg	Sphericity Assumed	.120	1	.120	.123	.732	.009	.123	.062
	Greenhouse-Geisser	.120	1.000	.120	.123	.732	.009	.123	.062
	Huynh-Feldt	.120	1.000	.120	.123	.732	.009	.123	.062
	Lower-bound	.120	1.000	.120	.123	.732	.009	.123	.062
Leg * Group	Sphericity Assumed	.290	1	.290	.296	.596	.022	.296	.080
	Greenhouse-Geisser	.290	1.000	.290	.296	.596	.022	.296	.080
	Huynh-Feldt	.290	1.000	.290	.296	.596	.022	.296	.080
	Lower-bound	.290	1.000	.290	.296	.596	.022	.296	.080
Error(Leg)	Sphericity Assumed	12.755	13	.981					
	Greenhouse-Geisser	12.755	13.000	.981					
	Huynh-Feldt	12.755	13.000	.981					
	Lower-bound	12.755	13.000	.981					
Time	Sphericity Assumed	4.655	2	2.328	3.811	.035	.227	7.621	.641
	Greenhouse-Geisser	4.655	1.926	2.418	3.811	.037	.227	7.338	.628
	Huynh-Feldt	4.655	2.000	2.328	3.811	.035	.227	7.621	.641
	Lower-bound	4.655	1.000	4.655	3.811	.073	.227	3.811	.440
Time * Group	Sphericity Assumed	2.455	2	1.228	2.010	.154	.134	4.019	.377
	Greenhouse-Geisser	2.455	1.926	1.275	2.010	.156	.134	3.870	.369
	Huynh-Feldt	2.455	2.000	1.228	2.010	.154	.134	4.019	.377
	Lower-bound	2.455	1.000	2.455	2.010	.180	.134	2.010	.260
Error(Time)	Sphericity Assumed	15.882	26	.611					
	Greenhouse-Geisser	15.882	25.033	.634					
	Huynh-Feldt	15.882	26.000	.611					
	Lower-bound	15.882	13.000	1.222					
Leg * Time	Sphericity Assumed	.983	2	.492	3.285	.053	.202	6.571	.572
	Greenhouse-Geisser	.983	1.471	.668	3.285	.072	.202	4.834	.483
	Huynh-Feldt	.983	1.741	.565	3.285	.062	.202	5.719	.530
	Lower-bound	.983	1.000	.983	3.285	.093	.202	3.285	.389
Leg * Time * Group	Sphericity Assumed	.118	2	.059	.395	.678	.029	.790	.107
	Greenhouse-Geisser	.118	1.471	.080	.395	.617	.029	.581	.098
	Huynh-Feldt	.118	1.741	.068	.395	.650	.029	.688	.103
	Lower-bound	.118	1.000	.118	.395	.541	.029	.395	.090
Error(Leg*Time)	Sphericity Assumed	3.892	26	.150					
	Greenhouse-Geisser	3.892	19.126	.203					
	Huynh-Feldt	3.892	22.630	.172					
	Lower-bound	3.892	13.000	.299					

a. Computed using alpha = .05

#### Pairwise Comparisons

Measure:	MEASURE	≣_1					
		Mean Difference (I-			95% Confiden Differe		
(I) Time	(J) Time	J)	Std. Error	Sig. <sup>a</sup>	Lower Bound	Upper Bound	
1	2	494	.197	.079	-1.035	.047	
	3	473	.188	.077	988	.043	
2	1	.494	.197	.079	047	1.035	
	3	.021	.221	1.000	585	.627	
3	1	.473	.188	.077	043	.988	
	2	021	.221	1.000	627	.585	

Based on estimated marginal means

# APPENDIX AU: KNEE ROM ANALYSIS

### Tests of Within-Subjects Effects

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power <sup>a</sup>
Leg	Sphericity Assumed	118.640	1	118.640	5.315	.038	.290	5.315	.569
	Greenhouse-Geisser	118.640	1.000	118.640	5.315	.038	.290	5.315	.569
	Huynh-Feldt	118.640	1.000	118.640	5.315	.038	.290	5.315	.569
	Lower-bound	118.640	1.000	118.640	5.315	.038	.290	5.315	.569
Leg * Group	Sphericity Assumed	7.126	1	7.126	.319	.582	.024	.319	.082
	Greenhouse-Geisser	7.126	1.000	7.126	.319	.582	.024	.319	.082
	Huynh-Feldt	7.126	1.000	7.126	.319	.582	.024	.319	.082
	Lower-bound	7.126	1.000	7.126	.319	.582	.024	.319	.082
Error(Leg)	Sphericity Assumed	290.161	13	22.320					
	Greenhouse-Geisser	290.161	13.000	22.320					
	Huynh-Feldt	290.161	13.000	22.320					
	Lower-bound	290.161	13.000	22.320					
Time	Sphericity Assumed	91.700	2	45.850	3.286	.053	.202	6.572	.573
	Greenhouse-Geisser	91.700	1.431	64.101	3.286	.073	.202	4.701	.475
	Huynh-Feldt	91.700	1.682	54.523	3.286	.064	.202	5.526	.520
	Lower-bound	91.700	1.000	91.700	3.286	.093	.202	3.286	.390
Time * Group	Sphericity Assumed	7.730	2	3.865	.277	.760	.021	.554	.089
	Greenhouse-Geisser	7.730	1.431	5.404	.277	.687	.021	.396	.083
	Huynh-Feldt	7.730	1.682	4.596	.277	.723	.021	.466	.086
	Lower-bound	7.730	1.000	7.730	.277	.608	.021	.277	.078
Error(Time)	Sphericity Assumed	362.796	26	13.954					
	Greenhouse-Geisser	362.796	18.597	19.508					
	Huynh-Feldt	362.796	21.864	16.593					
	Lower-bound	362.796	13.000	27.907					
Leg * Time	Sphericity Assumed	10.640	2	5.320	.784	.467	.057	1.568	.169
	Greenhouse-Geisser	10.640	1.386	7.679	.784	.428	.057	1.086	.146
	Huynh-Feldt	10.640	1.617	6.579	.784	.444	.057	1.268	.155
	Lower-bound	10.640	1.000	10.640	.784	.392	.057	.784	.130
Leg * Time * Group	Sphericity Assumed	24.063	2	12.032	1.773	.190	.120	3.545	.337
	Greenhouse-Geisser	24.063	1.386	17.367	1.773	.202	.120	2.456	.276
	Huynh-Feldt	24.063	1.617	14.879	1.773	.198	.120	2.867	.300
	Lower-bound	24.063	1.000	24.063	1.773	.206	.120	1.773	.235
Error(Leg*Time)	Sphericity Assumed	176.470	26	6.787					
	Greenhouse-Geisser	176.470	18.012	9.797					
	Huynh-Feldt	176.470	21.024	8.394					
	Lower-bound	176.470	13.000	13.575					

Measure: MEASURE\_1