ABSTRACT

Title of Thesis: NEURAL MODULATION OF LEG
                                    STIFFNESS IN RESPONSE TO
                                    NEUROMUSCULAR FATIGUE

                                    Edward Chu, Master of Arts, 2016

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The spring-mass model is able to accurately represent hopping spring-like behavior (leg and joint stiffness), and leg and joint stiffness changes can reveal overall motor control responses to neural and muscular contributors of neuromuscular fatigue. By understanding leg stiffness modulation, we can determine which variables the nervous system targets to maintain motor performance and stability. The purpose of this study was to determine how neuromuscular fatigue affects hopping behavior by examining leg and joint stiffness before and after a single-leg calf raise fatiguing protocol. Post-fatigue, leg stiffness decreased for the exercised leg, but not for the non-exercised leg. Ankle and knee joint stiffness did not significantly change for either leg. This indicates that leg stiffness decreases primarily from muscular fatigue, but was not explained by ankle and knee joint stiffness. The decrease in leg stiffness may be an attempt to soften landing impact, while at the same time maintaining performance.

Neural Modulation of Leg Stiffness in Response to Neuromuscular Fatigue

by

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CHAPTER 1: INTRODUCTION

1.1. General Introduction

Human locomotion and the neuromuscular control of locomotion are often studied to enhance exercise training and recovery programs. To understand the neuromuscular control of locomotion, general relationships must be established to identify important variables, and underlying mechanisms must be understood to define the roles of those variables. To provide insight into the neuromuscular control of locomotion, an important aspect of human movement science is to understand how the human body reacts and adapts to physiological changes, which can include functional and chemical changes within the body. One physiological change that every individual experiences at some point throughout life is neuromuscular fatigue, which is typically associated with detrimental effects on performance.

In this current study we aimed to determine how neuromuscular fatigue, specifically the neural and muscular contributors of fatigue, affect performance of a locomotor task, single-leg hopping. Specifically, we examined changes in the stiffness variables (vertical leg stiffness and joint stiffness) of the lower body as a response to neural (fatigue from the nervous system sending the signal) and muscular (fatigue from the muscles performing the work) fatigue through the use of the spring-mass model.

When studying human locomotion, oftentimes the system is more complex and contains more degrees of freedom than necessary to perform the task (Bernstein, 1967). This overabundance of possible ways to interact with the environment becomes problematic when trying to study human behavior; therefore, to simplify the system, the spring-mass model was utilized to model single-leg hopping. The spring-mass model allows us to decrease the degrees of freedom, while at the same time accurately and effectively predicting the linear spring-like behavior of bouncing gait (Blickhan, 1989). By simplifying the complex behavior of human locomotion, we are able to better depict the general relationships, while at the same time providing insight into the mechanism behind these relationships.

Single-leg hopping in place is not a common movement performed in daily life; however, single-leg hopping is well suited for examining the effects of neural and muscular fatigue since each limb can receive a different exercise treatment. Assigning each limb to a different treatment is important because this allows the muscles of one leg to be exercised while the physical demands of the other leg are minimal. This design allows us to estimate the effects, and roles of neural and muscular fatigue because the exercised leg experiences a combination of neural and muscular fatigue, while the non-exercised leg experiences neural fatigue in the absence of muscular fatigue. Therefore, any stiffness changes observed for the non-exercised leg can be attributed to neural fatigue, and any stiffness changes observed for the exercised leg and not observed for the non-exercised leg can be attributed to muscular fatigue.

This thesis contains five chapters. The first chapter provides a literature review and an introduction to the study. The second chapter explains the methodology utilized. The third chapter reports the results from the study. The fourth chapter discusses the findings, addresses the limitations, and offers suggestions for future research. The fifth chapter concludes the thesis.

1.2. Literature Review

This review of literature starts with an introduction neuromuscular fatigue, delving specifically into the neural and muscular contributions of fatigue (Section 1.2.1.). Then we will introduce the spring-mass model (Section 1.2.2.), which is a simple model utilized to model bouncing gait such as running and hopping. One of the main variables of the spring-mass model is the stiffness of the spring (Section 1.2.3.), which is commonly used to model overall hopping spring-like behavior (leg stiffness) and joint spring-like behavior (joint stiffness). Stiffness in mechanical objects is typically invariant, but in human bouncing gait, stiffness can be altered and modulated in response to perturbations (Section 1.2.4.), and neuromuscular fatigue (Section 1.2.5.). Then we will explain the reasoning and justification behind this study (Sections 1.3.). Chapter 1 will conclude with the importance of understanding the neuromuscular control locomotion under conditions of neural and muscular fatigue (Section 1.4.), and the specific aims and hypotheses of this study (Section 1.5.).

1.2.1. Neuromuscular Fatigue

In human performance, fatigue is generally used as a very broad term and oftentimes the definition of fatigue can often be ambiguous. Fatigue is typically used to describe an acute impairment of performance, which can range from a psychological factor, such as the perception of the task becoming more difficult, to a physical factor, such as the physiological inability to perform the task (Enoka & Stuart 1985; Enoka & Stuart, 1992). Neuromuscular fatigue can be elicited from many different mechanisms, which many times cannot be parsed, so in turn, distinguishing the exact mechanistic causes to neuromuscular fatigue can be an arduous task. The origins of fatigue in many studies can be extremely difficult to extract because the complexity of the human body and motor control typically result in multiple causes to fatigue.

Neuromuscular fatigue occurs at multiple locations and levels throughout the motor pathway, such as, the motor cortex, spinal cord, and individual muscle fibers (Taylor et al., 2006). Neuromuscular fatigue is also thought to affect reflexes, which can affect muscle activation (Woods et al., 1987). When accounting for the effects of fatigue on reflexes, the origins of the effects of fatigue on reflexes becomes even more difficult to establish. Although establishing the exact origins of neuromuscular fatigue are often challenging, fatigue is thought to originate either centrally or peripherally. Studies on fatigue date back to the 1800s and one major finding in physiology was when Angelo Mosso suggested that fatigue has a central component, an individual's will and nervous system, and a peripheral component, an individual's muscles (Di Guilio et al., 2006). To this day, neuromuscular fatigue can still be

decomposed into central and peripheral components, albeit the definitions have evolved (Enoka & Duchateau, 2008; Taylor et al., 2006). The definitions of peripheral and central fatigue are not entirely consistent among studies due to the difficulty of determining the origins of the fatigue. In this study we classify central fatigue as fatigue of the nervous system and refer to central fatigue as neural fatigue. We will also classify peripheral fatigue as fatigue of the muscle and we will refer to peripheral fatigue as muscular fatigue.

Physiologically, muscular fatigue reduces the ability of muscles to produce torque by impairing the system at and distal to the neuromuscular junction, while neural fatigue occurs proximal to the neuromuscular junction and results from fatigue from the central nervous system (CNS) drive (Gandevia et al., 1996; Liu et al., 2005; Taylor et al., 2006; Yang, et al., 2009). Functionally, muscular fatigue decreases the ability of muscles to produce force, while neural fatigue decreases the ability of the nervous system to demand this force (Enoka & Stuart, 1992). In our current study, we will utilize the functional definitions of neural and muscular fatigue.

Muscular fatigue is thought to be mainly a result of metabolic inhibition, but the origins of neural fatigue are less clear (Enoka & Stuart, 1992). Some of the discrepancies in the role of neural fatigue on neuromuscular fatigue may result from both neural and muscular origins of fatigue possibly being task dependent. An example of task dependence fatigue is how the duration of the exercise has been shown to affect the origins of neuromuscular fatigue; long duration exercises have been shown to primarily elicit neural fatigue resulting from activation impairment,

while short duration exercises have been shown to primarily elicit muscular fatigue resulting from metabolic mechanisms (Baker et al., 1993).

Neural fatigue has been shown to slightly affect the voluntary muscle activation of the contralateral limb, which might be a result of the impairment of the motor cortex (Todd et al., 2003). A previous study that examined ankle dorsiflexor muscle fatigue demonstrated that muscular fatigue contributed more so than neural fatigue to the developed neuromuscular fatigue, specifically with neural fatigue contributing about 20% of the total neuromuscular fatigue (Kent-Braun, 1999).

Understanding the origins of neuromuscular fatigue are important because the various components of fatigue affect exercise recovery time. Previous research has shown that neural fatigue recovery occurs in hours, and muscular fatigue recovery occurs in minutes (Baker et al., 1993; Edwards et al., 1977). In our current study, we aim to study the biomechanical response to the various origins of neuromuscular fatigue.

1.2.2. Spring-Mass Model

Through the use of simple models, revealing and identifying general behaviors of the neuromuscular control of human locomotion are possible. Models allow us to simplify the system to just the necessary variables that accurately predict the behavior. The spring-mass model (Figure 1) is often used to model "bouncing" movements such as running and hopping because during these forms of locomotion the human body behaves similar to that of a spring (Cavagna et al., 1977; Full &

Koditschek, 1999). This model represents the human leg as a massless, linear spring that stores elastic energy (Cavagna et al., 1977; Blickhan, 1989).

A key parameter to the spring-mass model is the stiffness of the spring, which is often described as the leg stiffness (McMahon & Cheng, 1990). Leg stiffness is typically calculated as the ratio of the change in force and change in displacement following Hooke's Law. Leg stiffness is a simple parameter that is modeled as a linear spring, but in actuality the human bouncing gait is different from that of a mechanical spring because locomotion depends on a complex combination of muscles, tendons, ligaments, and neural commands. Despite the spring-mass model greatly simplifying bouncing gait behavior, the spring-mass model accurately describes the mechanics and energetics of running and hopping, and thus provides an elegant biomechanical explanation for the economy of these movements and a simple template for motor control (Blickhan, 1989; Full & Koditschek, 1999).
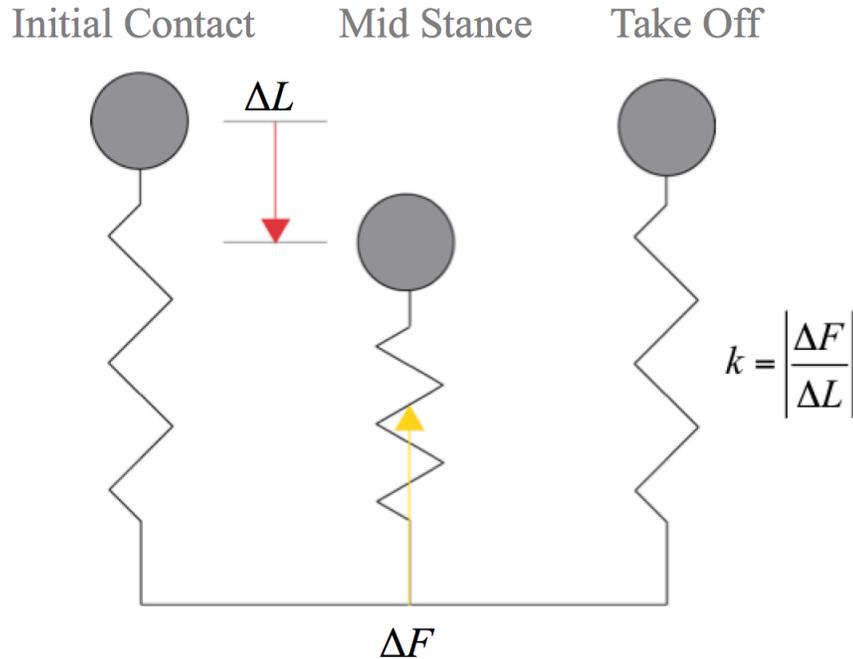
Figure 1. The spring-mass model representing hopping behavior as a massless, linear-spring attached to a point mass (whole body center of mass), with the stiffness of the spring (k) calculated as the absolute value of the ratio of the change in displacement (ΔL) and the change in force (ΔF) during the stance phase for hopping. Adapted from Farley and Morgenroth (1999).

1.2.3. Stiffness

Leg Stiffness. Leg stiffness has been widely studied and previous research suggests that leg stiffness can be modulated (Farley et al., 1991; Farley & González, 1996; Ferris & Farley, 1997; Hobara et al., 2013; McMahon & Cheng, 1990; McMahon et al., 1987). Leg stiffness in running and hopping has been found to change as a response to variations in movement frequency, surface stiffness, and joint stiffness (Blickhan, 1989; Farley et al., 1991; Farley & González, 1996; Farley et al., 1998; Farley & Morgenroth, 1999; Ferris & Farley, 1997; Hobara et al., 2009; Moritz et al., 2004).

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Human hopping exhibits spring-like behavior across a broad range of hopping frequencies. When hopping frequencies are within the range that the human body behaves like a spring, as hopping frequency increases, leg stiffness typically increases as well to maintain the body's spring-like behavior (Farley et al., 1991). Humans can choose a wide range of frequencies at which to hop, but the preferred hopping frequency for humans is about 2.2Hz (Farley et al., 1991). The preferred hopping frequency is selected based on the frequency at which the greatest amount of elastic energy can be stored and released, which leads to greater metabolic efficiency (Blickhan, 1989).

Changes in leg stiffness were also observed in running. Leg stiffness changes as a response to running frequency were similar to the leg stiffness changes as a response to hopping frequency (Farley & González, 1996). Both hopping and running studies found that leg stiffness can change by twofold as a response to changes in frequency (Farley et al., 1991; Farley and González, 1996).

Previous research has found that leg stiffness is modulated in response to varying surface stiffness, and leg stiffness during hopping greatly increases as a response to surfaces that are less stiff (Farley et al., 1998; Ferris & Farley, 1997; Moritz et al., 2004). The change in leg stiffness allowed for many of the other hopping mechanics to remain similar (Ferris & Farley, 1997). The increase in leg stiffness during hopping as a response to more compliant surfaces was predominantly caused by changes in the ankle, specifically increased ankle stiffness, and also knee touchdown angle (Farley et al., 1998; Moritz et al., 2004). Based on these findings, joint stiffness may play a great role in determining the overall leg stiffness.

Joint Stiffness. Leg stiffness is reliant on the stiffness of the joints in the lower body (joint stiffness). The stiffness of the joints is modeled as a torsion spring for the joints of the lower body (ankle, knee, and hip). The ankle, knee, and hip joint stiffness are connected in series and the stiffness of the lower body joints determines the stiffness of the whole body. This means that if the ankle, knee, and hip joint stiffness are very stiff, the stiffness of the whole body model (leg stiffness) would in turn be very stiff as well. Similarly, if the ankle, knee, and hip joint stiffness less stiff, leg stiffness would in turn also be less stiff.

In human hopping, leg stiffness was found to be predominantly dependent and very sensitive to the changes in ankle stiffness, while not as sensitive to the changes in the knee or hip stiffness (Farley & Morgenroth, 1999); however, in maximal jumping, leg stiffness was primarily determined by knee stiffness (Hobara et al., 2009). These results from the study indicate that joint stiffness plays a great role on determining leg stiffness, and the response in joint stiffness may be task specific.

Overall, leg stiffness can change as a response to changes in hopping/running frequency, surface stiffness, and joint stiffness. Based on these findings, we can deduce that leg stiffness is a biomechanical variable that is a major determinant of the human spring-like behavior in bouncing gait. We can also conclude that leg stiffness is a property of the spring-mass model that can be modulated.

1.2.4. Stiffness Response to Perturbations

The modulation of the body's stiffness is thought to be a mechanism for maintaining postural stability. Leg stiffness and joint stiffness are adjusted to help the body maintain stability when there are external perturbations to the body. A common

motor control strategy for adaptations to perturbations that cause instability is to stiffen the body (Fitzpatrick et al., 1992; Hortobágyi & DeVita, 2000). In elderly adults, leg stiffness was shown to increase as a result of increased muscle co-activation as a response to neuromotor function impairment (Hortobágyi & DeVita, 2000). Previous research also suggests that the human body increases joint stiffness as a response to external perturbations to maintain stability (Fitzpatrick et al., 1992). This phenomenon was observed when the ankle was subject to an external torque during quiet standing, and the response was to increase ankle stiffness when compared to unperturbed standing (Fitzpatrick et al., 1992).

1.2.5. Leg Stiffness Adaptations to Fatigue

While some aspects of the adaptations of leg stiffness are well-defined, previous literature pertaining to neuromuscular fatigue and leg stiffness are mixed (Dal Pupo et al., 2013; Dutto & Smith, 2002; Fourchet et al., 2015; Hayes & Caplan, 2014; Morin et al., 2012; Padua et al., 2006; Rabita et al., 2013). Previous studies examining the motor control of leg stiffness when the body experiences neuromuscular fatigue has mainly focused on running. Most of the studies that examined the changes in vertical leg stiffness during running in response to fatigue found no statistically significant change in leg stiffness when leg stiffness was defined as the ratio of the peak vertical ground reaction force (VGRF) and the change in center of mass (Fourchet et al., 2015; Hayes & Caplan, 2014; Morin et al., 2012; Rabita et al., 2013), while one study found that vertical leg stiffness decreased after fatigue (Dutto & Smith, 2002); however, when these studies examined leg stiffness using an alternative method for calculating leg stiffness (Morin et al., 2005), these

studies found that leg stiffness significantly decreases as a result of fatigue (Dal Pupo et al., 2013; Dutto & Smith, 2002; Fourchet et al., 2015; Hayes & Caplan, 2014; Morin et al., 2012; Rabita et al., 2013). The justification for leg stiffness decreasing during running, as a response to fatigue is that leg compression increased to a larger extent than the peak vertical ground reaction force (Fourchet et al., 2015).

The running studies that examined the changes of leg stiffness as a response to fatigue used varying methods to determine when subjects became fatigued. A couple of the studies used maximal oxygen consumption, VO2 max, (Fourchet et al., 2015; Hayes & Caplan, 2014: Rabita et al., 2013), while other studies utilized volitional fatigue resulting from high speed running or sprints to define fatigue (Dutto & Smith, 2002: Morin et al., 2012). These studies that examined the change of leg stiffness as a response to fatigue during running show that leg stiffness can be modulated as a response to fatigue, but the mechanism causing the response is still unknown. The possible effect of fatigue on leg stiffness has been more commonly studied in running; however, examining the effect of fatigue on leg stiffness in hopping can further reveal motor control strategies.

Response in Hopping. Examining the changes in leg stiffness as a result of fatigue during running is more ecological than examining the changes in leg stiffness as a result of fatigue in hopping; however, hopping may reveal the mechanism behind the modulation of leg stiffness as a response to fatigue.

Two previous studies have examined the changes of leg stiffness as a response to fatigue (Moritani et al., 1990; Padua et al., 2006). Moritani and colleagues (1990)

examined the changes in leg stiffness over 60 s of maximal height hopping, and determined that leg stiffness increased as a result of fatigue. On the other hand, Padua and colleagues (2006) compared the effects of fatigue on leg stiffness between males and females in a two-leg hopping task at set frequencies, with the fatiguing protocol consisting of a series of weighted squats (Padua et al., 2006). The results indicated that leg stiffness did not significantly change as a result of fatigue, and there were no difference between the sexes (Padua et al., 2006). Although the leg stiffness results from both studies differ, both studies show that muscle activation is significantly altered in response to fatigue.

1.3. Gap in Literature

Overall, there have been some studies that examined the changes in leg stiffness as a response to fatigue in running and hopping, but the conclusions seem to be uncertain, especially in hopping. The previous studies provide a good starting point, but many questions still remain in how the human body modulates leg stiffness when the body becomes fatigued. Much of the literature regarding the changes in leg stiffness as a result of neuromuscular fatigue is not well established.

Moritani and colleagues (1990) determined that leg stiffness increased over a 60 s bout of maximal height hopping; however, the methods utilized in this study were not idea because the task was inherently changing as the participants experienced fatigue. From the pre- to post-fatigue conditions, the maximum hopping height significantly decreased (Moritani et al., 1990), which could be a confounding factor and cause leg stiffness to increase. Even though Moritani and colleagues

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(1990) determined leg stiffness to increase with fatigue, the effects of fatigue on leg stiffness should be examined utilizing a task that is consistent from the pre- to post-fatigue conditions.

Padua and colleagues (2006) also examined the changes in leg stiffness as a result of fatigue, and their results showed no significant changes in leg stiffness as a result of fatigue. Padua and colleagues (2006) did find that muscle activation during hopping changed to a more ankle-dominant strategy as a response to the fatiguing protocol, which consisted of a weighted squat exercise that predominantly targeted the muscles about the knee. As a result, the findings from Padua and colleagues (2006) that muscle activation changed to a more ankle-dominant strategy.

The idea that the human body activates muscles that are not fatigued is supported by a study by Bonnard and colleagues (1994). In this study, participants performed submaximal hopping at a frequency of 2Hz until the hopping task became maximal performance (Bonnard et al., 1994). The fatiguing exercise ended up being the hopping protocol itself, which predominantly targets the muscles about the ankle. The results from that study indicate that muscle activation increased in the rectus femoris and participants also landed with greater knee flexion (Bonnard et al., 1994). A couple of limitations to the Bonnard study are that the sample size was very small (n=4) and the study did not examine leg stiffness (1994).

The combination of the studies by Padua and colleagues (2006) and Bonnard and colleagues (1994) both suggest that when muscles are under conditions of fatigue, the human body may change the muscle recruitment strategy; however, these

studies did not show any biomechanical adaptations to the changes in muscle activation.

The finding that muscle activation strategy changed, but leg stiffness did not change is particularly interesting (Padua et al., 2006). Based on these results, a couple of questions are raised. First, was the lack of change in leg stiffness a result of the fatiguing protocol targeting the certain muscles? In other words, squats was used as the fatiguing exercise in the Padua and colleagues (2006) study, but squats predominantly target the muscles about the knee and previous research has determined that hopping leg stiffness is mostly affected by the muscles about the ankle (Farley & Morgenroth, 1999). Therefore, the lack of change in the leg stiffness might be due to lack of fatigue in the muscles about the ankle. The second question being, since leg stiffness stayed the same, did the ankle, knee, and hip joint stiffness also not change or did the joint stiffness change to maintain the overall hopping leg stiffness? These are the two questions that drive the aims of this current study.

The mechanisms of the control of leg stiffness are not well known, especially the changes in leg stiffness as a response to neuromuscular fatigue. The previous literature on the modulation of neuromuscular fatigue on leg stiffness during hopping is lacking, and the influence of the sources of neuromuscular fatigue on leg stiffness has not previously been examined. Previous studies only examined the overall effects of fatigue without delving into the contributions of neural and muscular fatigue on the biomechanical adaptations. Understanding the origins and contributions of the various components of fatigue are key to providing insight into the mechanisms causing the biomechanical adaptations.

1.4. Implications of Study

Many of the mechanisms behind the control of leg stiffness and the basis of neuromuscular fatigue are still not well known. Through the use of a simple spring-mass model and a single-leg hopping task, this proposed study aims to provide greater insight into how neuromuscular fatigue of the muscles about the ankle alters leg stiffness and lower body joint stiffness.

The use of a simple spring-mass model was utilized to establish a general leg stiffness response to neuromuscular fatigue, and the use of joint stiffness was utilized to explain the changes in leg stiffness. Single-leg hopping was utilized to allow us to determine the role of neural fatigue by minimizing the physical demands on one leg and the role of muscular fatigue with the other leg performing the fatiguing exercise. The modulation of leg stiffness and joint stiffness has many implications in motor control to help understand what biomechanical variables the human body controls to maintain performance when experiencing physiological changes. By understanding the body's motor control response to the components of fatigue, exercise training and recovery programs can be altered to decrease fatigue and increase recovery time.

1.5. Specific Aims & Hypotheses

In this study, we will clarify the physiological source of fatigue-related changes in leg stiffness. The study by Padua and colleagues (2006) determined that there was no change in leg stiffness as a result of fatigue, but that study did not investigate whether or not ankle, knee, or hip joint stiffness changes to maintain the same overall leg stiffness. Neuromuscular fatigue can have many causes, which

include neural and muscular components to fatigue, and previous literature has yet to examine the role of each component on modulating leg stiffness. The main goal of this study is to determine how neuromuscular fatigue affects leg stiffness and to determine whether neural or muscular fatigue contributes to the changes in spring like behavior of human locomotion. Based on previous hopping and fatigue research, we hypothesized:

(i) Leg stiffness would increase in the post-fatigue hopping conditions from the pre-fatigue hopping conditions for the exercised leg and non-exercised leg, but leg stiffness would increase to a greater extent for the exercised leg than the non-exercised leg.

(ii) The increase in leg stiffness with fatigue would result from increases in ankle, knee, and hip joint stiffness.

CHAPTER 2: METHODS

2.1. Subjects and Sampling

An a priori power analysis was performed using G*Power and the program determined that 14 participants was the minimum number of subjects necessary to achieve a moderate effect size (Faul et al., 2007). A limitation to the a priori power analysis that was performed was that a moderate effect size was chosen primarily because the moderate effect size allowed us to maintain a reasonable sample size, and not based on previous studies. As a result, 15 healthy adults (8 female, 7 male; age 21.5±2.7 years; 65.7±10.3 kg; 1.7±0.12 m) were recruited for this study. The local ethics committee of the University of Maryland, College Park Institutional Review Board approved this study and participants provided written informed consent prior to participating. Men and women both participated in this study because prior research has shown no statistically significant difference between men and women when examining leg stiffness in response to fatigue (Padua, 2006). Participants were recruited through the University of Maryland College Park listserv and were contacted through e-mail.

Individuals with a history of lower limb injuries or trauma, or neurological or orthopedic issues were excluded from the study. Participants were asked about limb dominance because leg dominance has been shown to affect muscle strength

(Balogun and Onigbinde, 1992). Limb dominance was determined by asking the participants the following questions: "which leg is your preferred kicking leg?" and "which hand is your preferred writing hand?" In this study, the non-dominant limb was defined as the leg that was primarily used for stability, while the dominant limb was defined as the limb used for more precise motor tasks. Limb dominance was noted, but was not utilized as an exclusion criterion because the treatment leg was randomly assigned and the study followed a within-subject design.

2.2. Design and Variables

Independent Variables. This study had two independent variables, SIDE and FATIGUE. The first independent variable was SIDE, which consisted of two levels: exercised leg and non-exercised leg. The exercised leg was the leg that performed a fatiguing protocol, while the non-exercised leg rested. The second independent variable was FATIGUE, which consisted of two levels: pre-fatigue and post-fatigue. The pre-fatigue conditions consisted of the hops before one of the legs performed the fatiguing protocol, while the post-fatigue conditions consisted of the hops after one of the legs performed the fatiguing protocol.

Dependent Variables. The main dependent variables were leg stiffness and joint stiffness. Leg stiffness was derived from Hooke's Law:

$$F = \left| k \Delta X \right| \qquad \text{(Eq. 1)}$$

where F is the force needed to compress the spring stiffness, k, by some distance ΔX.

Hooke's Law can be rewritten with the same variables as:

$$k = \left| \frac{F}{\Delta X} \right| \qquad \text{(Eq. 2)}$$

Based on Hooke's Law, leg stiffness (k_{leg}) was calculated as:

$$k_{leg} = \left| \frac{\Delta VGRF}{\Delta COM} \right| \qquad \text{(Eq. 3)}$$

where $\Delta VGRF$ was the change in vertical ground reaction force (VGRF) and ΔCOM was the vertical center of mass (COM) displacement. $\Delta VGRF$ was calculated as the ground contact phase maximum VGRF and ΔCOM was calculated as the ground contact phase maximum vertical COM displacement. COM displacement, $d_y(t)$, was calculated using the double integration of the VGRF, $F(t)$:

$$d_y(t) = \int \left[\int \frac{F(t) - mg}{m} dt \right] dt + v_0 t + r_0 \qquad \text{(Eq. 4)}$$

where m the participant's total body mass, g is the acceleration due to gravity, and $v_0 t$ and r_0 are the integration constants relative to the velocity and position of the COM, respectively, as previously explained (Blickhan, 1989; Blum et al., 2009; McMahon et al., 1987; Ranavolo et al., 2008).

Joint stiffness (k_{joint}), was calculated at the ankle, knee, and hip as:

$$k_{joint} = \left| \frac{\Delta M}{\Delta \Theta} \right| \qquad \text{(Eq. 5)}$$

where $\Delta \Theta$ is the joint angular displacement from the beginning of ground contact until the instant of maximum joint flexion, and ΔM is the change in joint moment during this same time.

All stiffness variables were calculated utilizing absolute values so that leg and joint stiffness could be reported as positive values to allow for easier comparisons of the changes in stiffness. All stiffness values were only calculated for the eccentric portion of the hop, which was the beginning of the ground-contact phase to the instant of maximal flexion of the joint (Farley et al., 1998).

Experimental Design. This study utilized a within-subject design, which was chosen for this study because each subject served as his or her own control. This is important when examining leg stiffness because each individual will have different physical properties (height, weight, leg length), which can greatly change leg stiffness and cause inconsistencies when compared among participants. By employing a within-subject design, we were able to calculate the change in leg stiffness for each participant and the difference in leg stiffness when comparing the left and right legs.

The study design followed this framework (Figure 2). Participants came into the Neuromechanics Lab without any neuromuscular fatigue. Once in the lab, participants underwent the pre-fatigue hopping conditions and were prompted to perform single-leg hops on each leg. The pre-fatigue hopping established a baseline for participants' leg-stiffness. This current study examined leg stiffness based the following assumptions (Figure 2). During the pre-fatigue conditions, theoretically, participants were not fatigued (no neural or muscular fatigue) because the participants have yet to undergo the fatiguing protocol. After the pre-fatigue hopping conditions, the participant performed the fatiguing protocol on a randomly selected leg. The fatiguing protocol allowed us to have an exercised leg with a combination of neural and muscular fatigue, and a non-exercised leg with only neural fatigue and without

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muscular fatigue. Once the fatiguing protocol was complete, participants performed the post-fatigue single-leg hopping conditions and participants hopped on each leg following the same protocol as the pre-fatigue hopping conditions. The post-fatigue hopping allowed us to calculate the change in leg stiffness as a result of fatigue in both the exercised and non-exercised leg.

Pre-Fatigue

Central Fatigue	No		No
Peripheral Fatigue	No		No

Fatiguing Protocol	Non-Exercised		Exercised

Post-Fatigue

Central Fatigue	Yes		Yes
Peripheral Fatigue	No		Yes
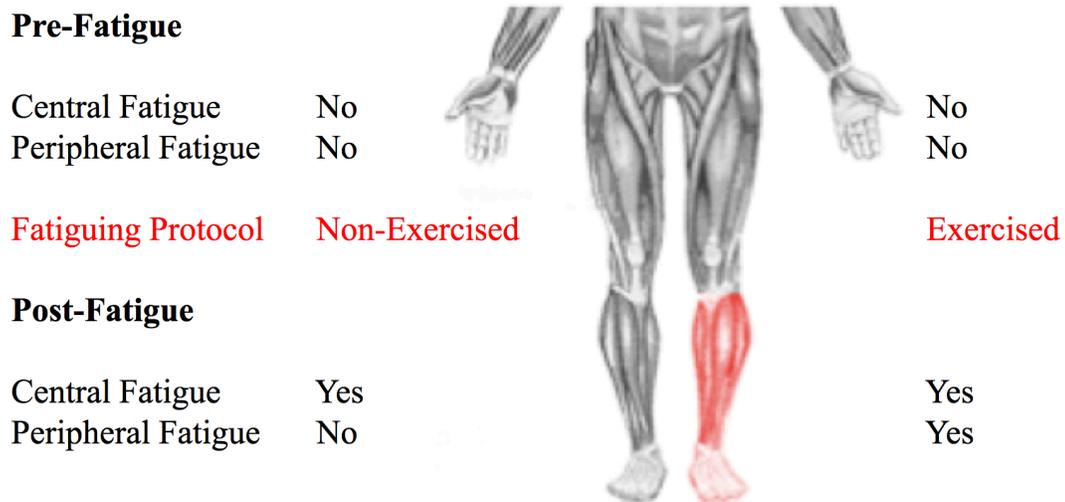
Figure 2. Conceptual framework of the experimental design pre-fatigue and post-fatigue assumptions.

2.3. Instrumentation

The dependent variables in the study consisted of leg stiffness and joint stiffness. To calculate leg stiffness, the vertical ground reaction (VGRF) force was measured using a Kistler force platform (Kistler Holding AG, CH). Force plates are widely used in biomechanics to obtain ground reaction forces and the reliability and validity have been confirmed (Walsh et al., 2006).

To calculate joint stiffness, 3-D kinematic motion analysis using Vicon motion capture cameras (Vicon Motion Systems Ltd, United Kingdom) were used to

track human movements, and joint moments were calculated using 6-degree of freedom (DOF) kinematic analysis and iterative Newton-Euler inverse dynamics using Visual3D software (C-Motion, Inc., USA). All data were filtered using a 4th order dual-pass Butterworth filter with cutoff frequencies set at 50 Hz for kinetic data and 10Hz for kinematic data prior to any calculations.

For each subject, the pelvis and both left and right thigh, shank, and foot segments were linked to create a lower body model based on a static standing calibration trial. The ankle joint centers were calculated based on the midpoint between the lateral and medial malleolus of each limb. The knee joint centers were calculated based on the midpoint between the lateral and medial epicondyle of each limb. The hip joint centers were calculated based on the Coda pelvis model within Visual3D (C-Motion, Inc., USA), which are based on the positions of the left and right anterior superior iliac spine (ASIS), and the left and right posterior superior iliac spine (PSIS). Joint angles were calculated utilizing an XYZ cardan sequence. Joint angles and moments in the sagittal plane were utilized to calculate joint stiffness.

Muscle activation, electromyography (EMG) data was collected with the Delsys Trigno^{TM} EMG system (Delsys Inc., USA) and synced with Vicon (Vicon Motion Systems Ltd, United Kingdom) for future analysis.

2.4. Data Collection Procedures

Consent Process. Data collection for this study consisted of a 3-hour visit to the Neuromechanics Laboratory. Upon arrival to the lab, the participant was given a consent form that was approved by the University of Maryland Institutional Review Board. A physical copy of the consent form was presented to the participant and the

consent form was verbally explained to the participant. The experimental protocol was reviewed with the participant and written consent was obtained.

Subject Preparation. Prior to the start of the experiment, the participant changed into compression shorts and shirt provided by the Neuromechanics Lab. Participants were asked to remove their shoes and socks to keep the surface stiffness the same for all subjects, which has been found to affect leg stiffness (Ferris & Farley, 1997). Participants' height and weight were measured. Participants were then asked about previous and current neuromuscular, neurological, and orthopaedic injuries and issues. Hand and leg dominance was determined with the following questions: "which hand is your preferred writing hand?" and "which leg is your preferred kicking leg?" Participants were then prepped with alcohol pads and shaved, if needed, in preparation for EMG placement.

For muscle activation data collection, the Delsys Trigno^{TM} Wireless EMG System (Delsys Inc., USA) was used and 14 EMG surface electrode sensors (7 sensors per leg) were adhered onto the lower body. EMG sensors were adhered with double-sided tape provided by Delsys (Delsys Inc., USA). Surface EMG was collected for the rectus femoris, vastus lateralis, biceps femoris, semitendinosus, tibialis anterior, medial gastrocnemius, and soleus for both legs. EMG sensor locations were placed as accurately as possible based on the recommendations from SENIAM (http://www.seniam.org/). After EMG sensors were adhered, signals were tested with specific movements to elicit the desired muscle activation.

After the placement of the EMG sensors were complete, the participants were be prepped for kinematic data collection. Retro-reflective markers were used for data

collection so that a 3-D virtual image of body could be recreated. The kinematic marker set consisted of 40 markers that were adhered to the skin with double-sided tape. Anatomical landmarks were marked with an alcohol based permanent marker and then reflective markers were placed on the anatomical landmarks (Figure 3).
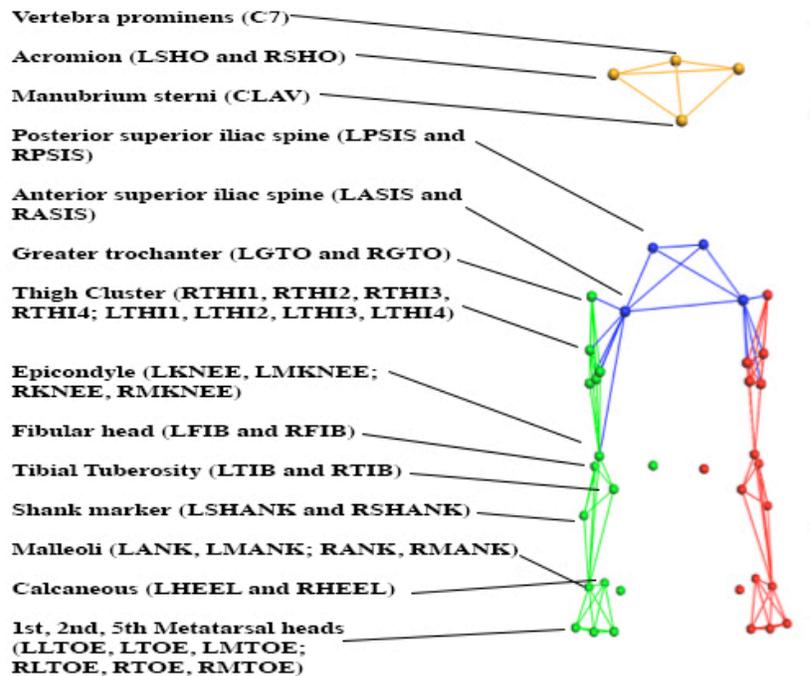
Figure 3. Marker set used in Vicon Nexus (Vicon Motion Systems Ltd, United Kingdom) for kinematic motion analysis. Abbreviations that start with "L" denote the left side and abbreviations that start with an "R" denote the right side.

Data Collection Pre-Test. Proceeding subject preparation, a static calibration trial was taken. Participants were asked to stand comfortably on 2 force platforms (the left foot on one platform and the right foot on another) with both arms raised out to the side in a "T-pose." The static trial was utilized for kinematic and kinetic calibration.

After the static trial, the pre-test protocol consisted of having the subject perform two tasks (Tasks A and B) barefoot and with no rest time between tasks, sets, and reps.

- Task A – Participants performed 2 maximum force counter movement jumps with each foot on its own force plate. Participants were instructed to place arms across their chests and hands on their shoulders.

- Task B – Participants performed single-leg hopping while matching a digital metronome set at 2Hz. Participants were instructed to try perform all the hopping on one force platform, and to perform the hops with arms across the subjects' chests and hands on their shoulders. Participants hopped on each leg for 30 s and were instructed when to start and stop. Participants performed the single-leg hopping for both legs.

No rest time was allotted between tasks so that the neuromuscular fatigue did not wear off prior to the post-fatigue trials. All subjects performed Task A and then Task B for the pretest. For Task B, the single-leg hopping tasks, the side (left or right) order was randomized for all subjects.

During all of the tasks, 3-D kinetic data was collected with Kistler force platforms (Kistler Holding AG, CH) at a data collection frequency of 1,000Hz; muscle activation data were collected with 14 Delsys Trigno^{TM} wireless EMG surface electrodes (Delsys Inc., US) at a data collection frequency of 1,000Hz; and 3-D kinematic data were collected with a 13 camera Vicon motion capture system (Vicon Motion Systems Ltd, United Kingdom) at a data collection frequency of 200Hz.

Fatiguing Protocol. After the pre-fatigue conditions were complete, participants performed the ankle fatiguing protocol. Prior to administering the fatiguing protocol, participants were advised that they would experience soreness in their lower leg, but if the participants experienced any pain, the testing would be stopped immediately. Participants were randomly assigned which leg was going to perform the exercise, while the other leg rested.

The fatiguing protocol consisted of 15 sets of 10 repetitions of single-leg calf raises, with 30 seconds of rest in between sets. This protocol was similar to the protocol used by (Miyamoto et al., 2011). Subjects were instructed to perform each calf raise repetition with a smooth and controlled motion, and to hold the maximum plantarflexion at the top of the raise for 1 s. The same administrator counted each repetition and 1 s maximum plantarflexion hold for all sets for every subjects. Calf raises were performed on an elevated surface to allow the exercised leg to perform full ankle range of motion dorsiflexion and plantarflexion, while the non-exercised leg hung off the side.

The single-leg calf raises were performed facing a wall with the participants' palms placed against the wall to maintain balance. Participants were instructed not to lean against the wall and asked to only exert minimal force on the wall to maintain balance. After each set of calf raises participants were verbally encouraged to elicit maximal effort and reminded to maintain proper form.

Data Collection Post-Test. Directly after the participants completed the fatiguing protocol, participants performed the post-fatigue hopping trials. The post-fatigue conditions consisted of exactly the same tasks (Tasks A and B) as the pre-

fatigue conditions. The only difference between the post-fatigue and the pre-fatigue conditions was that for the post-test, Task B, single-leg hopping for 30-s to a hopping frequency of 2Hz, was performed before Task A, maximal counter movement jumps. Similar to the pre-test, the order of the hopping legs was randomly assigned for all subjects.

2.5. Statistical Analysis

Two-way repeated measures ANOVA ($p < 0.05$), with two factors, SIDE (exercised and non-exercised leg) and FATIGUE (pre- and post-fatigue) tested for main effect changes in stiffness. When significant main effects in the presence of significant interaction effects were found, post-hoc testing was performed using paired samples t-tests.

The amount of change in leg stiffness was calculated as percent change relative to the pre-fatigue condition. The effect size of the percent change was calculated based on calculating Cohen's D utilizing the pooled standard deviation (Cohen, 1988; Rosnow & Rosenthal, 1996), which was calculated as:

$$d = \frac{M_1 - M_2}{s_{pooled}} \qquad \text{(Eq. 6)}$$

where M_1 is the mean of one group and M_2 is the mean of the second group and s_{pooled} is the pooled standard deviation, which is calculated as:

$$s_{pooled} = \sqrt{\frac{\left(s_1^2 + s_2^2\right)}{2}} \qquad \text{(Eq. 7)}$$

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where s_1 is the standard deviation of one group and s_2 is the standard deviation of the

second group.

CHAPTER 3: RESULTS

3.1. Leg Stiffness

This study modeled single-leg hopping as a simple linear spring attached to a point mass utilizing the spring-mass model (Figure 1). Leg stiffness was calculated for the leg compression of the ground contact phase (Eq. 3). The results from this study indicated that the overall hopping behavior fit the spring-mass model because the maximum VGRF occurred around the maximum COM displacement, and the relationship between VGRF and COM displacement is relatively linear (Figure 4).

For leg stiffness, the two-way repeated measures ANOVA revealed a significant main effect for FATIGUE ($p = 0.009$), but no significant difference for SIDE ($p = 0.822$) and interaction SIDE x FATIGUE ($p = 0.072$). Post-hoc revealed that for the exercised leg, leg stiffness significantly decrease by $3.22 \pm 3.41\%$ ($p = 0.002$) from the pre-fatigue to the post-fatigue conditions, with a moderate effect size of 0.32; however, for the non-exercised leg, leg stiffness did not significantly change ($p = 0.233$) between the pre-fatigue and post-fatigue conditions (Figure 5).
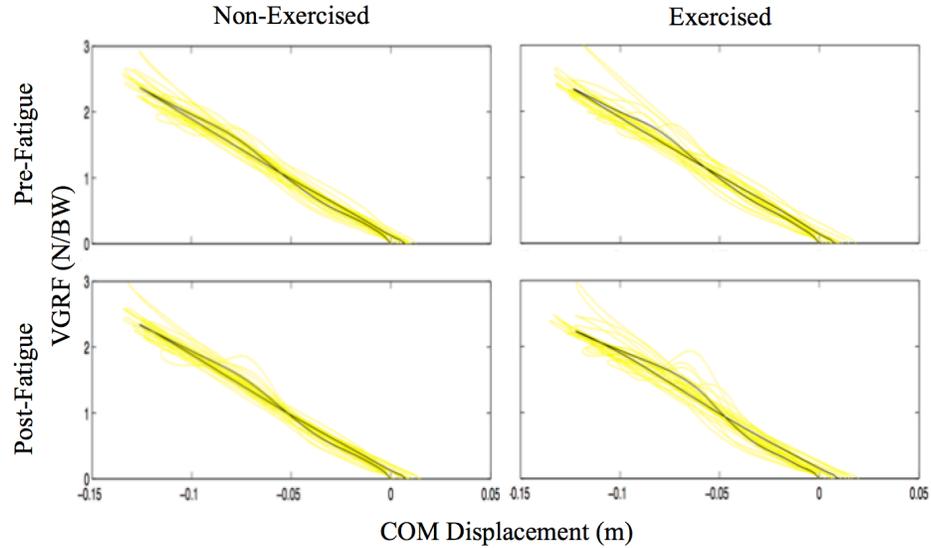
Figure 4. Vertical ground reaction force (VGRF) standardized to body weight (N/BW) versus center of mass (COM) displacement during the ground contact phase of hopping. The initial COM displacement was shifted to start at 0 m. Yellow lines are individual subject means for each condition. Black lines represent the mean of all subjects for each condition. Plots on the top row depict the pre-fatigue condition, while plots on the bottom row depict the post-fatigue condition. Plots on the left depict the non-exercised leg, while plots on the right depict the exercised leg.
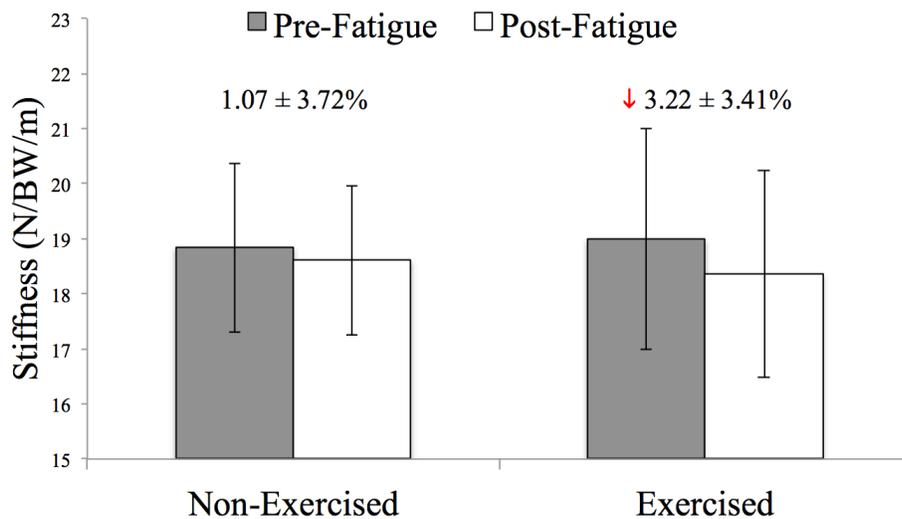
Figure 5. Mean and standard deviation leg stiffness (k_{leg}) pre-fatigue (gray) and post-fatigue (white) for non-treatment (left) and treatment (right) legs. Mean percent change and percent change standard deviation are displayed for the exercised and non-exercised legs. ↓ denotes significant decrease in leg stiffness from pre-fatigue to post-fatigue conditions.

Leg stiffness was calculated utilizing the change in VGRF and the change in COM. The change in VGRF was calculated as the difference between maximum VGRF and the instant before the initial ground contact VGRF, which was 0 N. The change in COM displacement was calculated as the difference between the maximum COM displacement and the initial ground contact COM displacement.

Variables	Non-Exercised Leg (Percent Change)	Exercised Leg (Percent Change)
$\Delta VGRF$	$0.54 \pm 5.32\%$ (p = 0.543)	↓$3.92 \pm 6.44\%$ (p = 0.020)
ΔCOM	$0.52 \pm 3.25\%$ (p= 0.606)	$0.80 \pm 4.03\%$ (p = 0.380)

Table 1. Variables utilized to calculate leg stiffness. Values are displayed as percent change ± standard deviation of the percent change. $\Delta VGRF$ and ΔCOM represents the change in vertical ground reaction force and center of mass, respectively. Percent change was calculated as the difference of the pre-fatigue and post-fatigue conditions relative to the pre-fatigue condition. P-values from the post-hoc paired t-tests are provided in parentheses. ↓ denotes significant decrease from pre-fatigue to post-fatigue conditions.

The two-way repeated measures ANOVA for the change in VGRF revealed no significant main effects for SIDE (p = 0.054) and FATIGUE (p = 0.068), but did reveal a significant interaction SIDE x FATIGUE effect (p = 0.036). Post-hoc revealed a significant decrease by $3.92 \pm 6.44\%$ (p = 0.020) for the exercised leg from the pre-fatigue to the post-fatigue conditions with a moderate effect size of 0.34, but there was no significant difference for the non-exercised leg (p = 0.543; Table 1). In other words, the change in VGRF significantly decreased as a result of neuromuscular fatigue for the exercised leg (Table 1).

Analysis of the change in COM displacement revealed a significant main effect for SIDE (p = 0.006), but no significant main effect for FATIGUE (p = 0.759)

and interaction SIDE x FATIGUE ($p = 0.085$); however, post-hoc revealed that there was no significant difference between the exercised ($p = 0.112$) and non-exercised legs ($p = 0.323$) for the pre-exercise and post-exercise conditions. Simply put, there was no significant change in COM displacement as a result of neuromuscular fatigue (Table 1).

Overall, leg stiffness decreased from the pre-fatigue to the post-fatigue condition for the exercised leg only, and leg stiffness did not change for the non-exercised leg (Figure 5). The decrease in leg stiffness in the exercised leg was a result of the significant decrease of the change in VGRF, while the change in COM did not significantly change (Table 1). These results indicate that leg stiffness may have decreased as a response to muscular fatigue experienced by the exercised leg, but leg stiffness did not respond to the neural fatigue experienced by the non-exercised leg; therefore, our first hypothesis that leg stiffness would increase as a result of muscular and neural fatigue for both the exercised and non-exercised leg was not supported.

3.2. Joint Stiffness

The joints of the lower body were modeled as torsion springs. Joint stiffness was calculated as the ratio of the maximal moment and the change in joint angle along the sagittal plane for the ground contact phase. The results of the study indicate that the behavior of the ankle joint (Figure 6) and knee joint (Figure 7) can be modeled as torsion springs because the maximum moment occurred around the maximum joint angle displacement, and the relationship between joint moment and joint angular displacement is relatively linear. Unlike the ankle and knee joints, the

hip joint (Figure 8) did not behave similarly to that of a torsion spring because the maximum moment did not occur around the maximum joint angle displacement, and the relationship between the joint moment and joint angle was non-linear. Since the hip joint did not behave similarly to that of a torsion spring, the hip joint results were not included because the hip stiffness value would not accurately reflect the behavior of the hip joint.
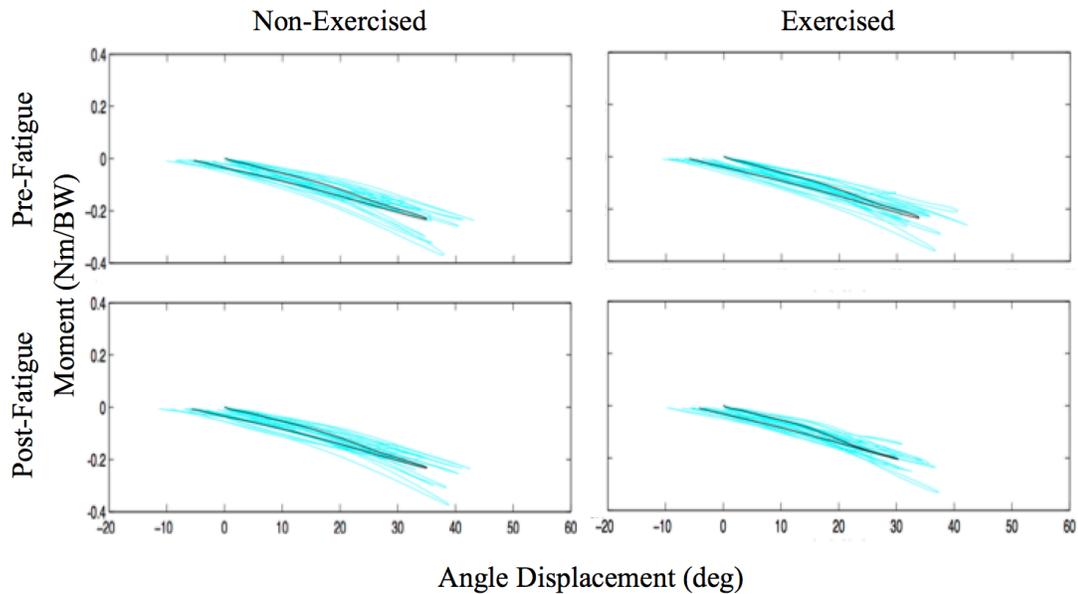
Figure 6. Ankle joint moment standardized to body weight (Nm/BW) versus change in ankle angle (deg) for the ground contact phase of hopping. The initial ankle angle was shifted to start at 0 deg. Blue lines are individual subject means for each condition. Black lines represent the mean of all subjects for each condition. Plots on the top row depict the pre-fatigue condition, while plots on the bottom row depict the post-fatigue condition. Plots on the left depict the non-exercised leg, while plots on the right depict the exercised leg.
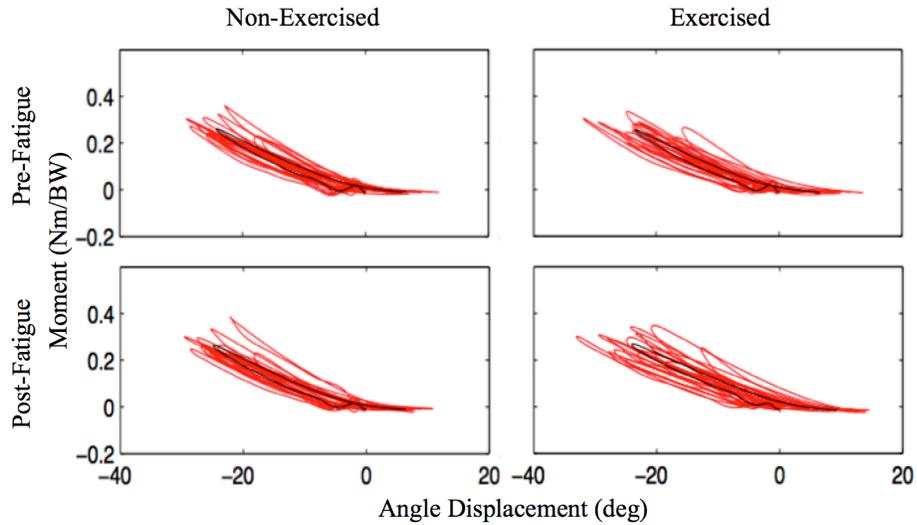
Figure 7. Knee joint moment standardized to body weight (Nm/BW) versus change in knee angle (deg) for the ground contact phase of hopping. The initial knee angle was shifted to start at 0 deg. Red lines are individual subject means for each condition. Black lines represent the mean of all subjects for each condition. Plots on the top row depict the pre-fatigue condition, while plots on the bottom row depict the post-fatigue condition. Plots on the left depict the non-exercised leg, while plots on the right depict the exercised leg.
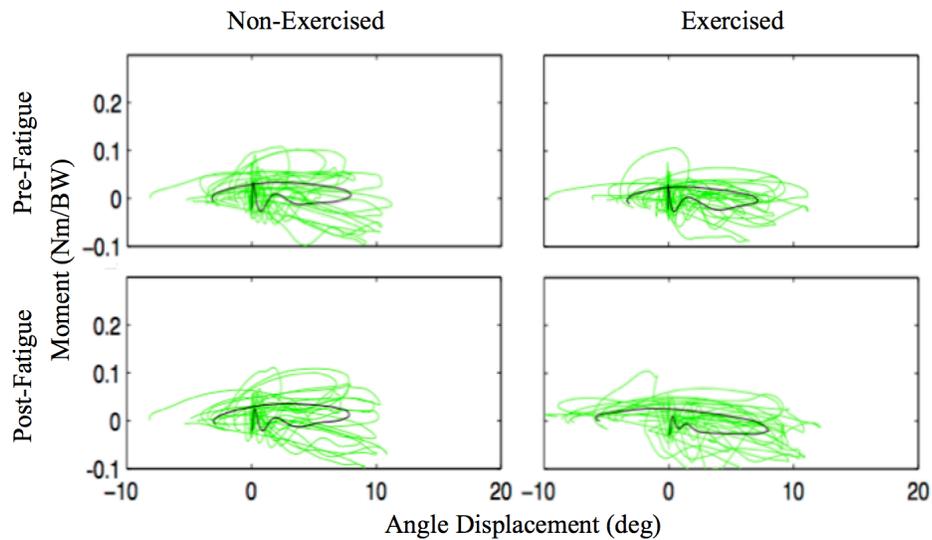
Figure 8. Hip joint moment standardized to body weight (Nm/BW) versus change in hip angle for the ground contact phase of hopping. The initial knee angle was shifted to start at 0 deg. Green lines are individual subject means for each condition. Black lines represent the mean of all subjects for each condition. Plots on the top row depict the pre-fatigue condition, while plots on the bottom row depict the post-fatigue condition. Plots on the left depict the non-exercised leg, while plots on the right depict the exercised leg.

Joint stiffness was calculated based on the ratio between the change in joint moment and the change in joint angle (Eq. 5). The change in joint moment was calculated as the difference between maximum joint moment and the initial ground contact joint moment. The change in COM displacement was calculated as the difference between the maximum joint angle displacement and the initial ground contact joint angle.

Ankle Stiffness. Two-way repeated measures ANOVA revealed that there were no significant main effect differences for SIDE ($p = 0.492$), FATIGUE ($p = 0.466$), and interaction SIDE x FATIGUE ($p = 0.310$). In other words, there were no significant changes in ankle joint stiffness in either the exercised or non-exercised leg (Figure 9). Based on these results, neuromuscular fatigue did not significantly affect the ankle joint stiffness.
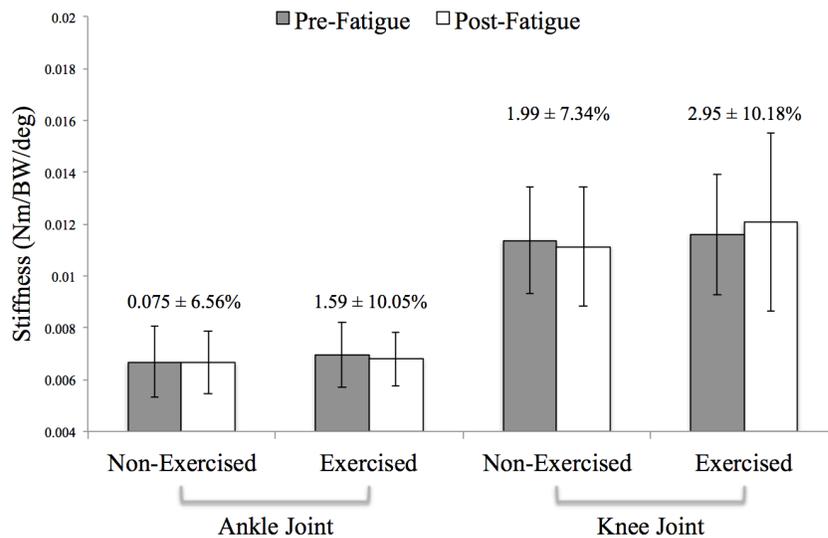
Figure 9. Mean and standard deviation ankle joint stiffness and knee joint stiffness pre-fatigue (gray) and post-fatigue (white) for exercised and non-exercised legs. Mean percent change and percent change standard deviation are displayed for the exercised and non-exercised legs. There were no statistically significant changes in joint stiffness.

Although ankle joint stiffness did not reveal any significant effects, there were significant changes for the two variables utilized to calculate ankle stiffness, ankle joint moment and ankle joint angle (Table 2). When examining ankle joint moment there was no significant main effect for SIDE ($p = 0.248$), but there were significant main effect for FATIGUE ($p = 0.049$) and interaction effect for SIDE x FATIGUE ($p = 0.013$). Post-hoc analysis revealed that ankle joint moment significantly decreased by $10.73 \pm 16.68\%$ ($p = 0.023$), with a moderate effect size of 0.58 for the exercised leg, but there was no significant change for the non-exercised leg ($p = 0.994$; Table 2). In other words, ankle joint moment significantly decreased from the pre-fatigue to post-fatigue conditions in the exercised leg, but not the non-exercised leg (Table 2).

Variables	Non-Exercised Leg (Percent Change)	Exercised Leg (Percent Change)
ΔM_{ankle}	$0.38 \pm 6.81\%$ ($p = 0.994$)	↓$10.73 \pm 16.68\%$ ($p = 0.023$)
$\Delta \Theta_{ankle}$	$0.36 \pm 3.74\%$ ($p = 0.686$)	↓$9.88 \pm 11.32\%$ ($p = 0.006$)
ΔM_{knee}	$0.54 \pm 7.38\%$ ($p = 0.866$)	$5.45 \pm 17.31\%$ ($p = 0.243$)
$\Delta \Theta_{knee}$	$2.80 \pm 6.68\%$ ($p = 0.103$)	$2.44 \pm 11.94\%$ ($p = 0.374$)

Table 2. Variables utilized to calculate joint stiffness. Values are displayed as percent change ± standard deviation of the percent change. ΔM and $\Delta \Theta$ represent change in joint moment and change in joint angle, respectively. Subscripts represent the joint analyzed. Percent change was calculated as the difference of the pre-fatigue and post-fatigue conditions relative to the pre-fatigue condition. P-values from the post-hoc paired t-tests are provided in parentheses. ↓ denotes significant decrease from pre-fatigue to post-fatigue conditions.

Analysis of the change in ankle angle displacement revealed that there were main effects for SIDE ($p = 0.001$) and FATIGUE ($p = 0.008$), and interaction effect for SIDE x FATIGUE ($p = 0.007$). Post-hoc analysis determined that ankle joint

angle significantly decreased from the pre-fatigue to post-fatigue conditions by $9.88 \pm 11.32\%$ (p = 0.006), with a large effect size of 0.81 for the exercised leg, but there was no significant change for the non-exercised leg (p = 0.686; Table 2). The interaction effect was a result of the post-fatigue joint angles for the non-exercised and exercised legs being significantly different (p < 0.001), while the pre-fatigue joint angles for the non-exercised and exercised legs not being significantly different (p = 0.153). Simply put, ankle joint angle significantly decreased from the pre-fatigue to post-fatigue hopping conditions for the exercised leg, but not for the non-exercised leg (Table 2).

Overall ankle joint stiffness did not significantly change when comparing pre- and post-fatigue conditions, but there were significant changes in ankle joint moment and ankle joint angle when comparing pre- and post-fatigue conditions (Table 2). These results suggest that neuromuscular fatigue (neural or muscular) did not affect the non-exercised leg, but neuromuscular fatigue, specifically muscular fatigue, did significantly decrease ankle joint moment and ankle joint angle. When examining the percent change of ankle joint moment and ankle joint angle from the pre-fatigue to post-fatigue conditions in the exercised leg, both the ankle joint moment and ankle joint angle decreased by a similar amount, $10.73 \pm 16.68\%$ and $9.88 \pm 11.32\%$, respectively (Table 2). As a result, ankle joint stiffness was not affected by neuromuscular fatigue because the ratio of ankle joint moment to ankle joint angle does not change even though both ankle joint moment and ankle joint angle are significantly affected by neuromuscular fatigue.

Knee Stiffness. Two-way repeated measures ANOVA revealed no significant difference for SIDE (p = 0.295), FATIGUE (p = 0.445), and interaction SIDE x FATIGUE (p = 0.204). In other words, there were no significant changes in knee joint stiffness in either the exercised or non-exercised leg. Based on these results, fatigue (neural and muscular) did not significantly affect the knee joint stiffness.

The variables utilized to calculate knee joint stiffness are the change in knee joint moment and change in knee joint angle. Analysis of the knee joint moment revealed no significant main effects for SIDE (p = 0.939) and FATIGUE (0.297), and no significant interaction effects for SIDE x FATIGUE (p = 0.274). The results of the knee joint angle also indicate no significant main effects for SIDE (p = 0.201) and FATIGUE (p = 0.208), and no significant interaction effect for SIDE x FATIGUE (p = 0.955). Simply put, there were no statistically significant changes in the knee joint stiffness, knee joint moment, and knee joint angle.

Joint Stiffness. Joint stiffness was calculated during the ground contact phase, specifically during leg spring compression of the single-leg hop. Ankle stiffness did not change as a result of neural or muscular fatigue based on the results of the non-exercised and exercised leg, respectively (Figure 9); however, when subject to muscular fatigue, ankle stiffness in the exercised leg did not change because the change in ankle joint moment and the change in ankle joint angle both decreased by a similar percentage (Table 2). Knee joint stiffness, knee joint moment, and knee joint angle all did not change as a result of fatigue (Figure 9; Table 2). Hip joint stiffness, change in hip joint moment, and change in hip joint angle were not calculated because the hip joint did not behave like a torsion spring. Based on the joint stiffness

results, our second hypothesis, joint stiffness would increase as a result of neuromuscular fatigue, was not supported.

CHAPTER 4: DISCUSSION

4.1. Findings

The aim of this study was to determine the effects of neuromuscular fatigue, specifically the role of neural and muscular fatigue, on the spring-like behavior, leg stiffness and joint stiffness, of a single-leg hopping task. The first hypothesis was that leg stiffness would increase from the pre-fatigue to post-fatigue conditions in both the exercised leg and the non-exercised leg, but to a greater extent in the exercised leg. The second hypothesis tested was that the increase in leg stiffness was a result of an increase in ankle, knee, and hip joint stiffness from the pre-fatigue to post-fatigue conditions.

Based on the results, our hypotheses were not supported. The results indicate that leg stiffness significantly decreased in the exercised leg from the pre-fatigue to the post-fatigue conditions because the change in VGRF decreased by a greater amount than the change in COM displacement; however, leg stiffness did not change in the non-exercised leg from the pre-fatigue to post-fatigue conditions. Our results also show that ankle and knee joint stiffness did not change from the pre-fatigue to the post-fatigue conditions for both the exercised and non-exercised leg, while the hip joint could not be modeled as a torsion spring. A particularly interesting finding was that for the exercised leg ankle joint stiffness did not change from the pre-fatigue to

post-fatigue conditions because both the ankle joint moment and ankle joint angle decreased by the same percentage.

4.2. Leg Stiffness

The recent findings in our study differ from the two previous studies that have examined the effects of fatigue on leg stiffness in hopping (Moritani et al., 1990; Padua et al., 2006). One study found that leg stiffness increased as a result of fatigue (Moritani et al., 1990), while another study found that leg stiffness did not change as a result of fatigue (Padua et al., 2006).

The difference in findings among the studies can be a result of the differing protocols. The Moritani and colleagues (1990) study examined maximal height hopping for 60 s and found that leg stiffness increased from the hops at the beginning to hops at the end of the 60 s. The fatiguing protocol utilized focused primarily on fatiguing the ankles because participants were instructed to minimize knee bending (Moritani et al., 1990). The findings that leg stiffness increased is most likely a result of the maximum height of the post-fatigue hops decreasing. The decrease in maximum height would cause leg stiffness to decrease because the change in center of mass displacement would decrease with a decrease in hopping height.

On the other hand, Padua and colleagues (2006) found no significant change in leg stiffness before and after a fatiguing protocol that consisted of a series of weighted squats, which primarily fatigued the muscles about the knee. The pre- and post-fatigue hopping frequency were controlled in that participants performed hops at their preferred hopping frequency and a hopping frequency set at 3 Hz (Padua et al.,

2006). The findings that leg stiffness did not change as a result of fatigue seem to be a result of the fatiguing protocol targeting muscles about the knee that might not greatly affect hopping behavior, while not targeting the muscles about the ankle that predominantly affect hopping behavior.

Both of the previous studies differed from our current study in terms of fatiguing protocol and hopping constraints. In our current study, we had participants hop at 2 Hz, and did not instruct them to hop as high as possible. Our fatiguing protocol targeted primarily the muscles about the ankle by utilizing a series of single-leg calf raises. The results of all three studies differ most likely because each study utilized different hopping and fatiguing protocols.

The Moritani and colleagues (1990) study was similar to our current study in that both of the studies utilize a primarily ankle fatiguing protocol, but the hopping tasks differed. The Moritani and colleagues (1990) study utilized maximal hopping, while our current study constrained the participants by setting the hopping frequency at 2 Hz. Based on the hopping method utilized in the Moritani and colleagues (1990) study, the findings that leg stiffness increased from fatigue seem to be a result of a decrease in maximum hopping height with fatigue. When compared to our current study, we did not ask participants to achieve maximal height for each hop; instead, we asked participants to hop to a set hopping frequency of 2 Hz. We utilized this protocol because by utilizing a consistent hopping frequency, the outcome goal of the pre- and post-fatigue protocols are consistent.

The results of our current study determined that leg stiffness decreased as a result of fatigue in the exercised leg because of a decrease in the max VGRF, while there was no significant change in COM displacement. From our results, we conclude that leg stiffness decreases due to neuromuscular fatigue, primarily from muscular fatigue with minimal contributions from neural fatigue. This suggests that the decrease in leg stiffness may be an attempt to soften landing impact, as postulated by Butler and colleagues (2003), while at the same time maintaining hopping performance. This notion is supported with studies that examined step-off landings from an elevated platform determined that decreased VGRF was associated with a less stiff landing technique, although stiffness was not measured (Devita & Skelly, 1992; Zhang et al., 2000). The softening of the landing impact may be a response to muscular fatigue to act as an attempt to decrease the loads experienced by the muscles.

4.3. Joint Stiffness

In our current study, we found that the ankle and knee joint stiffness did not change in either the exercised or non-exercised leg from the pre- to post-fatigue conditions. The results indicate that ankle stiffness did not change for the exercised leg because the change in ankle joint moment and the change in ankle joint angle both decreased by the same percentage; therefore, the ankle joint stiffness did not change. These findings suggest that fatigue, predominantly muscular fatigue, affects the ankle joint by decreasing the ankle joint moment, and as a response the CNS might be decreasing the ankle angle to maintain constant ankle stiffness, so that overall hopping performance can be maintained.

Knee joint stiffness also did not significantly change as a response to neuromuscular fatigue. The finding that knee joint stiffness did not change might be due to the high variability exhibited for the change in knee joint moment and change in knee joint angle.

In addition to ankle and knee joint stiffness, studies often calculate and report the hip joint stiffness (Farley & Morgenroth, 1999; Hobara et al., 2009); however, in our current study, hip joint stiffness was not reported because hip joint stiffness does not accurately represent the behavior of the hip joint. The maximum hip joint moment did not occur near the maximum hip joint angle displacement. These findings were similar to previous studies, which also did not report hip joint stiffness because the hip joint did not behave like a torsion spring (Farley et al., 1998; Kuitunen et al., 2011). Since our current study was unable to examine hip joint behavior by modeling the hip as a spring, the decrease in leg stiffness as a result of muscular fatigue may be a result of changes at the hip joint that we could not quantify. This notion is further supported because ankle and knee joint stiffness were not able to explain the decrease in leg stiffness as a result of muscular fatigue.

4.4. Neuromuscular Fatigue

The results from our study indicate that leg stiffness decreased as a result of neuromuscular fatigue, primarily resulting from muscular fatigue. The presence of neuromuscular fatigue was established by a change in leg stiffness for the non-exercised and exercised leg. Changes in the leg stiffness from the pre- to post-fatigue conditions for the non-exercised leg represented the effects of neural fatigue. On the

other hand, the exercised leg experienced both neural and muscular fatigue for the post-fatigue condition. Since there were significant leg stiffness changes for the exercised leg, but not for the non-exercised leg, then the exercised leg was used to represent the effects of muscular fatigue.

Our findings that muscular fatigue was the major contributor to fatigue are similar to that of a previous study (Kent-Braun, 1999). In addition, our findings that neural fatigue was not exhibited in the contralateral limb were similar to a study that examined maximum voluntary knee extensions (Arora et al., 2015); however, another study determined the contralateral limb experienced slight impairment in muscle activation (Todd et al., 2003). Based on these findings, there is a possibility that the contralateral limb may experience slight impairment, but this impairment may not be great enough to cause changes in overall task behavior.

4.5. Limitations

There are a couple limitations to our current study. One limitation of this current study was that our study modeled human hopping behavior and joint behavior during hopping as passive springs, but in actuality, the human body is much different. The human body is composed of joints, actively controlled muscles, dampened by soft tissue, and not a massless spring attached to a point mass. The utilization of leg stiffness and joint stiffness are widely contested as being inaccurate and over simplifying the system (Latash & Zatsiorsky, 1993); however, the human body is much too complicated to examine every detail, and often times a simple model is needed to reveal general relationships (Full & Koditschek, 1999).

Another limitation of this current study was that the hip joint did not exhibit spring-like behavior, and the change in leg stiffness was not explained by the ankle and knee joint stiffness, so perhaps the changes may be a result of changes in the hip joint behavior; therefore, further analysis of the joint behaviors is required to provide insight into the mechanism behind the changes in overall hopping behavior.

This current study aimed to examine the changes in hopping behavior as a result of neuromuscular fatigue, but the neural and muscular components of fatigue were not quantified; therefore, the exact contributions of the neural and muscular factors on overall fatigue are not known.

4.6. Future Research

This current study was able to establish that leg stiffness decreases as a result of neuromuscular fatigue, but joint stiffness was unable to explain the hopping behavior changes. This current study only focused on the eccentric phase (initial contact to maximal compression) of hopping, so perhaps also examining the concentric phase (maximal compression to take off) may help explain the hopping behavioral changes as a result of neuromuscular fatigue. Future research should focus on examining the mechanistic causes to the decrease in leg stiffness as a result of neuromuscular fatigue, perhaps through examination of electromyography or more in-depth kinetic and kinematic analysis. By examining changes in muscle activation, neural responses to fatigue can be examined.

CHAPTER 5: CONCLUSION

Overall, this study was able to establish that neuromuscular fatigue significantly affects overall hopping behavior, utilizing the spring-mass model. The results of this study indicate that fatigue, a combination of neural and muscular fatigue, significantly decreased leg stiffness; however, neural fatigue on its own does not appear to significantly affect leg stiffness. The role of neural fatigue seemed to be minimal because all stiffness variables did not change for the non-exercised leg when comparing pre- and post-fatigue conditions. These findings suggest that muscular fatigue is most likely the major contributor of neuromuscular fatigue that affects overall hopping behavior, while neural fatigue may play a minimal role. Although the general relationship that neuromuscular fatigue decreases leg stiffness was established, this study was unable to explain those changes by modeling the joints of the lower body (ankle, knee, and hip) as torsion springs.

The results indicate that ankle and knee joint stiffness did not change as a result of fatigue, neural (non-exercised leg) or muscular (exercised leg) fatigue, while the hip joint stiffness was not examined because the hip joint did not behave like a torsion spring. The ankle joint stiffness did not significantly change as a result of neuromuscular fatigue in the exercised leg because the change in joint moment and change in joint angle from the pre-fatigue to post-fatigue conditions both significantly decreased by the same amount. The ankle joint results indicate that muscular fatigue

most likely decreases the ankle joint moment and as a response the CNS decreases the ankle joint angle to maintain constant ankle joint stiffness. When examining the knee joint, the knee joint behavior does not significantly change as a response to neuromuscular fatigue.

In conclusion, the spring-mass model was able to establish that leg stiffness decreases as a result of neuromuscular fatigue primarily due to muscular fatigue; however, the changes in overall hopping behavior were not explained by joint stiffness. Based on these results, utilizing the spring-mass model can be useful for establishing basic relationships and changes in overall behavior, but the model may not be sensitive enough to provide insight into the mechanism causing the changes. Our findings also indicate that leg stiffness decreases as a response to muscular fatigue possibly in attempt to decrease the loads experienced by the muscles of the lower body.

REFERENCES

Arora, S., Budden, S., Byrne, J.M., & Behm, D.G., 2015. Effect of unilateral knee extensor fatigue on force and balance of the contralateral limb. European Journal of Applied Physiology, 115, 2177-2187.

Baker, A.J., Kostov, K.G., Miller, R.G., & Weiner, M.W., 1993. Slow force recovery after long-duration exercise: metabolic and activation factors in muscle fatigue. Journal of Applied Physiology, 74, 2294-2300.

Balogun, J.A., & Onigbinde, A.T., 1992. Hand and leg dominance: Do they really affect limb muscle strength?. Physiotherapy Theory and Practice, 8, 89-96.

Bernstein, N., 1967. The coordination and regulation of movements. Pergamon Press, Oxford.

Blickhan, R., 1989. The spring-mass model for running and hopping. Journal of Biomechanics, 22, 1217-1227.

Blum, Y., Lipfert, S.W., & Seyfarth, A., 2009. Effective leg stiffness in running. Journal of Biomechanics. 42, 2400-2405.

Bonnard, M., Sirin, A.V., Oddsson, L., & Thorstensson, A., 1994. Different strategies to compensate for the effects of fatigue revealed by neuromuscular adaptation process in humans. Neuroscience Letters, 166, 101-105.

Butler, R.J., Crowell, H.P. 3rd, & Davis, I.M., 2003. Lower extremity stiffness: implications for performance and injury. Clinical Biomechanics, 18, 511-517.

Cavagna, G.A., Heglund, N.C., & Taylor, C.R., 1977. Mechanical work in terrestrial locomotion: two basic mechanisms for minimizing energy expenditure. The American Journal of Physiology, 233, 243-261.

Cohen, J., 1988. Statistical power analysis for the behavior sciences. Lawrence Earlbaum Associates, New Jersey.

Dal Pupo, J., Dias, J.A., Gheller, R.G., Detanico, D., & dos Santos, S.G., 2013. Stiffness, intralimb coordination, and joint modulation during a continuous vertical jump test. Sports Biomechanics, 12, 259-271.

DeVita, P., & Skelly, W.A., 1992. Effect of landing stiffness on joint kinetics and energetics in the lower extremity. Medicine and Science in Sports and Exercise, 24, 108-115.

Di Giulio C., Daniele, F., & Tipton, C.M., 2006. Angelo Mosso and muscular fatigue: 116 years after the first Congress of Physiologists: IUPS commemoration. Advances in Physiology Education, 30, 51-57.

Dutto, D.J., & Smith, G.A., 2002. Changes in spring-mass characteristics during treadmill running to exhaustion. Medicine and Science in Sports and Exercise, 24, 1324-1331.

Edwards, R.H., Hill, D.K., Jones, D.A., & Merton, P.A., 1977. Fatigue of long duration in human skeletal muscle after exercise. Journal of Physiology, 272, 769-778.

Enoka, R.M., & Duchateau, J., 2008. Muscle fatigue: what, why and how it influences muscle function. The Journal of Physiology, 586, 11-23.

Enoka, R.M., & Stuart, D.G., 1985. The contribution of neuroscience to exercise studies. Federation Proceedings, 44, 2279-2285.

Enoka, R.M., & Stuart, D.G., 1992. Neurobiology of muscle fatigue. Journal of Applied Physiology, 72, 1631-1648.

Farley, C.T., Blickhan, R., Saito, J., & Taylor, C.R., 1991. Hopping frequency in humans: a test of how springs set stride frequency in bouncing gaits. Journal of Applied Physiology, 71, 2127-2132.

Farley, C.T., & González, O., 1996. Leg stiffness and stride frequency in human running. Journal of Biomehanics, 29, 181-186.

Farley, C.T., Houdijk, H.H., Van Strien, C., Louie, M., 1998. Mechanism of leg stiffness adjustment for hopping on surfaces of different stiffnesses. Journal of Applied Physiology, 85, 1044-1055.

Farley, C.T., & Morgenroth, D.C., 1999. Leg stiffness primarily depends on ankle stiffness during human hopping. Journal of Biomechanics, 32, 267-273.

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A., 2007. G*Power 3: A flexible statistical power analysis program for social, behavioral, and biomedical sciences. Behavior Research Methods, 39, 175.191.

Ferris, D.P., & Farley, C.T., 1997. Interaction of leg stiffness and surfaces stiffness during human hopping. Journal of Applied Physiology ,82, 15-22.

Fitzpatrick, R.C., Taylor, J.L., & McCloskey, D.I., 1992. Ankle stiffness of standing humans in response to imperceptible perturbation: reflex and task-dependent components. The Journal of Physiology, 454, 533-547.

Fourchet, F., Girard, O., Kelly, L., Horobeanu, C., & Millet, G.P., 2015. Changes in leg spring behaviour, plantar loading and foot mobility magnitude induced by an exhaustive treadmill run in adolescent middle-distance runners. Journal of Science and Medicine in Sport, 18, 199-203.

Full, R.J., & Koditschek, D.E., 1999. Templates and anchors: neuromechanical hypotheses of legged locomotion on land. The Journal of Experimental Biology, 202, 3325-3332.

Gandevia, S.C., Allen, G.M., Butler, J.E., & Taylor, J.L., 1996. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. The Journal of Physiology, 490, 529-536.

Hayes, P.R., & Caplan, N., 2014. Leg stiffness decreases during a run to exhaustion at the speed at $\dot{V}$O2max. European Journal of Sports Science, 14, 556-562.

Hobara, H., Inoue, K., & Kanosue, K., 2013. Effect of hopping frequency on bilateral differences in leg stiffness. Journal of Applied Biomechanics, 29, 55-60.

Hobara, H., Muraoka, T., Omuro, K., Gomi, K., Sakamoto, M., Inoue, K., & Kanosue, K., 2009. Knee stiffness is a major determinant of leg stiffness during maximal hopping. Journal of Biomechanics, 42, 1768-1771.

Hortobágyi, T., & DeVita, P., 2000. Muscle pre- and coactivity during downward stepping are associated with leg stiffness in aging. Journal of Electromyography and Kinesiology, 10, 117-126.

Kent-Braun, J.A., 1999. Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. European Journal of Applied Physiology and Occupational Physiology, 80, 57-63.

Kuitunen, S., Ogiso, K., & Komi, P.V., 2011. Leg and joint stiffness in human hopping. Scandinavian Journal of Medicine and Science in Sports, 21, 159-167.

Latash, M.L., & Zatsiorsky, V.M., 1993. Joint stiffness: myth or reality? Human Movement Science, 12, 653-692.

Liu, J.Z., Zhang, L., Yao, B., Sahgal, V., & Yue, G.H., 2005. Fatigue induced by intermittent maximal voluntary contractions is associated with significant losses in muscle output but limited reductions in functional MRI-measured brain activation level. Brain Research, 1040, 44-54.

McMahon, T.A., & Cheng, G.C., 1990. The mechanics of running: how does stiffness couple with speed?. Journal of Biomechanics, 23, 65-78.

McMahon, T.A., Valiant, G., & Frederick, E.C., 1987. Groucho running. Journal of Applied Physiology, 62, 2326-2337.

Miyamoto, N., Hirata, K., Mitsukawa, N., Yanai, T., & Kawakami, Y., 2011. Effect of pressure intensity of graduated elastic compression stocking on muscle fatigue following calf-raise exercise. Journal of Electromyography and Kinesiology, 21, 249-254.

Morin, J.B., Dalleau, G., Kyröläinen, H., Jeannin, T., & Belli, A., 2005. A simple method for measuring stiffness during running. Journal of Applied Biomechanics, 21, 167-180.

Morin, J.B., Tomazin, K., Samozino, P., Edouard, P., & Millet, G.Y., 2012. High-intensity sprint fatigue does not alter constant-submaximal velocity running mechanics and spring-mass behavior. European Journal of Applied Physiology, 112, 1419-1428.

Moritani, T., Oddson, L., & Thorstensson, A., 1990. Electromyographic evidence of selective fatigue during the eccentric phase of stretch/shortening cycles in

man. European Journal of Applied Physiology and Occupational Physiology, 60, 425-429.

Moritz, C.T., Greene, S.M., & Farley, C.T., 2004. Neuromuscular changes for hopping on a range of damped surfaces. Journal of Applied Physiology, 96, 1996-2004.

Padua, D.A., Arnold, B.L., Perrin, D.H., Gansneder, B.M., Carcia, C.R., & Granata, K.P., 2006. Journal of Athletic Training, 41, 294-304.

Rabita, G., Couturier, A., Dorel, S., Hausswirth, C., & Le Meur, Y., 2013. Changes in spring-mass behavior and muscle activity during an exhaustive run at $\dot{V}$O2max. Journal of Biomechanics, 46, 2011-2007.

Ranavolo, A., Don, R., Cacchio, A., Serrao, M., Paoloni, M., Mangone, M., & Santilli, V., 2008. Comparison between kinematic and kinetic methods for computing the vertical displacement of the center of mass during human hopping at different frequencies. Journal of Applied Biomechanics, 24, 271-279.

Rosnow, R.L., & Rosenthal, R., 1996. Computing contrasts, effect sizes, and counternulls on other people's published data: general procedures for research consumers. Pyschological Methods, 1, 331-340.

Taylor, J.L., Todd, G., & Gandevia, S.C., 2006. Evidence for a supraspinal contribution to human muscle fatigue. Clinical and Experimental Pharmacology and Physiology, 33, 400-405.

Todd, G., Petersen, N.T., Taylor, J.L., & Gandevia, S.C., 2003. The effect of a contralateral contraction on maximal voluntary activation and central fatigue in elbow flexor muscles, 150, 308-313.

Walsh, M.S., Ford, K.R., Bangen, K.J., Myer, G.D., Hewett, T.E., 2006. The validation of a portable force plate for measuring force-time data during jumping and landing tasks, Journal of Strength and Conditioning Research, 20, 730-734.

Woods, J.J., Furbush, R., & Bigland-Ritchie, B., 1987. Evidence for a fatigue-induced reflex inhibition of motoneuron firing rates. Journal of Neurophysiology. 58, 125-137.

Yang, Q., Fang, Y., Sun, C.K., Siemionow, V., Ranganathan, V.K., Khoshknabi, D., Davis, M.P., Walsh, D., Sahgal, V., & Yue, G.H., 2009. Weakening of functional corticomusclar coupling during muscle fatigue. Brain Research, 1250, 101-112.

Zhang, S.N., Bates, B.T., & Dufek, J.S., 2000. Contributions of lower extremity joints to energy dissipation during landings. Medicine and Science in Sports and Exercise, 32, 812-819.