Does force enhancement occur during the stretch-shorten cycle?

by

John William Fox

A dissertation submitted to the Graduate Faculty of
Auburn University
in partial fulfillment of the
requirements for the Degree of
Doctor of Philosophy

Auburn, Alabama May 9, 2015

Keywords: force enhancement, torque enhancement, electromechanical delay

Copyright 2015 by John William Fox

Approved by

Wendi H. Weimar, Chair, Professor of Kinesiology L. Bruce Gladden, Professor of Kinesiology Nels H. Madsen, Professor of Mechanical Engineering Gretchen D. Oliver, Associate Professor of Kinesiology Wei Liu, Assistant Professor of Kinesiology

Abstract

Recently, research into mechanisms for muscle force enhancement has gained popularity. Force enhancement is a term used for the increase in force output of muscle above that predicted by the muscle tension-length relationship, which results from active muscle stretch. Due to its dependence on active stretch it is plausible that the force enhancement property of muscle may play a role in stretch-shorten cycle movements, specifically vertical jump. Therefore the purpose of this project was: 1) To establish a curve relating force and torque to the magnitude of leg extension and lower extremity joint angles, respectively, in upright multiarticular leg extension, 2) To evaluate endpoint force enhancement as a result of a novel countermovement, the traditional countermovement, and no-countermovement, 3) To evaluate torque enhancement in each stretch-shorten cycle (SSC) condition, 4) To evaluate electromechanical delay (EMD) during each SSC condition, and 5) To examine jump height, take-off velocity, rate of force development, and impulse in each SSC condition. Results indicated that force enhancement but not torque enhancement occurs in SSC conditions. EMD was not changed even with higher rate of force development (RFD) in the self-induced drop (SD) condition. Peak normalized electromyography (EMG) was also similar across conditions. Finally, countermovement (CM) outperformed the SD and squat jump (SJ) conditions in jump height, take-off velocity and impulse, but not SD in RFD. Results suggest that high eccentric load does not necessarily alter torque enhancement, EMD or performance in SSC movements.

Acknowledgments

The author would like to thank Dr. Wendi Weimar for her constant, positive energy and her dedication to seeing this project through to the end. Without her this project would not have been completed. Also members of the Sports Biomechanics Laboratory should be thanked for their incredible efforts throughout the data collection process (Adam Jagodinsky, Lorraine Smallwood, Chris Wilburn, and Jared Rehm). Additionally, I would like to thank all of the committee members for their assistance, which has greatly improved the final product (Dr. Wendi Weimar, Dr. L. Bruce Gladden, Dr. Nels Madsen, Dr. Gretchen Oliver, Dr. Wei Liu, and Dr. Stanley Reeves). Finally, I would like to thank my family (wife, children, parents, sister, and in-laws), especially my wife and children for their incessant encouragement. All who are mentioned here equally share in the victory.

Table of Contents

| Abstract | ii |
|---------------------------------|-----|
| Acknowledgments | iii |
| List of Tables | vi |
| List of Illustrations | vii |
| List of Abbreviations | ix |
| Chapter 1: Introduction | 1 |
| Purpose | 8 |
| Hypotheses | 8 |
| Limitations | 9 |
| Delimitations | 9 |
| Definitions of Terms | 9 |
| Chapter 2: Review of Literature | 11 |
| Muscle Structure and Function | 12 |
| Force Enhancement | 21 |
| Muscle-tendon Interaction | 32 |
| Countermovements | 50 |
| Summary | 52 |
| Chapter 3: Methods | 55 |
| Participants | 55 |

| | Setting | 56 |
|--------|---|-----|
| | Materials | 56 |
| | Instrumentation | 59 |
| | Design and Procedures | 63 |
| | Data Analysis | 66 |
| Chapte | er 4: Results | 69 |
| | Participant Demographics | 69 |
| | Force and Torque Enhancement | 71 |
| | Electromechanical Delay | 84 |
| | Electromyography | 92 |
| | Performance | 94 |
| Chapte | er 5: Discussion | 99 |
| | Force and Torque Enhancement | 100 |
| | Electromechanical Delay | 107 |
| | Electromyography | 110 |
| | Performance | 110 |
| | Synthesis | 111 |
| | Future Research | 114 |
| Refere | ences | 116 |
| Apper | ndix A: Participant Screening Questionnaire | 125 |
| Apper | ndix B: Informed Consent | 126 |
| Apper | ndix C: Free Body Diagrams and Equations | 129 |

List of Tables

| Table 1: Retroreflective marker locations | 62 |
|--|----|
| Table 2: Participant Demographics | 70 |
| Table 3: Ground Reaction Force Enhancement Results | 75 |
| Table 4: Torque Enhancement Results | 83 |
| Table 5: Electromechanical Delay Results | 91 |

List of Illustrations

| Figure 1: Functional Units of Muscle | 3 |
|--|---|
| Figure 2: Sarcomere Structure | 5 |
| Figure 3: Force-length relationship | 7 |
| Figure 4: Force-length relation for multi-joint leg extension |) |
| Figure 5: Muscle-tendon Interaction | 1 |
| Figure 6: Power rack | 3 |
| Figure 7: Motion capture system arrangement |) |
| Figure 8: Anterior view of retroreflective markerset |) |
| Figure 9: Posterior view of retroreflective markerset | 1 |
| Figure 10: Graphical representation of Santello and McDonagh (1998) method | 7 |
| Figure 11: Ground Reaction Force versus Trochanter Height | 1 |
| Figure 12: Mean Ground Reaction Force Enhancement in Males and Females72 | 2 |
| Figure 13: Mean Ground Reaction Force Enhancement between SSC Conditions | 3 |
| Figure 14: Right Leg Ground Reaction Force Enhancement | 4 |
| Figure 15: Left Leg Ground Reaction Force Enhancement | 5 |
| Figure 16: Ankle Torque-angle Relationship | 7 |
| Figure 17: Knee Torque-angle Relationship | 7 |
| Figure 18: Hip Torque-angle Relationship | 9 |
| Figure 19: Right Hip Torque Enhancement | 1 |

| Figure 20: Electromechanical Delay of the Tibialis Anterior in Males and Females | 82 |
|--|-----------------|
| Figure 21: Right Soleus Electromechanical Delay | 85 |
| Figure 22: Left Soleus Electromechanical Delay in Females | 85 |
| Figure 23: Left Soleus Electromechanical Delay in Males | 86 |
| Figure 24: Left Soleus Electromechanical Delay in Males and Females | 87 |
| Figure 25: Right Vastus Lateralis Electromechanical Delay | 88 |
| Figure 26: Left Vastus Lateralis Electromechanical Delay | 88 |
| Figure 27: Right Gluteus Maximus Electromechanical Delay | 89 |
| Figure 28: Left Gluteus Maximus Electromechanical Delay | 90 |
| Figure 29: Peak Normalized Electromyography Amplitude | 94 |
| Figure 30: Jump Height | 95 |
| Figure 31: Take-off Velocity | 96 |
| Figure 32: Impulse | 97 |
| Figure 33: Rate of Force Development | 98 |
| Figure 34: Phases of the Vertical Jump | 101 |
| Figure 35: Relative Transition Phase in SJ | 105 |
| Figure 36: Relative Transition Phase in SD | 106 |
| Figure 37: Relative Transition Phase in CM | 10 6 |

List of Abbreviations

EMD electromechanical delay

EMG electromyography

MVIC maximal voluntary isometric contraction

RFD rate of force development

SSC stretch-shorten cycle

SD novel countermovement jump or self-induced drop technique

CM countermovement jump

SJ squat jump or no-countermovement jump

CHAPTER I

INTRODUCTION

Humans move. One way in which humans are able to move is by rotating segments of the body (van Ingen Schenau, 1989). For segment rotation to occur there must be an actuator that interacts with the segment in such a way as to yield a net torque. Muscles are the actuators that provide the impetus for segment rotation. Therefore, muscle mechanics has an impact on segmental dynamics and should be the rule for understanding how humans move.

A vital tenet in muscle mechanics is the relationship between the force a muscle can apply to its segments of attachment and muscle length. More simply stated, active muscle force is constrained by its current length (Gordon, Huxley, & Julian, 1966). Gordon, Huxley, and Julian (1966) published the widely accepted length-tension relation for a sarcomere, which established that myofilament overlap is the primary determinant of muscle force capacity (Rassier, MacIntosh, & Herzog, 1999). However, it should be noted that the length-tension relation neglects the effect of contractile history on muscle force capacity. In other words, the length-tension relation is not a complete description of active muscle tension capacity (Rassier, MacIntosh, & Herzog, 1999). Prior to the work of Gordon, Huxley, and Julian (1966) results from Abbott and Aubert (1952) demonstrated that the current active, isometric force capacity in whole muscle is affected by prior active stretch and shortening. More precisely, when muscle is stimulated to contract and forcefully lengthened just prior to an isometric contraction, it is capable of producing force at its current length greater than predicted by the length-tension relation (Abbott & Aubert, 1952). This is called force enhancement (Herzog, 2004)

Regardless of mechanisms at the structural level, force enhancement occurs in muscle. For example, *in vitro* studies demonstrate force enhancement at the sarcomere (Minozzo, Baroni, Correa, Vaz, & Rassier, 2013), fiber (Edman, Elzinga, & Noble, 1978), and whole muscle levels (Abbott & Aubert, 1952). While force enhancement has been studied extensively using *in vitro* techniques, *in vivo* investigations are increasing. Force enhancement has been examined in the knee extensors and flexors (Shim & Garner, 2012), the adductor pollicis (Oskouei & Herzog, 2009), and multi-articular leg extensions (Hahn, Seiberl, Schmidt, Schweizer, & Schwirtz, 2010). Taking these results together force enhancement may play a role in everyday human movement, or humans may take advantage of force enhancement in stretch-shorten cycle type motions such as in countermovement jumps.

Motions involving a stretch-shorten cycle (SSC), such as the countermovement (CM) jump, cause the muscle to stretch via segment rotation or changes in joint angles (i.e. hip flexion, knee flexion, and ankle dorsiflexion). After the stretch, the muscle develops enough tension to return to rest length and bring the segments back to their original configuration. On the other hand *in vitro* force enhancement studies using isometric contractions suggest that stretch-shorten cycles cannot take advantage of force enhancement. However, while *in vitro* techniques play a vital role in discovering mechanisms for force enhancement, these studies do little to evaluate the role of force enhancement on segmental mechanics in human movement. For example, Herzog and Leonard (2000) examined force enhancement following stretch-shortening cycles and shortening-stretch cycles in a cat soleus. The authors determined that in stretch-shorten cycles, muscle tension, during shortening and immediately after shortening stops, is less than predicted by the length-tension relationship. The diminished tension is termed force depression, and it occurs despite prior forceful stretching of the muscle. Furthermore, stretch prior to shortening

did not attenuate the amount of force depression upon shortening (Herzog & Leonard, 2000). This suggests that motions involving the stretch-shortening will not take advantage of the force-enhancement property of muscle in producing segmental accelerations. Conversely, Cavagna, Dusman, and Margaria (1968) found that *in vitro* stretch-shortening cycles resulted in enhanced work output of the muscle, but these were not fixed-end contractions. Work enhancement occurred for each given velocity, meaning that the muscle produced greater force while shortening when shortening was preceded by active stretch (Cavagna, Dusman, & Margaria, 1968). Comparing the results of these two studies shows that stretch-shortening cycles provide no performance impact in fixed-end contractions. However, in dynamic contractions a prior stretch optimizes muscular performance by increasing the amount of force produced for a given shortening velocity, which suggests a potential increase in segmental acceleration *in vivo* (Cavagna, Dusman, & Margaria, 1968).

Despite the work by Cavagna, Dusman, & Margaria (1968) some authors suggest that force enhancement does not impact segmental accelerations in SSC motions (McGowan, Neptune, & Herzog, 2013). McGowan, Neptune, and Herzog (2013) performed simulations of a CM vertical jump utilizing Hill-type muscle models modified to incorporate history-dependent effects of muscle force. The authors suggested that force enhancement occurred in the hip extensors, knee extensors, and plantarflexors, but force enhancement had little effect due to force depression during the propulsive phase of the jump (McGowan, Neptune, & Herzog, 2013). Therefore, force enhancement may not carry importance in the traditional countermovement jump. Because these authors only examined the traditional CM, there is no evidence against the effect of force enhancement in novel loading conditions. Since muscle-tendon behavior may be altered based on the loading characteristics, any change in loading necessitates a different

contractile history (Earp, Newton, Cormie, & Blazevich, 2013). Therefore, novel movement initiation schemes should be examined as the altered loading may cause a change in muscle-tendon interaction.

Hence, examining only the CM limits understanding of the role of muscle-tendon interactions and differences in loading. Muscle-tendon interactions have the ability to broaden or narrow the force-length relation when the tendon is compliant or stiff, respectively (Lieber, Brown, & Trestik, 1992; Kawakami & Lieber, 2000). In essence, with a compliant series elastic component the optimal length of the muscle is shifted to longer lengths and the muscle is able to shorten a greater distance. It should be noted that these results come from studies using fixed-end contractions. In human movement studies, however, the distance between muscle attachment sites changes when joint angles change. Therefore, changes in joint angle must be taken into account with the compliance of series elasticity. When the tendon is compliant, the tendon may take up the change in length necessitated by joint rotations (Lieber, Brown, & Trestik, 1992; Roberts, Marsh, Weyand, & Taylor, 1997). On the other hand, when the tendon is stiff, the muscle fibers may absorb the length change necessitated by joint rotations. For example Biewener and Blickhan (1988) examined hopping in kangaroo rats, which have stiff tendons. As a result of stiff tendons muscle fibers lengthened prior to shortening during hopping. Stretchshortening in this case allowed the plantarflexors to produce force greater than the maximum isometric value. In human studies the tendon of the gastrocnemius appears to be compliant, allowing the muscle fibers to contract concentrically and isometrically during a CM (Kurokawa, Fukunaga, Nagano, & Fukashiro, 2003; Kawakami, Muraoka, Kanehisa, & Fukunaga, 2002). Because no stretch phase was found, these results indicate that force enhancement does not occur in the gastrocnemius during the CM jump. In more proximal musculature (vastus lateralis) of

humans, it has been shown that muscle fibers undergo active stretch prior to shortening in drop jumps (Ishikawa, Niemalä, & Komi, 2005). As a result force enhancement is likely to occur in the proximal musculature of the lower extremity provided the eccentric load is great enough. Furthermore, greater drop height resulted in higher rates of force development within the patellar tendon (Ishikawa, Niemalä, & Komi, 2005). Higher rates of force development may cause the quadriceps tendon to behave with increased stiffness and act as a rigid force transducer (Earp, Newton, Cormie, & Blazevich, 2013). These results suggest that muscle-tendon characteristics play an important role in the muscle force produced during jumping, the CM does not induce fiber stretch, and increasing the eccentric load in an SSC motion may result in force enhancement.

Muscle-tendon interactions are generally studied using ultrasound techniques (Ishikawa, Niemalä, & Komi, 2005; Ishikawa, Komi, Finni, & Kuitunen, 2006; Earp, Newton, Cormie, & Blazevich, 2013). Other methods for gaining insight into muscle-tendon behavior such as measurement of electromechanical delay (EMD) are possible. Research has shown that changes in electromechanical delay suggest differences in muscle-tendon interaction in eccentric versus concentric muscle actions (Norman & Komi, 1979). It is suggested that EMD represents the time necessary to strain the aponeurosis and tendon to the point that muscle force is detectable (Cavanagh & Komi, 1979; Norman & Komi, 1979). Assuming that the current explanation of EMD is correct and that high rates of force development cause tendons to behave with greater stiffness (Earp, Newton, Cormie, & Blazevich, 2013), then SSC motions involving high eccentric loads should result in shorter EMD. Conversely, lower loading rates should result in longer EMD. Therefore, despite the absence of visual data on muscle-tendon behavior, EMD

should provide sufficient evidence that muscle tendon interactions have altered to accommodate the rate of force development.

High loading rates appear to alter muscle-tendon interaction, which affects EMD, and high loading rates may cause force enhancement. Despite this knowledge, it is difficult to find research that measures force enhancement while performing a countermovement. However, force enhancement is implied by Ishikawa, Niemalä, and Komi (2005) and Ishikawa, Komi, Finni, and Kuitunen (2006). Both of these studies examined the drop jump, which increases the loading on the lower extremity (Bobbert, Mackay, Schinkelshoek, Huijing, & van Ingen Schenau, 1986). Furthermore, both found active lengthening of muscle during force development of drop jumps on a sled. Active lengthening suggests force enhancement. Hence, to elicit the force enhancement property of muscle in a vertical jump the eccentric loading must be increased.

To increase eccentric loading a drop prior to the CM is effective. Bobbert, Mackay, Schinkelshoek, Huijing, and van Ingen Schenau (1986) examined three loading techniques in the vertical jump: 1) the CM 2) a drop jump involving minimal ground contact time and 3) a drop jump involving a larger amplitude countermovement. The drop with minimal ground contact time required greater muscular force as the joint moments and ground reaction forces were greater (Bobbert, Mackay, Schinkelshoek, Huijing, & van Ingen Schenau, 1986). Given the results of Ishikawa, Niemalä, and Komi (2005) it is possible that the force enhancement property of muscle provided the ability to produce joint moments capable of withstanding the large endpoint forces. However, the drop jump involving a larger countermotion and the CM yielded higher jump heights compared to the drop jump with short contact time. Therefore, it is also possible that force enhancement can occur in the absence of performance enhancement and vice versa. One of the purposes of this study is to evaluate different loading configurations to

determine whether force enhancement occurs in an SSC and whether force enhancement results in a performance enhancement.

In humans, performance enhancement has been demonstrated in movement initiation schemes that involve a brief aerial phase prior to the countermotion (Fujii, Yoshioka, Isaka, & Kouzaki, 2013; Nieminen, Piirainen, Salmi, & Linnamo, 2013; Uzu, Shinya, & Oda, 2009). Specifically, Nieminen, Piirainen, Salmi, and Linnamo (2013) and Uzu, Shinya, and Oda (2009) evaluated the performance effect of the "split step" used in tennis. The split step requires the center of mass to travel vertically prior to a free fall into the SSC. Increased ground reaction forces and decreased time to complete a step and reach task resulted from the use of the split step (Nieminen, Piirainen, Salmi, & Linnamo, 2013; Uzu, Shinya, & Oda, 2009). Not only does the split step improve performance, but it is feasible in competition as exemplified by its use. Additionally, movements have been noticed in canines that utilize an aerial phase prior to the countermotion of a sprint start (Angle, Gillette, & Weimar, 2012). The canines raise their paws 4-8 cm from the ground, but this is without vertical motion of the center of mass. In other words, the canines free fall into a countermotion (Angle, Gillette, & Weimar, 2012). However, a movement initiation technique similar to that used by canine sprinters has not been examined in humans.

By addressing force and torque enhancement in conjunction with a new countermovement initiation technique this study added to the literature in two ways. First, the research in muscle mechanics was advanced by exploring the role of muscle force enhancement in an SSC but within a natural motion or setting. Second, there is little research examining variations of countermovement initiation. As a result this study attempted to fill the research void

on movements that take advantage of muscle mechanics yet may be more effectively utilized in the athletic arena.

Purpose

The aforementioned research contributions are constructed by the following objectives:

1] This study established a curve relating force and torque to the magnitude of leg extension and lower extremity joint angles, respectively, in upright multi-articular leg extension. 2] This study evaluated endpoint force enhancement as a result of a novel countermovement, the CM, and no-countermovement. 3] This study evaluated torque enhancement in each SSC condition. 4] This study evaluated EMD during each SSC condition. 5] This study examined jump height, take-off velocity, rate of force development, and impulse in each SSC condition.

Hypotheses

- H01: Endpoint force enhancement occurs during the novel technique but not in the traditional countermovement and no-countermovement.
- H02: Torque enhancement will occur at the hip, knee, and ankle in the novel countermovement but not in the traditional and no-countermovement conditions.
- H03: Endpoint force will linearly increase with leg extension, whereas torque will demonstrate a nonlinear relation with joint extension (Hahn, Olvermann, Richtberg, Seiberl, & Schwirtz, 2011).
- H04: EMD will decrease with increased eccentric load (i.e. novel countermovement will yield shortest EMD).

H05: Performance measures such as jump height, rate of force development, and impulse will be greater in the novel condition compared to the traditional and no-countermovement conditions.

Limitations

Limitations in this study are the following:

- Data for endpoint force-leg extension and torque-angle relations were collected on a day separate from the vertical jump data.
- 2. Ultrasound techniques were not used.
- 3. The novel condition (SD) is traditionally used for horizontal accelerations, not vertical.

Delimitations

Delimitations for the current study are the following:

- Force-leg extension data were collected with an isometric squat which allowed for comparable segment geometry with the jumps.
- 2. Participants wore retroreflective markers bilaterally.
- 3. Surface electrodes were placed bilaterally on the gluteus maximus, vastus lateralis, gastrocnemius, and tibialis anterior.
- 4. Data collection occurred in the Sports Biomechanics Laboratory.

Definition of Terms

Force enhancement: The ground reaction force produced above that predicted by a forceleg extension curve. *Torque enhancement:* Net joint torque produced above that predicted by a maximum torque-angle curve.

Electromechanical delay: The time interval between rise in muscle activity and a change in segment velocity.

CHAPTER II

REVIEW OF LITERATURE

Muscles and their tendons actuate human movement. All human movement outcomes are dependent on the behavior of the muscle-tendon unit. An integral part of understanding how humans move or why humans move a particular way involves understanding the complex interaction between a muscle and its tendon. In an applied research setting understanding muscle-tendon interaction provides a framework of knowledge for developing movement schemes that exploit muscle-tendon behavior.

To embark on the endeavor to understand how the mechanics of the musculature affects movement schemes the following objectives have been established: 1) To evaluate the relationship between force and torque to segment orientation in an upright maximum voluntary leg extension exercise, 2) to utilize the aforementioned objective as a reference for evaluating endpoint force torque enhancement in vertical jumps using a SD, CM, and SJ, 3) to evaluate torque enhancement in each SSC condition, 4) to evaluate EMD as a reflection of muscle-tendon interaction during each SSC condition, and 5) to examine jump height, take-off velocity, rate of force development, and impulse in each SSC condition. The following chapter addresses each objective in the following sections: 1) muscle structure and function, 2) force enhancement, 3) muscle-tendon interaction, and 4) vertical jump performance.

Muscle Structure and Function

Muscle structure is complex, and consequently muscle function is complex. Muscle length, in particular, has a profound impact on muscle force production (Rassier, MacIntosh, & Herzog, 1999). Since muscles cross joints, length changes necessarily occur during human movement. Therefore, force production capacity changes during human movement. As muscle tension capacity is altered, joint torque production varies. It should be noted that joint torque also varies with geometric positioning of limb segments (Zatsiorsky, 2003). Endpoint force (i.e. ground reaction force in vertical jump) is determined by lower extremity joint torques and relative limb segment position (Zatsiorsky, 2003). Hence, the structure of muscle can function to generate endpoint force and accelerate the mass center of the body.

Structurally muscle may be viewed as bundles of functional units descending in size (Herzog, 2007) (Figure 1). The first bundle is the muscle, which is encased in fascia and the epimysium. Each muscle is comprised of fascicles surrounded by the perimysium. Fascicles consist of bundles of muscle fibers, muscle cells, covered by the endomysium. A bundle of myofibrils surrounded by the sarcolemma forms each fiber. Each myofibril is a series of sarcomeres. Sarcomeres are the contractile units of muscle (Herzog, 2007). Sarcomeres contain protein filaments, actin and myosin, which cause the striated appearance of the myofibril (MacIntosh, Gardiner, & McComas, 2006). These functional units are separated by connective tissue, which provides structure and therefore a means to transmit force to the tendon (MacIntosh, Gardiner, & McComas, 2006).

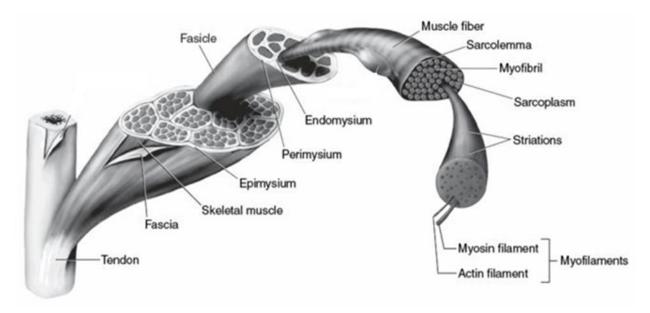


Figure 1. Picture demonstrating the bundles of function units that comprise muscle. Adapted from Whiting and Rugg (2005).

Force that is transmitted to the tendon is generated by the series of sarcomeres forming each myofibril (Herzog, 2007; MacIntosh, Gardiner, & McComas, 2006). Myofibrils appear as a pattern of alternating light and dark bands, striations, which are separated by z-lines demarcating the ends of the sarcomere. The dark bands, A-bands, are anisotropic and correspond to the presence of myosin, which is the thicker of the two protein filaments. Myosin molecules are comprised of a light meromyosin tail and a heavy meromyosin head. The globular head spans outward from the myosin molecule forming the cross-bridge portion of the thick filament. A cross-bridge is present every 14.3 nm along the length of the thick filament. Looking down the length of the thick filament each subsequent cross bridge is offset 60° radially from the previous cross-bridge. As a result the subsequent cross-bridge with the same orientation is 42.9 nm down the myosin filament. Myosin molecules are oriented with their tails at the middle of the thick filament, which orients the myosin head of each half sarcomere in opposite directions. Due to cross-bridge arrangement the myosin heads are believed to attach to actin and pull actin toward

the center of the sarcomere. Actin filaments are thin and form I-bands, the isotropic or light bands. Each actin molecule is a chain of actin globules. Two chains of actin wrap over each other every 5-8 globules. Wrapping forms a groove for binding of tropomyosin. Every 35-38.5 nm troponin is attached to tropomyosin. Troponin consists of 3 units: 1. troponin C binds calcium; 2. troponin T binds troponin to tropomyosin; and 3. troponin I inhibits cross-bridge attachment when calcium is absent. Hence myosin and actin perform contractile duties, while tropomyosin and troponin regulate cross-bridge attachment (Herzog, 2007).

Even while the muscle is at rest a portion of the A-band contains actin and myosin joined in an overlapping manner, although the overlap is not complete. Overlap of the thick and thin filaments is partially due to their relative lengths. Length of the thick filament, measured at 1.6 μ m, is constant across vertebrates (Herzog, 2007; Gohkin, Kim, Lewis, Heinz, D'Lima, & Fowler, 2012). However, the thin filament length varies across vertebrates (Herzog, 2007) and across muscles and fiber types (1.19 \pm 0.08 to 1.37 \pm 0.04 μ m) within a species (Gohkin et al., 2012). The length of the two filaments and the amount of overlap between them strongly influences the length and magnitude of force that a muscle may produce.

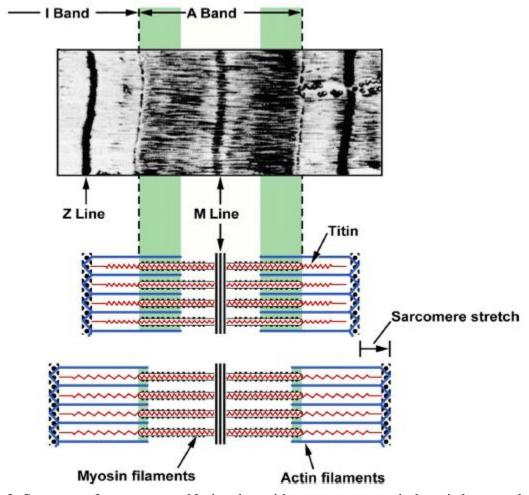


Figure 2. Structure of a sarcomere. Notice that with sarcomere stretch there is less overlap between actin and myosin. Adapted from Leonard and Herzog (2010).

Force production is dictated by the orderly structure of the musculature (Figure 2). Muscle structure provides the basis of the current models of muscle contraction: the sliding filament theory and the cross-bridge theory. These theories are important for providing an understanding of the length dependence of force. The sliding filament theory states that the shortening of the sarcomere occurs due to the relative motion of the myofilaments, actin and myosin (Huxley & Niedergerke, 1954; Huxley & Hanson, 1954; Rassier, MacIntosh, & Herzog, 1999). Motion of the myofilaments past one another is generated by cross-bridges (Huxley & Niedergerke, 1954; Huxley & Simmons, 1971; Rassier, MacIntosh, & Herzog, 1999). Each

cross-bridge is assumed to produce the same average force as other cross-bridges; therefore, the force of the sarcomere is directly proportional to the overlap between the actin and myosin filaments (Huxley & Niedergerke, 1954; Huxley & Simmons, 1971). A theoretical prediction of the length-tension curve can be derived based on the lengths of the myofilaments. Derivations of this kind can be found in Herzog (2007) and Rassier, MacIntosh, and Herzog (1999). The derivation is as follows: Thick filaments are 1.65µm long, which is nearly constant across species, and actin, although variable across fiber type (Gohkin et al., 2012), can be taken to be 0.95µm (Rassier, MacIntosh, & Herzog, 1999). Z-lines are 0.1µm wide combined, and a 0.2µm gap between cross-bridges exists at the center of the myosin filament. Given these dimensions one can sum the width of the z-lines and double the length of actin to obtain that maximal overlap of actin and myosin occurs at 2.0µm. The sarcomere can extend an additional 0.2µm and maintain maximal overlap because the central 0.2µm of myosin does not possess cross-bridges. Therefore, maximal overlap should extend from 2.0 to 2.2 µm. Finally, overlap goes to zero at 3.65 µm, which can be calculated as the sum of two times the length of actin, the width of the zlines, and the length of myosin. Experimental evidence for these lengths was provided by Gordon, Huxley, and Julian (1966) on frog muscle. In this length-controlled study of isometric tension in muscle fiber it was discovered that sarcomere tension plateaus between the lengths of 2.0µm and 2.2µm. Below 2.0µm tension begins to decline, and the slope of the decline increases below 1.7 µm. Tension falls to zero at a length of 1.3 µm. Beyond the length of 2.2 µm tension also declines and falls to zero at 3.65 µm. Hence, the supposed force-length relation was confirmed experimentally (Gordon, Huxley, & Julian, 1966).

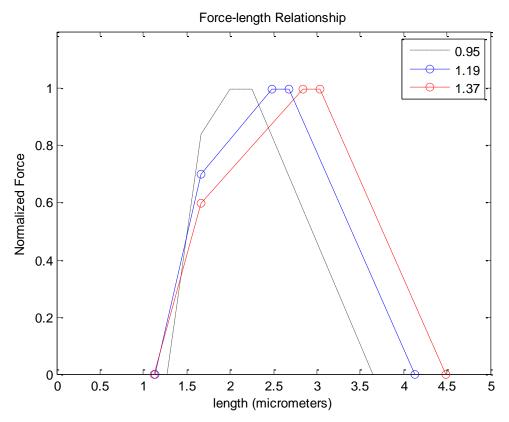


Figure 3. The figure above is a representation of the theoretical force-length curve for a sarcomere. The black line is based on data from Gordon et al. (1966). The blue and red lines are based on Gohkin et al. (2012). The legend denotes the actin filament length.

With the variable length of actin across species, muscles, and fiber type (Gohkin et al., 2012) a shift in the force-length curve will also occur between species, muscles and fiber type (Figure 3). For example, Gohkin and colleagues (2012) found actin isolated from the human deltoid to be 1.19μm long, whereas actin from the human pectoralis major was 1.37μm long. Based on the calculations above this would result in a plateau from 2.48 to 2.68μm in the deltoid. The plateau for the pectoralis major would be 2.84 to 3.04μm. A shift of .48-.84μm is present in the plateau of the force-length relation between humans and frogs used by Gordon, Huxley, & Julian (1966). More importantly the difference between the deltoid and the pectoralis major is a shift to the right of 0.36μm. In essence maximum isometric tension is developed at longer

sarcomere lengths, if the thin filament is long (Figure 3). Additionally the range of lengths over which a sarcomere may produce force is greater with longer actin length (Figure 3).

Muscle structure, therefore, greatly affects force production capabilities of muscle (i.e. muscle function) (Figures 2 and 3). Actin length, specifically, will have important implications for the moment-angle relationship of a particular joint as the length of actin appears to determine the shape of the force-length curve (Rassier, MacIntosh, & Herzog, 1999). Herzog and ter Keurs (1988a, 1988b) effectively demonstrated this by deriving the force-length relation of biarticular muscles from the moment-angle relationships of the knee and ankle. It is clear that the force-length property of muscle directly affects the moment-angle property of a joint. This effect can be seen in the equation that follows:

$$\vec{M} = \vec{r} \times \vec{F}$$

M in the above equation represents moment or torque, *r* represents the vector from the line of action of the muscle force to the joint center of rotation, and *F* represents the force vector. Since muscles actuate human movement, *F* in the above equation can be replaced with muscle force. Muscle force is determined by the force-length property. Muscle length will be determined by the change in joint angle. That is as the angle of a joint opens the muscle will lengthen, and as the joint angle closes the muscles will shorten. Hence, the moment-angle relationship of a joint is determined by the length of the muscle and the length of the moment arm (Rassier, MacIntosh, & Herzog, 1999; Anderson, Madigan, & Nussbaum, 2007).

Furthermore, joint moments produced by muscles and the respective moment arms result in endpoint forces (Zatsiorsky, 2003; Zatsiorsky & Prilutsky, 2012). Specifically, joint moments control the endpoint forces that are used to accelerate the body (Zatsiorsky, 2003). However, while the relationship between joint moments and endpoint force is directly proportional in the

single joint case, the relationship in the multi-joint case is determined by the joint torques and the system geometry (Zatsiorsky, 2003). Hahn (2008) performed an experiment to determine the relationship between leg length (defined by the distance from the hip to the floor) and external force. Kinematics and kinetics were measured during maximal isometric leg extensions in a leg press dynamometer. Data were collected in 10 degree increments through a range from 30 to 100 degrees of knee flexion. Figure 4 shows results taken from Table 1 in Hahn (2008). Despite research demonstrating that the muscles of the lower extremity function on the ascending limb of their force length relation (Rubenson, Pire, Loi, Pinniger, & Shannon, 2012; Maganaris, 2001), the data from Hahn (2008) have the appearance of the descending limb. As the extensor muscles of the lower extremity were lengthened, external force decreased. Therefore, the relationship between external force during multi-joint leg extension and muscle length is not congruent to *in vitro* force-length relations.

In contrast, torque-angle relationships during multi-joint leg extension more closely approximate the shape of muscle length-tension curves (Figure 4). Torque-angle relationships in multi-joint leg extensions were evaluated by Hahn, Olvermann, Richtberg, Seiberl, and Schwirtz (2011). Most torque-angle relationships are investigated in single-joint protocols, which in lower extremity cases suggest that the torque-angle relation follows an ascending limb (Anderson, Madigan, & Nussbaum 2007). In multiarticular leg extensions Hahn, Olvermann, Richtberg, Seiberl, and Schwirtz (2011) reveal that knee and ankle joint torque-angle relationships have an ascending- descending and plateau-descending pattern, respectively. For example, maximum knee torque of 289.5 ± 43.3 Nm was developed at an angle of $50.4 \pm 8.9^{\circ}$. Below 50.4° less torque was produced about the knee. Similarly, knee angles above 50.4° resulted in decreased

torque. Clearly there is a striking resemblance to the ascending, plateau, and descending limbs of muscle length-tension relationships (Figure 4).

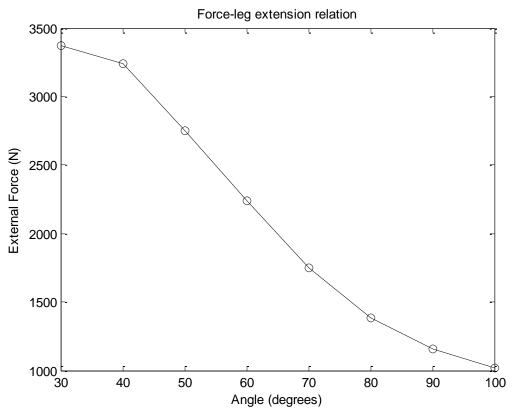


Figure 4: Force-length relation for multi-joint leg extension. The abscissa represents the magnitude of knee flexion with zero being no flexion. Data are taken from Hahn (2008).

Despite having well defined relationships between force and leg extension, or if preferred, torque and joint angles, the effects of movement history on these relationships is not well researched. Therefore, a study comparing force and torque during a movement involving stretch-shorten cycle (i.e. vertical jump) to force and torque during maximal voluntary isometric leg extensions should be conducted. It should be noted that the torque-angle data from Hahn, Olvermann, Richtberg, Seiberl, and Schwirtz (2011) is taken from horizontally performed leg extensions. As a result gravitational forces will act to extend the legs or generate extension torques about the joint which would lead to an underestimation of torque enhancement. A better

comparison would involve upright leg extensions since the body may be arranged in a more similar configuration relative to vertical jump. Also gravitational forces will generate flexion torques about the joints, which more closely approximate the dynamics of vertical jump. Hence, by comparing force and torque in vertical jump to an upright, maximal, voluntary, multi-articular leg extension one may determine whether isometric multi-articular leg extensions provide a complete picture of force producing capabilities in normal tasks.

Force Enhancement

Movement history affects force production capabilities in normal tasks. As joints rotate, points of muscle attachment move closer together or farther apart. Movement of attachment sites of muscle necessitates length changes in the muscle. As the muscle is set to new lengths the force produced is impacted by the new length. As it turns out the force produced is also influenced by the change in length (Herzog, 2004). Therefore, movement history begets muscle contractile history. Furthermore, while the force-length and single-joint moment-angle relations are vital to understanding muscle function and actuation of movement, these relations are not complete pictures of force production capacities of muscle due to history dependence. The logical consequence is that force-leg length relations and torque-angle relations in multi-articular leg extensions are also dependent on history, particularly movement history.

Movement history dependence begins with contractile history dependence. For example, force production of muscle is history dependent (Herzog, 2004). When muscle is actively stretched to a determined length, the force produced is greater than the force produced during an isometric contraction at the same length (Abbot & Aubert, 1952; Edman, Elzinga, & Noble, 1978; Herzog & Leonard, 2000; Herzog, 2004; Oskouei & Herzog, 2009; Peterson, Rassier, &

Herzog, 2004; Herzog, Joumaa, & Leonard, 2010; Edman, 2012). Force enhancement of muscle has been examined in myofibril (Joumaa & Herzog, 2013; Herzog, Joumaa, & Leonard, 2010; Linari et al., 2000), fiber (Edman, Elzinga, & Noble, 1978; Edman & Tsuchiya, 1996; Sugi & Tsuchiya, 1988; Peterson, Rassier, & Herzog, 2004), whole muscle (Abbott & Aubert, 1952; Cavagna & Citterio, 1974; Hisey, Leonard, & Herzog 2009; Herzog & Leonard, 2000; Brown & Loeb, 2000), and single joint and multi-joint *in vivo* studies (De Monte & Arampatzis, 2009; Oskouei & Herzog, 2009; Seiberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013; De Monte & Arampatzis, 2008; Seiberl, Hahn, Herzog, & Schwirtz, 2012; Shim & Garner, 2012).

In force enhancement studies there are two distinct phases of force enhancement: 1] a transient phase and 2] a residual phase. The transient phase of force enhancement occurs during the active lengthening. During the transient phase, force exerted by the muscle is overcome by a resistance, and the muscle stretches. Conceptually this is an eccentric contraction. During active stretch a rapid rise in force occurs, which increases with increasing stretch velocity (Abbott & Aubert, 1952; Edman, Noble, & Elzinga, 1978; Sugi & Tsuchiya, 1988; Koppes, Herzog, & Corr, 2013). However, force during this phase does not increase infinitely with increasing velocity magnitude, but force can reach up to 1.8 times the isometric maximum of the muscle (Katz, 1939). To understand the mechanism for the transient rise in force Sugi and Tsuchiya (1988) measured increased stiffness in fibers during stretch. Additionally, Linari et al. (2000), through stiffness measurements, estimated that while muscle fibers lengthen the number of cross bridges formed increases to 180% of the isometric number. As a result the force produced by the stretched muscle will rise to a maximum at the end of the stretch, and this increase in force is governed by cross bridge mechanics. Once a maximum force is reached and stretching ceases, the force decreases exponentially (Abbott & Aubert, 1952; Edman, Noble, & Elzinga, 1978; Sugi & Tsuchiya, 1988; Koppes, Herzog, & Corr, 2013). The exponential decay rate does not correlate significantly with total force enhancement, but the active component of force enhancement significantly correlates with the rate of force decay after stretch (Koppes, Herzog, & Corr, 2013). Rassier and Herzog (2004) treated muscle fibers with 2, 3-butanedione monoxime (BDM), which is believed to stabilize cross bridges in a weak binding state and to slow cross bridge turnover. As a result isometric forces decreased in the fibers while relative force enhancement during stretch and after stretch increased. Furthermore, the stiffness of the muscle fibers increased relative to the isometric force produced and rate of force decay after stretch decreased (Rassier & Herzog, 2004). This further solidifies the role of the cross-bridges in providing a large transient force enhancement during stretch and establishes the role of cross bridges in residual force enhancement.

Once the transient phase concludes, the muscle force decreases to a residual phase that is greater than the force produced by an isometric contraction of the same length without contractile history (Edman, Elzinga, & Noble, 1982; Edman & Tsuchiya, 1996). While most research demonstrates residual force enhancement on the descending limb of the force-length relation (Edman, Elzinga, & Noble, 1978; Edman, Elzinga, & Noble, 1982; Edman & Tsuchiya, 1996; Schachar, Herzog, & Leonard, 2004), residual force enhancement has been shown to occur for stretches on the ascending limb of the force-length relationship (Peterson, Rassier, & Herzog, 2004; Hisey, Leonard, & Herzog, 2009). Therefore, the mechanism for residual force enhancement applies throughout most of the force-length relation.

The importance of the residual phase is that it reveals an alternative mechanism involved in force enhancement. Edman and Tsuchiya (1996) compared quick releases of fibers that contracted isometrically to fibers undergoing active stretch prior to the quick release. In fibers

that were actively stretched prior to release the initial length change was rapid compared to fibers without prior stretch. Edman and Tsuchiya (1996) deduced that an elastic mechanism played a role in residual force enhancement and was responsible for the shift in the force-velocity relation. As should be expected from an elastic component, force enhancement in the residual phase increases with the magnitude of the stretch. Research at the myofibril and sarcomere levels has argued that the elastic mechanism contributing to force enhancement is titin (Joumaa, Rassier, Leonard, & Herzog, 2007; Leonard & Herzog, 2010; Minozzo, Baroni, Correa, Vaz, & Rassier, 2013). An important study by Journaa, Rassier, Leonard, and Herzog (2007) depleted myofibrils of troponin C, which renders the myofilaments incapable of forming cross-bridges. Despite lack of actin-myosin binding, myofibrils placed in activating solution produced force 25-30% greater than force produced by troponin C depleted myofibrils in relaxing solution. The increase in force resulting from the activating solution was abolished when myofibrils were further treated with trypsin, which degraded titin. The authors concluded that titin stiffness is increased in the presence of calcium, but this does not account for the other 75% of the force enhancement in intact myofibrils (Joumaa, Rassier, Leonard, & Herzog, 2007). In another study Leonard and Herzog (2010) demonstrated that force rises more steeply and to a greater magnitude in myofibrils undergoing active stretch to lengths beyond myofilament overlap compared to nonactivated myofibrils. Additionally this force increases with magnitude of stretch similar to an elastic mechanism. However, if the elastic mechanism is purely passive then the same forces during activation should be seen during stretches in relaxing solution. A number of tests were performed to evaluate this non-intuitive behavior. First, myofibrils were placed in relaxing solution and stretched beyond potential for myofilament overlap. Next myofibrils were placed in activating solution and stretched beyond myofilament overlap. Because activated myofibrils

produced more force than non-activated myofibrils even at lengths beyond actin-myosin overlap, it was necessary to evaluate whether the enhanced force was due to cross-bridge kinetics or titin. Trypsin was used to remove titin. In the titin depleted preparations forces did not rise substantially above zero, which was true in both the activating solution and relaxing solution. This result suggested that titin plays an important role in force enhancement. Next myofibrils were treated with BDM and placed in activating solution. Because BDM is a cross-bridge inhibitor, the myofibrils treated with BDM and placed in activating solution produced no more force than the intact myofibrils in non-activating solution. This insinuates that cross-bridge kinetics play a role in force enhancement. However, stretches were pulled to lengths beyond the potential for cross-bridge formation. Force produced during stretches beyond myofilament overlap demonstrates that another mechanism is responsible for force enhancement. To better understand this phenomenon, the authors passively stretched myofibrils until sarcomeres were at 2.4 µm (corresponding to optimal length in rabbit psoas muscle), placed them in activating solution, and stretched them to 5.0 µm. With another group of myofibrils, the activating solution was not used until the sarcomeres were at 3.4 µm (corresponding to minimum overlap of actin and myosin in rabbit psoas). Then the myofibril was stretched to 5.0 µm. Myofibrils activated from 2.4 µm produced higher forces. The authors concluded that titin is the main component of passive force enhancement, it is stiffened by calcium, and that force regulates titin-actin binding, which is responsible for the steep rise in force in myofibrils stretched in activating solution (Leonard & Herzog, 2010). From these studies it is clear that titin is a prime mechanism for enabling sarcomeres to produced force above that predicted by the isometric force-length relation, and it is likely the passive elastic mechanism contributing to the force-velocity shift witnessed by Edman and Tsuchiya (1996).

Provided that parallel elastic mechanisms are responsible for force enhancement, it is reasonable that force should increase with amplitude of stretch. Indeed, as stretch magnitude is increased the amount of force enhancement increases, but this is dependent on the ending length of the muscle after stretch (Edman, Elzinga, & Noble, 1978; Edman, Elzinga, & Noble, 1982, Hisey, Leonard, & Herzog, 2009). Edman, Elzinga, and Noble (1978) suggested that residual force enhancement is not dependent on stretch amplitude provided the sarcomere length is below 1.8 µm. Later, Edman, Elzinga, and Noble (1982) found that residual force enhancement plateaus at sarcomere lengths above 2.9 µm. More recently a study by Hisey, Leonard, and Herzog (2009) using whole cat soleus performed stretches to lengths of 3 mm, 9 mm, and -3 mm with respect to resting length. The magnitude of the stretches ranged from 3 mm to 24 mm. Force enhancement was seen in all tests. Stretches to 9 mm beyond resting length resulted in the greatest residual force enhancement, which was significantly greater than stretches to 3 mm beyond rest length. Stretches to 3 mm past rest length showed residual force enhancement that was significantly greater than stretches to -3 mm with respect to resting length. When the muscle was stretched to 9 mm past resting length, force enhancement increased with the magnitude of stretch up to a 12 mm stretch. When the soleus was stretched to 3 mm past rest length, force enhancement was not dependent on the magnitude of the stretch. When stretches were 3 mm below rest length, force enhancement decreased with stretch magnitude. The authors noted that muscle fibers stretched approximately 50% of the distance of the applied stretch. When the muscle was stretched 24 mm, the fibers only lengthened about 12 mm. The unequal length change was attributed to properties of the aponeuroses. As the whole muscle was stretched further the aponeurosis lengthened less due to a characteristic nonlinear strain. Less strain of the aponeurosis necessitates that the fibers stretch more, leading to force enhancement. Therefore,

given a particular ending length, force enhancement achieved is dependent on the muscle-tendon interaction.

Appropriate muscle-tendon interactions for force enhancement occur *in-vivo*. This has been shown in single-joint and multi-articular movements. For example, force enhancement has been shown in vivo in the plantar flexors (De Monte & Arampatzis, 2008; De Monte & Arampatzis, 2009), the knee extensors and flexors (Shim & Garner 2012), and the adductor pollicis (Oskouei & Herzog, 2009). Oskouei and Herzog (2009) evaluated force enhancement with contractions of the adductor pollicis muscle at 30% of maximal voluntary effort. The purpose of this study was to test if sub-maximal, electrically induced contractions produced force enhancement. A second purpose was to compare forces at 30% of maximal voluntary contraction to 6 seconds of constant activation levels at 10, 60, and 100% prior to isometric contraction without stretch. Force output was controlled via constant voltage and changing the frequency or via constant frequency and changing the voltage. In Protocol 1 each participant completed sets of trials involving an isometric reference and an isometric-stretch-isometric test contraction. Protocol 2 determined if force enhancement could be achieved without stretch but with activation. Protocol 1 showed a statistically significant steady state force enhancement following stretch of the adductor pollicis for all submaximal electrically evoked contractions. For the constant voltage method at 10, 30, and 60% force enhancement was 19±2%, 13±2%, and 9±2%. For the constant frequency method, force enhancement was 46±11%, 25±5%, and 15±3%. For activations at 60% and 100% of MVC and no muscle stretch, five out of nine and six out of nine subjects showed consistent activation induced force enhancement at 30% effort. With 10% activation force depression was shown. Activation-induced force enhancement was smaller than stretch-induced. Hence, in voluntary contractions there appears to be an activation and stretchdependent component in some subjects. Force enhancement was greater at lower levels of stimulation. This means that force enhancement is not directly proportional to force. It is suggested that a passive component provides a constant contribution to stretch and may be the culprit. A passive component explains greater force enhancement at low activation (Oskouei & Herzog, 2009).

Muscle activation and its role in force enhancement are more complex with larger muscles. According to Seiberl, Hahn, Herzog, and Schwirtz (2012), force enhancement in the quadriceps may be hidden by redundancies in recruitment strategies. That is, several motor control strategies may exist to maintain a submaximal contraction level. Residual force enhancement was analyzed for sub-maximal voluntary contractions when controlled via muscle activation and torque. Subjects were seated with a 100-degree angle between the thigh and trunk. Pure isometric contractions were performed, and isometric-eccentric-isometric contractions from 80- to 100-degree knee angles were performed with the eccentric phase at 60 degrees/s. MVCs at 80- and 100-degree knee angles were performed to measure maximum torque and activation. Then submaximal stretch contractions and isometric reference contractions were performed at 30% and 60% MVC. Residual force enhancement was seen in all experimental conditions. Activation reduction was also seen in joint torque control trials. Activation reduction increased with increasing torque. (Seiberl, Hahn, Herzog, & Schwirtz, 2012). Such results insinuate that force enhancement may manifest as activation reduction after stretch. As a result if muscle activity is less in the propulsive phase of a vertical jump, this could be due to force enhancement occurring in muscle, due to prior stretch.

Because vertical jump is a multi-joint movement, studies examining force enhancement in multi-articular protocols are more pertinent. Indeed, force enhancement has been shown in

multi-joint leg extensions involving the hip, knee, and ankle (Seiberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013). Seiberl, Paternoster, Achatz, Schwirtz, and Hahn (2013) investigated the presence and characteristics of residual force enhancement for a submaximal multi-articular leg extension. The authors hypothesized that enhanced force and enhanced joint torques are present after forced flexion in a multi-joint leg extension task. The characteristic transient and residual forces appeared. Force enhancement was found for endpoint force, and knee and ankle torques at all instances in time (Sieberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013). Taking into account Equation 1, if force enhancement occurs at the muscular level, the moment about a joint increases due to the direct relationship between moment and force. Endpoint force also increases, if the geometry of the segments allows. Therefore, endpoint force and torque enhancement are indicators of force enhancement at the muscular level. Seiber, Paternoster, Achatz, Schwirtz, and Hahn (2013) found no difference in EMG data between purely isometric extensions and those with a pre-stretch. As a result there was less activation per unit of force, which bears a resemblance to the results of Oskouei and Herzog (2009) and Seiberl, Hahn, Herzog, and Schwirtz (2012). The authors suggested that force enhancement should be considered in SSC (Sieberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013).

One study that has quantified the magnitude of force enhancement during the countermovement jump, which involves an SSC, was performed by McGowan, Neptune, Herzog (2013). This study involved a mathematical simulation of the countermovement jump. The rationale was lack of knowledge concerning the degree to which force enhancement affects mechanical output and the lack of knowledge of the extent to which force depression and force enhancement cancel one another in functional movement tasks. The authors formulated a Hill-type muscle model to take into account contractile history effects on muscle. Previous Hill-type

muscle models have not taken into account contractile history. This muscle model was incorporated into a forward dynamics simulation in order to evaluate the history-dependent influence of muscle on human movement. The effects of history-dependent properties on the gluteus maximus, vastus lateralis, and soleus were examined. According to the simulation all three muscles experienced force enhancement during the active stretch. Similarly, all three muscles experienced force depression due to shortening. Specifically, the gluteus maximus was active for the duration of the motion. The active stretch began on the ascending limb of the forcelength relation and continued onto the descending limb. However, the force enhancement plateaued due to the magnitude of the stretch. The active shortening produced some force depression, but this was offset by muscle activation. Overall force enhancement and depression were similar for the three muscles. It must be remembered that force was affected by other factors such as the force-velocity and force-length relations. Finally, the authors suggested that in most submaximal activities, history dependent effects will probably be compensated by changes in muscle excitation. In other words activation is reduced during force enhancement, and activation is increased to offset force depression effects. Hence, history-dependent effects have little impact on movement dynamics (McGowan, Neptune, & Herzog, 2013).

The conclusion that contractile history has little effect on movement dynamics is consistent with the results of Herzog and Leonard (2000). Herzog and Leonard (2000) performed a study on isolated cat soleus to determine the effect of contractile history on force production after stretch-shortening and shortening-stretch cycles. One might expect that the effects of active stretching and active shortening would be summative. That is, provided that the muscle is given equal amplitude stretch and equal amplitude shortening the effect on force production would be zero. A larger amplitude stretch would out weight the effect of shortening and vice versa. Herzog

and Leonard (2000) found that this is true in the case of a shortening-stretch cycle. When the amount of shortening was greater than the stretching, a net force depression resulted. That is, the muscle produced less force than predicted by the force-length relation set forth by Gordon, Huxley, and Julian (1966). On the other hand, when the magnitude of shortening was less than the amount of stretching, a net force enhancement resulted. This summative property does not apply to stretch-shortening cycles. Herzog and Leonard (2000) found the amount of lengthening prior to shortening has no effect on the relative force depression that occurs, if the lengthening is slow. At high lengthening speeds (i.e. 256 mm/s) the force depression is increased slightly (Herzog & Leonard, 2000). Results like these in conjunction with the simulations performed by McGowan, Neptune, and Herzog (2013) strengthen the conclusion that stretch-shorten cycles will do little to increase force production of the muscle fascicles during the propulsive phase of a jump. However, jumps do not involve fixed segments, and Herzog and Leonard (2000) did not perform releases. The protocol involved fixed-end contractions. In contrast Cavagna, Dusman, and Margaria (1968) and Cavagna and Citterio (1974) performed releases on isometric contractions and muscle that was previously stretched to the same length. Cavagna, Dusman, and Margaria (1968) found the stretched muscle has the ability to perform more work for a given speed and length compared to a muscle that is released from a purely isometric contraction. This suggests that the stretched muscle produced greater force than predicted by the force-velocity curve (Hill, 1938). Similarly Cavagna and Citterio (1974) showed that the previously stretched muscle was able to pull a load equal to or larger than the isometric maximum for a given length, and the muscle was able to pull a load less than the isometric maximum at a higher velocity than described by the force-velocity curve. Taking these results into consideration, one cannot deny the force depression property of muscle. On the other hand, it appears that an aspect of force

enhancement could be a shift in the force-velocity curve toward higher forces. If this is true, then force enhancement should have an effect on segmental dynamics.

Force enhancement must impact movement. Contractile history impacts muscle force production from the sarcomere level to the whole muscle level. Similarly, *in vivo* studies show that movement history (i.e. forced flexion of the joints) results in increased joint torques for single-joint protocols as well as multi-joint protocols. As a result movements such as the countermovement or SSC could result in active muscle stretch and cause force enhancement. Therefore, in conjunction with measuring multi-articular leg extension strength via endpoint force and joint torques (objective 1), these measures should be taken during vertical jump to determine the relative force and torque enhancement during the transition phase of the jump.

Muscle-tendon Interaction

Force enhancement can only occur for a particular behavior of the muscle and its respective tendon (Figure 5). Series elasticity, in particular, is responsible for the shape of the force-length relationship (Lieber, Brown, & Trestik, 1992; Kawakami & Lieber, 2000). In essence, there is an interaction between the components of muscle that are active, the force-producing contractile element, and those that are passive, the force transmitting series elastic element. Consequently the interaction between the muscle and series elastic properties can affect both the magnitude of force and the range over which the force of the contractile element may be produced. For a given change in joint angle the properties of the elasticity will affect the length and change in length of the muscle fibers. Hence, the compliance of the aponeurosis and tendon influences the occurrence of force enhancement. Stiffness should necessitate stretch in muscle fascicles leading to force enhancement. In developing this concept one must understand what it

means for the series elastic element to be considered in series, what part of the muscle tendon unit makes the series elastic element, and the effect of compliance of the series elastic element must be demonstrated.

Conceptually the muscle-tendon unit is thought of as a two-component system comprising a contractile element, the muscle fibers, and an in-series element, the external tendon. When two structures are connected linearly the internal forces are equal throughout the system. Within this conceptual framework of muscle, the aponeurosis, which is located between the contractile element and the external tendon, is assumed to possess the same constituent properties as the external tendon. However, research shows that internal tendon, the aponeurosis, behaves mechanically different from the tendon (Lieber, Brown, & Trestik, 1992; Kawakami & Lieber, 2000). In these studies the aponeurosis is demonstrated to possess the bulk of the compliance, whereas the tendon is a stiff force transmitter. Provided that the aponeurosis is truly in-series, one may measure force in the tendon and track aponeurosis strain to calculate storage of elastic energy (Roberts, Marsh, Weyand & Taylor, 1997). This being the case, storage of elastic energy that is usually attributed to the tendon may actually be stored in the aponeurosis. On the other hand, Epstein, Wong, and Herzog (2006) suggest that the aponeurosis and tendon are not in-series. Through a series of hypothetical scenarios of increasing mathematical complexity, Epstein, Wong, and Herzog (2006) show that the force in the aponeurosis varies at different locations along the muscle. Consequently, internal forces of the aponeurosis and tendon are different. As a result studies that measure tendon force and subtract fascicle length from whole muscle-tendon unit length to obtain strain in the tendon are misleading. The change in length may occur in the aponeurosis, not the tendon. Furthermore, use of the tendon force and constitutive equations of tendon to calculate energy storage in the tendon results in

miscalculations, if the majority of strain occurs in the aponeurosis (Epstein, Wong, & Herzog, 2006). Furthermore, this has implications for the muscle-tendon interaction as the aponeurosis may be the element of muscle determining the length change of fascicles. Provided that Epstein, Wong, and Herzog (2006) are correct, there is much work to be done before *in vivo* properties of tendon and aponeurosis are fully understood.

However, much research suggests that the tendon is compliant. Compliance is made intuitive from the wavy pattern, visible upon magnification, present within the fibrils of tendon (Diamant, Keller, Baer, Litt, & Arridge 1972). Furthermore, this pattern of oscillations within relaxed tendon is due to collagen with a crimp that deforms in response to applied stress (Diamant, Keller, Baer, Litt, and Arridge, 1972; Rigby, 1959). Collagen crimp is the culprit for the toe region within the stress-strain curve for tendon. This region is characterized by an increasing rate of change of stress with respect to strain (Diamant, Keller, Baer, Litt, and Arridge, 1972; Rigby, 1959). Beyond the toe region collagen acts as a spring; it behaves in a Hookean manner. Once the tendon is strained enough that its response to stress is Hookean, the stress-strain curve takes the shape of a line. Beyond this Hookean portion to the stress strain curve the tendon begins to fail. Rigby (1959) suggests the wavy pattern may act as system designed for energy absorption.

Ability to absorb energy could be useful in locomotion. Ker (1981) investigated the mechanical properties of sheep tendon, suggesting that tendon properties affect locomotion. Similarly, knowledge of the tendon response to loading is vital for expanding knowledge of locomotion. Ker (1981), in an effort to simulate physiological loading, subjected the sheep tendon to cyclic loading ranging from 0.22 to 11 Hz. Ker (1981) assumed the experimental range of loading frequencies to simulate the frequencies experienced by sheep when walking. Within

this range of frequencies the sheep tendon tangent modulus was not dependent on frequency. However, tangent modulus varies between tendons. Ker (1981) found tendons to dissipate 7% of the energy stored during strain in their return to original form, which compares with 9.2% hysteresis measured in digital flexor of swine (Shadwick, 1990). Furthermore, this relatively small hysteresis was independent of the range of frequencies applied. Another group suggested that hysteresis is independent of loading frequency up to 70 Hz (Wang, Ker, & Alexander, 1995). Hysteresis is suggested to allow tendon to effectively absorb and store energy for locomotion at various speeds. Average tangent modulus was 1.65 GN/m² for sheep tendon. This is similar to the 1.66 GPa measured by Shadwick (1990) in swine digital flexor tendon. While Ker (1981) assumed that tendon properties affect locomotion, Shadwick (1990) suggested that locomotion affects tendon properties. This was due to the fact that the digital extensor in swine, which is not load bearing, exhibited a tangent modulus of roughly half and hysteresis of nearly double the digital flexor. Shadwick (1990) and Ker (1981) showed that under large enough loads tendons behave in a nearly Hookean manner. Therefore, elastic energy storage is possible once the crimp is pulled out of collagen and the material undergoes strain. Furthermore, the tendon is compliant, especially in the toe region, and the mechanical properties of tendon appear to be tuned to physiological demands (Ker, 1981; Shadwick, 1990).

While tendons are adapted to mechanical demand, they are also adapted to reduce metabolic cost. Alexander (2002) argues that tendons have the potential to store elastic potential energy. Compliant tendons allow the muscle to behave nearly isometrically in stretch-shortening cycles, which means the change in length of the muscle tendon unit is absorbed by the tendon. Provided that the tendon behaves elastically, the elastic recoil after stretch may allow for higher forces than would occur with muscle fascicle shortening. Also because tendon is passive there is

no metabolic cost associated with its shortening. Alexander cites Biewener and Blickhan (1988) as a contrasting study in which kangaroo rats exhibited fascicles that lengthened and subsequently shortened during hopping. In essence kangaroo rats employ stiff tendons for jumping and rapid accelerations. With stiff tendons fascicles lengthen and subsequently shorten during stretch-shortening cycle motions. Alexander (2002) suggests that cases in which the fascicles shorten are not metabolically optimal as it requires energy to shorten the fascicles. Therefore, stiff tendons are not optimal for hopping, in which minimizing energetic cost is important (Alexander, 2002).

In addition to minimizing energetic cost, tendons are generally believed to augment power, and this has been extended to the idea that tendons optimize vertical jump (Alexander, 2002). A model by Alexander (2002) demonstrated that tendon elasticity is utilized in both SJ and CM. However, the CM allows the tendon to be stretched without fascicle shortening. This allows the fascicles to remain closer to optimum length and produce greater force. Muscle properties necessitate a power amplifier, which Alexander (2002) touts is tendon, in order to generate the power outputs seen in vertical jumping.

As Alexander (2002) suggested the power amplification process occurs through the storage of elastic potential energy in the tendon. Therefore the tendinous tissue must experience strain. Arampatzis, Stafilidis, DeMonte, Karamanidis, More-Klapsing, and Bruggemann (2005) measured strain in the aponeurosis and tendon of the gastrocnemius during maximal voluntary plantarflexion. Plantarflexions were performed with a dynamometer and ultrasound was used to evaluate the change in length of the aponeurosis and tendon. Similar magnitudes of strain were found between the aponeurosis and the tendon (5.12±2.07% and 4.72±1.85%, respectively). These strains are comparable to a strain of 6.9% measured in the patellar tendon (Hansen,

Bojsen-Moller, Aagaard, Kjaer, & Magnusson, 2006) and aponeurosis strain of 6.5% in the tibialis anterior (Maganaris & Paul, 2000). Even though a change in joint angle of 7-9° occurred due to relative motion of the foot and dynamometer compliance, the aponeurosis and tendon elongations were 16.3±5.8mm and 10.9±3.3mm, respectively. These changes in length demonstrate that the tendinous tissues are compliant and could potentially store elastic energy. A large length change in the tendinous tissues compared to small ankle joint angular change suggests series compliance allows fibers to shorten even during isometric muscle actions (Arampatzis, Stafilidis, DeMonte, Karamanidis, More-Klapsing, & Bruggemann, 2005). Hence the results of *in vivo* studies insinuate that tendons are compliant and may augment power by allowing the fascicles to remain nearly isometric or shorten as the tendon undergoes strain. Once the tendon is lengthened it has the potential to recoil elastically.

Much of the research on tendons and aponeuroses suggests that both are compliant.

Typically the aponeurosis is found to be more compliant than the external tendon. However, until it is determined whether the aponeurosis is an in-series component versus a parallel component of the muscle, the amount of potential energy stored cannot be determined (Epstein, Wang, & Herzog, 2006). Nonetheless, it is clear that strain occurs and some potential energy is stored.

Compliance is important to this study, as it suggests that in many movements muscle fascicles will shorten or remain isometric during the force producing stages. In such cases force enhancement at the muscular level does not occur. However, force enhancement may occur under the right conditions.

First, muscle structure likely plays a role. Alexander and Ker (1990) examined musculature of the limbs of 40 mammals ranging from small rodents (0.1 kg body mass) to large elephants (2500 kg body mass). From the examination 3 types of muscles were identified. Type

1 muscles have long fascicles and are located on the proximal limb. Type 1 muscles are the largest muscles of the limb. These muscles are capable of performing a large amount of work because of cross sectional area and length. Additionally type 1 muscles have short tendons, which contribute very little to the work performed. Tendons of type 1 muscles merely transmit force. Type 2 muscles have thick tendons that do not experience a great deal of stress. These tendons are not involved in weight bearing and typically control joints far from the muscle belly. Type 3 muscles have long tendons that undergo a great deal of stress. The tendons of type 3 muscles are useful in saving energy due to compliance, which allows storage and release of potential energy (Alexander & Ker, 1990). More time might be required to strain tendons of type 1 muscles, whereas more proximal muscles have less compliance to remove. Furthermore, short tendons may allow large accelerations (Biewener, & Blickhan, 1988), whereas long compliant tendons may serve to take on the length change (Roberts, Marsh, Weyand & Taylor, 1997).

For example, Roberts, Marsh, Weyand, and Taylor (1997) used sonomicrometry crystals to measure changes in muscle fiber length in running turkeys. Strain gauges were implanted to measure force of the lateral gastrocnemius. Large changes in fiber length occurred during swing phase as force was produced to negatively accelerate the foot. Much of the negative work was done passively and the muscle stretched beyond optimal length. Azizi and Roberts (2010) found that frog fascicles are initially on the descending limb of the force length relation. This allows the muscle to shorten onto its plateau, yielding higher peak forces compared to a muscle initially on the plateau. This suggests that the passive stretch beyond optimal length may benefit turkeys in force production during running. During level running the turkey muscle generated force up to 35% of its maximum isometric force at the fastest speeds. At the fastest speed the muscle shortened by 6.6±1.9% of its optimal length, although the fascicles behaved nearly isometric.

Tendon energy storage contributed 60% of the work output by the muscle-tendon complex. Most of the strain occurred in aponeurosis. In other words large tendon compliance disallows force enhancement in running turkeys. When turkeys ran on inclined surfaces work performed by muscle fascicles increased in proportion to the incline. Increased activation resulted in the necessary force to run on the incline (Roberts, Marsh, Weyand, & Taylor, 1997).

In contrast to running, confining muscle fascicle lengths to the optimal length is not likely to produce great jumps (Azizi & Roberts, 2010). This is because to perform high workloads muscle should shorten over a great length. This is amply demonstrated by Azizi and Roberts (2010) who implanted electrodes and sonomicrometry crystals into the plantaris of bullfrogs. Motion capture data were also collected of multiple jumps from each frog. After the jumping was performed an *in vitro* preparation of each frog's plantaris was used to measure the force-length relationship. During jumping no countermovement was observed. The plantaris activated 42 ms prior to movement and shortened by 10% during that time. Fascicles continued to shorten during the jump for a total shortening of 25-30% of optimal length. Therefore, in this study muscle fibers did not lengthen. During in vitro examination, passive force was not evident until the muscle was stretched to 120% of optimal length. In vitro examination revealed that the muscle fascicles operated on the descending limb and plateau of the force-length relation. On average at rest fascicle lengths were 130% of optimal length, and at take-off fascicle length was optimal on average. An important result is that muscles that shorten from the descending limb to the plateau of the force-length relation are able to produce higher peak forces than muscles that shorten onto the ascending limb. The authors posit that muscles beginning on the plateau shorten onto the ascending limb early during activation, which leaves the muscles on the ascending limb once peak force is reached. In contrast muscles beginning on the descending limb are at the

plateau once peak force has been reached. High peak forces must be developed for optimal storage of elastic energy in tendons. It appears the long initial lengths of the frog fascicles are due to compliant passive elasticity existing in parallel to muscle fibers. Mammals exhibit higher passive tensions earlier in the range of motion. This may prevent mammals unlike frogs from using the descending limb (Azizi & Roberts, 2010). The results of this study extrapolated to humans suggest that stretch of the muscle fibers as a result of a countermovement may be beneficial in performing jumps, especially since it has been shown that lower extremity muscles tend to operate on the ascending limb of the force-length relationship (Rubenson, Pire, Loi, Pinniger, & Shannon, 2012). Clearly a stretch in the fibers preceding shortening would provide a greater length to shorten and perform work in movements involving jump (Figure 5).

Muscle-tendon Interaction

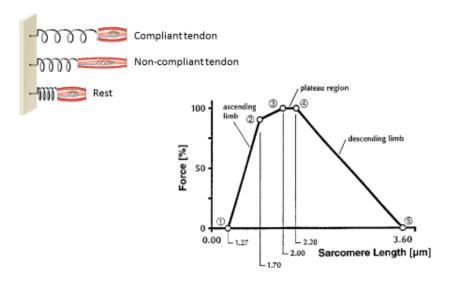


Figure 5: Muscle-tendon interaction. This figures demonstrates that a stiff tendon will cause muscle fiber stretch in an SSC movement. By changing the length of the muscle fiber, its force capacity is altered in part due to it force-length relation. Adapted from Rassier, MacIntosh, and Herzog (1999).

However, a stiff series-compliance is necessary to elicit muscle fiber stretch (Figure 5). Biewener and Blickhan (1988) suggested that the small kangaroo rat, exhibiting large muscles and tendons relative to its size, is not built for elastic energy storage. Little strain energy was recovered in the hopping of these animals. Rather than minimizing energy cost, this animal is built for rapid accelerations (i.e. jumping). During steady speed hopping tendon force increased with speed. The muscle stress for the highest jumps was 1.75 times the peak isometric stress. The authors suggest force enhancement allowed the muscle to generate such high stresses. The highest jumps were generally preceded by landing from a previous jump, which would increase the rate of eccentric load enough to cause muscle stretch (Griffiths, 1991). Furthermore, the relative compliance of the tendons in kangaroo rats is less, meaning they are not able to store as much elastic energy as longer, compliant tendons. Fascicles are able to shorten during loading provided the tendinous tissue is compliant. Therefore, it seems probable that muscles connected to stiff tendinous tissue are likely to undergo force enhancement and provide a means for large accelerations. On the other hand muscles connected to compliant tendons will allow muscles to remain nearly isometric during loading and conserve energy (Alexander, 2002; Ker, 1988).

In humans the effect of tendon compliance has been examined during a countermovement versus no countermovement (Kawakami, Muraoka, Kanehisa, & Fukunaga, 2002) jump.

Participants performed the countermovement and no-countermovement conditions on a horizontal, sliding table. The load of the countermovement was approximately 40% of maximal voluntary force. Results from ultrasound of the gastrocnemius showed that muscle fascicles initially lengthened passively. Further lengthening occurred in the whole muscle-tendon unit, but the change in length occurred in the tendon (Kawakami, Muraoka, Kanehisa, & Fukunaga, 2002). Then the whole muscle-tendon shortened during plantarflexion. Similarly Kurokawa,

Fukunaga, Nagano, and Fukashiro (2001) showed that muscle fascicles of the gastrocnemius fascicles shortened, remained isometric, and shortened again during an upright countermovement jump. Both groups suggested that tendon compliance allowed storage of elastic energy, which resulted in increased power (Kurokawa, Fukunaga, Nagano, & Fukashiro, 2001). Conversely, studies examining more proximal musculature and higher eccentric loading suggest that force enhancement may play a role in human movement. Ishikawa, Komi, Finni, and Kuitunen (2006) examined muscle-tendon interaction of the vastus lateralis during drop jumps on an inclined sled. Active muscle fascicle lengthening increased for higher drops, which cause greater eccentric load. Braking phase was shortest for the highest drop condition. Furthermore, tendon shortening began early in the push-off as a result the higher rate of force development (Ishikawa, Komi, Finni, & Kuitunen, 2006). In other words higher eccentric load not only led to active stretch of fascicles, but it also altered the timing of the muscle-tendon interaction.

This suggests that tendon behavior depends on rate of strain. Wren, Yerby, Beaupré, and Carter (2001) subjected human Achilles tendons to different strain rates. Failure loads were measured at 4617±17N and 5579±1107N for 1%/s and 10%/s strain rates, respectively. Mean failure stress in this study was 86 ± 24 MPa and 71 ± 17 MPa for the 10%/s and 1%/s strain rates, respectively. This was greater than previously reported in human Achilles tendon due to higher cross sectional areas of the material in this study. Failure occurred at strains of 16.1±3.6% and 12.8±1.7% for the faster and slower rates. Clearly, greater strain rate led to increased failure load, failure stress, and failure strain. Similarly, in an *in vivo* study Earp, Newton, Cormie, and Blazevich, (2013) found that high loads caused the patellar tendon to behave with increased stiffness. Loads of 20, 60, and 90% of one repetition maximum knee extension were applied during a knee SSC. Eccentric phase was shortest in the 90% load, which is similar to Ishikawa,

Komi, Finni, and Kuitunen (2006). Additionally rate of force development was highest during the greatest load. Tendon lengthening was greatest in the 20% load condition, while rate of force development was less. Hence, the authors concluded that tendons appear to behave as stiff force transmitters during high loads (Earp, Newton, Cormie, & Blazevich, 2013). Tendinous behavior may change depending on the load, which will change due to movement history. Therefore, movement history and loading affect muscle-tendon interaction and its timing.

Electromechanical Delay

The optimal way to demonstrate *in vivo* change in muscle-tendon interaction or altered muscle-tendon timing is through ultrasound techniques. Given that ultrasound instrumentation is not available for this study an alternative measure is necessary. Since tendon behavior depends on rate of load, a time variable may provide a characteristic distinction between muscle-tendon interactions in various loading conditions. For example, studies involving increasing load intensity show decreased eccentric time and time to tendon shortening or altered muscle-tendon behavior (Ishikawa, Komi, Finni, & Kuitunen, 2006; Earp, Newton, Cormie, & Blazevich, 2013). Furthermore, Hirayama, Yanai, Kanehisa, Fukunaga, and Kawakami (2012) found differences in the time between minimum force and onset of EMG in countermovement jumps after practice sessions. Reasonably it is likely that differences in loading intensity will result in shortening or lengthening of the interval between onset of EMG and angular acceleration of a limb segment. Electromechanical delay (EMD) or a similar measure is an appropriate measure of altered muscle-tendon behavior.

An early study pointing toward electromechanical delay was performed by A. V. Hill (1949) to examine the mechanics of muscle just after a maximal shock and a subsequent stretch.

The quick stretch after stimulation allowed Hill to measure the rate of rise of active state after the latent period. He noted that "... a force cannot be manifested externally until the contractile component has shortened enough to raise the tension in the series elastic component (SEC); and shortening is a slow process" (Hill, 1949, p. 401). The muscular force shown in this study was dependent on the amplitude and the timing of the applied stretch. Subsequently, Hill (1951) stated that muscular force is dependent on two factors: 1] the load-extension relation of the series elastic component and 2] the force-velocity characteristics of the contractile portion. To validate these statements Hill (1951) added compliance to the tested muscle. With added compliance the developed tension decreased and the duration to peak tension increased, which reciprocates Hill's (1949) study involving a quick stretch. Without compliance tension develops nearly immediately. With a more compliant series elastic component it is probable that a decrease in the developed tension resulted from a greater shortening of the contractile component (Abbott and Aubert, 1952). This is the converse result to Hill (1949) in which a quick stretch was applied, negating the series elastic effects, soon after stimulation. A quick stretch during the rise of active state could have two different effects, depending on the amplitude of stretch. First, if the stretch amplitude is only enough to negate the series compliance, the stretch simply prevents force depression (Abbott and Aubert, 1952) (i.e. no force is lost due to shortening of the contractile component for drawing slack out of the series elastic element). If the stretch amplitude is large enough and timed appropriately, the contractile element may be stretched also and exhibit force enhancement (Abbot and Aubert, 1952). When a stretch was applied the muscle produced greater force compared to the isometric force curve, and when compliance was added the magnitude of shortening was greater (Hill, 1951). In essence a compliant series elastic element results in force depression and an appropriately timed stretch results in force enhancement. Results from added

compliance demonstrate that series elasticity exists, and that the degree of its compliance determines the magnitude and duration to peak tension. Depending on the goal an optimal compliance may exist for certain movements. It may be implied that the countermovement is equivalent to Hill's (1949) quick stretch in that it eliminates a portion of the series elastic compliance thereby preventing force depression or causing force enhancement.

One of the early studies suggesting that EMD was due to series elasticity was performed by Cavanagh and Komi (1979). The authors referred to the time interval between onset of muscle activation, measured by electromyography, and onset of force as the electromechanical delay (EMD). To quantify the interval a dynamometer oscillated the elbow passively through extension and flexion at 0.5 rad/s. Upon seeing a stimulus, when the forearm reached 110 degrees, the participant maximally contracted the elbow flexors. Concentric muscle actions consistently yielded a significantly longer EMD compared to eccentric muscle actions. The authors concluded that EMD in the eccentric trials was shorter due to a more rapid stretch of the SEC. Furthermore, it was asserted that faster oscillations of the dynamometer would cause greater differences in EMD values. Such results were validated by Norman and Komi (1979), who used a similar approach to study EMD in the elbow flexors and extensors. However, the speed of the eccentric condition was varied. Eccentric EMD was shorter than concentric, but faster eccentric motion resulted in a shorter EMD compared to slower eccentric motion for the biceps brachii. The triceps brachii EMD was similar in both the slow and fast eccentric conditions. The more rapid stretch applied in the fast eccentric condition likely pulled slack out of the SEC more quickly. In this way the conclusions of Cavanagh and Komi (1979) are confirmed. The consequence may be that a more rapid eccentric phase in a countermovement results in shorter EMD, indicating rapid development of tension in the series elastic component. Rapid

development of tension in the series elasticity causes stiff behavior, which may lead to force enhancement.

Other more recent studies examined either concentric or eccentric EMD. For example, Osigo, McBride, and Komi (2002), examined the effect of muscle contractile history on stretch reflex latency in the plantarflexors. Muscle histories included pre-isometric, pre-shortening, and pre-lengthening muscle actions, which occurred at 0%, 35%, and 50% of maximal voluntary contraction (MVC) torque. The pre-isometric condition exhibited the shortest EMD, which is counterintuitive based on the results of previously mentioned studies. However, differences were not significant. The authors attributed this result to stability of filament overlap in pre-isometric conditions prior to stretch as compared to the dynamic muscle histories. EMD ranged from 5ms to less than 20ms. Duration of eccentric EMD in the soleus in Mackey and Robinovitch (2006) was measured during balance recovery. Participants achieved a maximum forward lean and were released from a tether. Upon release participants were expected to maximally contract the plantarflexors to recover balance. The authors reported EMD values of 27 ± 14ms for young participants and 29 ± 15ms for older participants.

Discrepancies in the EMD values between these two studies may be due to sample size differences. Osigo, McBride, and Komi (2002) recruited 7 male participants, whereas Mackey and Robinovitch (2006) recruited 25 females. Additionally, differences between the sexes in tendon stiffness and elongation under load have been reported (Kubo, Kanehisa, & Fukunaga, 2003), which might explain the greater EMD values reported by Mackey and Robinovitch (2006).

Additional discrepancies are revealed in looking at the literature on concentric EMD measurement. First, a study comparing voluntary contractions to supramaximally stimulated

contractions reported concentric EMD to be 22.8 ± 8.2 ms and 9.7 ± 3.1 ms for voluntary and involuntary contractions of the plantarflexors, respectively (Hopkins, Feland, & Hunter, 2007). The authors concluded that supramaximal stimulation provided greater recruitment of fast twitch muscle fibers, which would impose a greater rate of stretch on the SEC and decrease EMD duration. This contrasts with the concentric isokinetic contractions of the elbow flexors performed in Howatson (2010) and Howatson, Glaister, Brouner, and van Someren (2009). In both studies EMD was measured in isometric and concentric isokinetic elbow flexion at 60°s⁻¹ and 210°s⁻¹. Howatson, Glaister, Brouner, and van Someren (2009) measured EMD in the dominant versus non-dominant arms to determine the reliability of the measure. Differences between dominant and non-dominant arms were not significant. However, concentric isokinetic muscle actions at both 60°s⁻¹ and 210°s⁻¹ resulted in significantly longer EMD. Isometric EMD in the dominant arm was measured as 57.2 ± 5.4 ms. Slow and fast isokinetic exercises resulted in values of 72.3 ± 8.9 ms and 70.4 ± 7.1 ms (Howatson, Glaister, Brouner, & van Someren, 2009). It is expected that the isometric condition would have a shorter EMD duration due to a constant muscle-tendon length, which does not act to add slack.

Overall the literature indicates that eccentric muscle actions will have a shorter EMD when compared to concentric muscle actions. This is due to the limb segment moving counter to the muscle force which speeds up the removal of slack in the series elastic components of the muscle. One study (Hopkins, Feland, & Hunter, 2007) was presented that provided results contradictory to the notion that eccentric EMD is shorter. However, this was likely due to the measurement being taken on the weight-bearing gastrocnemius. Additionally, when comparing the results of Hopkins, Feland, and Hunter (2007) to Osigo, McBride, and Komi (2002), a study examining eccentric EMD in the plantarflexors, one will notice that EMD values are longer in

the voluntary concentric condition (22.8 ± 8.2 ms) as compared to the voluntary eccentric condition (<20 ms) of Osigo, McBride, and Komi (2002).

Clearly, movement history affects EMD. That is, eccentric motions yield shorter EMD compared to concentric. Results show the magnitude of EMD is dependent on the rate of change of joint angle also. High angular velocities will result in high rates of stretch of the muscle tendon unit, which is suggested to result in stretch of muscle fibers (Griffiths, 1991). In research on muscle-tendon interaction in human movement research rapid eccentric motions result in high rate of force development, stiff behavior of tendons, and force enhancement (Ishikawa, Komi, Finni, & Kuitunen, 2006; Earp, Newton, Cormie, & Blazevich, 2013). Therefore, loading conditions of increased intensity should demonstrate shorter EMD values.

In addition to rate of loading, EMD may be affected by the angular position of the limbs, as this will affect the length of the muscle at onset of muscle activity. For example, studies have manipulated joint angle in a stepwise fashion to determine the length dependence of EMD. For example Muraoka, Muramatsu, Fukunaga, and Kanehisa (2003) electrically stimulated the gastrocnemius after placing the ankle at various joint angles (-30°, -10°, 0°, and 5° where negative denotes plantarflexion). EMD at -30° was significantly longer than at -10°, 0°, and 5°. EMD at -20° was significantly longer than at 0° and 5°. There were no significant differences between -10°, 0°, and 5° where the tendon was longer than the tendon slack length observed at -16°. Relative EMD, normalized to max EMD for each subject, was negatively correlated with medial gastrocnemius tendon strain. Therefore, EMD decreases while muscle-tendon length increases until tendon slack is taken up (Muraoka, Muramatsu, Fukunaga, & Kanehisa, 2003).

Similarly, Morse, Thom, Birch, and Narici (2005) demonstrated that elongation of the tendon affects the duration of EMD. Tendon elongation was measured at -20°, 0°, and 20° in

young and elderly men. Again, negative angular values represent plantarflexion angles. Tendon elongation was significantly greater in elderly men at all joint angles. Hence, this study also demonstrates that EMD is positively correlated with the elongation of the muscle tendon unit.

Sasaki, Sasaki, and Ishii (2011) also varied joint angle of the elbow to study EMD. Ten different joint angles were examined. EMD was significantly affected by decreases in joint angle below 90°; however, biochemical processes were not affected by joint angle. Based on a muscle model used in this study, angles greater than 90° added slack to the muscle. At angles greater than 90° the time to transmit force through the SEC was no longer length dependent as EMD was not significantly different between these angles (Sasaki, Sasaki, & Ishii, 2011). Clearly, angular position that lengthens the muscle tendon unit acts to decrease EMD as some compliance will be removed.

It should be noted that the much larger values for EMD in Esposito, Limonta, and Ce (2010) are due to methodology of stimulation. Costa et al. (2010) used supramaximal stimulation, whereas Esposito, Limonta, and Ce (2010) applied tetanic stimulation to the motor point of the gastrocnemius. Such inconsistencies in results require that researchers understand the effects of stimulation on EMD. Lacourpaille, Nordez, and Hug (2013) performed a study evaluating the effect of six stimulation intensities (30%, 50%, 70%, 90%, 110%, and 130%). The results showed that EMD was longer at stimulus intensities of 30% and 50% of maximal stimulation. No differences were demonstrated between higher intensities from 70% to 130%. Therefore, the brief EMD periods recorded by Costa et al. (2010) compared to Esposito, Limonta, and Ce (2010) are likely due to stimulus intensity differences. Such differences point toward a neural drive component to EMD.

Electromechanical delay is a temporal characteristic of force transmission through the muscle. There is little research geared toward studying different muscle actions on the basis of EMD. This is due to the fact that under a given set of conditions eccentric EMD is shorter than concentric EMD based on a more rapid removal of muscle-tendon slack. Additionally, this removal of slack is rate dependent. Lastly, Nordez, Gallot, Catheline, Guevel, Cornu, and Hug (2009), using ultrasonography, have effectively fractionated EMD. However, this fractionation points out that mechanical properties of the muscle fibers, aponeurosis, and tendon play vital roles in the EMD. Therefore, further research should be done to understand the contribution of these parts of the muscle-tendon to EMD. The partitioning of EMD may provide insight into the mechanical properties of muscle-tendon components, which could be helpful in unraveling the mysteries of muscle force production, particularly in loading conditions of varying intensity.

Therefore, various loading intensities should be employed in SSC movements. Varying the loading intensity should impact the muscle-tendon interaction. If the loading rate is high enough the tendon may behave in a stiffer manner, which will result in a decreased electromechanical delay. Conversely, decreased loading rates will allow the tendon to behave in a more compliant manner, which will increase EMD. Since decreased EMD suggests a stiff tendon, it is likely that loading conditions which decrease EMD will result in force enhancement, although timing of stretch is important (Hill, 1949).

Countermovements

A plethora of research exists demonstrating that utilization of a countermotion or SSC provides an improvement in performance over no countermotion or the squat jump (SJ) (Bobbert & Casius, 2005; Bobbert, Gerritsen, Litjens, & Van Soest, 1996; Moran & Wallace, 2007;

McBride, McCauley, & Cormie, 2008; Kopper, Csende, Sáfár, Hortobágyi, & Tihanyi, 2013; Arakawa, Nagano, Yoshioka, & Fukahsiro, 2010). In contrast, little research exists to examine mechanisms of improved performance in novel countermotions. The "split step" used in tennis has been examined and found to improve reaction time and increase ground reaction forces in a step and reach task (Nieminen, Piirainen, Salmi, & Linnamo, 2013; Uzu, Shinya, & Oda, 2009). Increased EMG was found in the split step compared to no split step. The usefulness of the split step is demonstrated by its use in the arena. Another technique has been witnessed in canines (Angle, Gillette, & Weimar, 2012). Canine sprinters flex the joints of the lower extremity to remove their paws from the ground by 4-8 cm. This results in a brief aerial phase without upward movement of the center of mass. Hence the canines free fall into a countermotion or demonstrate a self-induced drop jump (SD) (Angle, Gillette, & Weimar, 2012). Mechanisms for this SD have not been examined in the literature.

Conversely, mechanisms for the traditional countermovement have been examined extensively. Mechanisms for the efficacy of the countermovement are: 1] stretch reflex, 2] coordination, 3] tendon elasticity, 4] active state, and 5] force enhancement (Bobbert & Casius, 2005; Arakawa, Nagano, Yoshioka, & Fukahsiro, 2010). While each of these mechanisms may play a role in any SSC technique, this study suggests the fifth mechanism is the most important in the self-induced drop for the following reasons. First, the stretch reflex is believed to increase stimulation and force production of the muscle, but differences in EMG are not always found between drop jumps and the countermovement (Bobbert, Huijing, van Ingen Schenau, 1987; Walshe, Wilson, & Ettema, 1998). If stimulation is increased due to stretch reflex, muscle activation should be increased in conjunction with force. While the stretch reflex is likely present, necessary activation to perform vertical jump may be high enough that the stretch reflex

is hidden (Walshe, Wilson, & Ettema, 1998). Secondly coordination implies that muscles are operating in more favorable regions of the force-length and force-velocity relations. As a result one is forced to turn to muscle properties as a mechanism, since muscles actuate human movement (Bobbert & Casius, 2005). Third, some research suggests that tendon elasticity is utilized even when no stretch-shorten cycle is used (Anderson & Pandy, 1993; Kurokawa, Fukunaga, & Fukashior, 2001). Additionally, not all muscles involved in vertical jump have appropriate architecture for exploiting tendon elasticity (Alexander & Ker, 1990). Furthermore, if increased force may be produced then the tendon may be stretched more. Increased stretch of the tendon will allow increased storage of elastic potential energy. Therefore, muscle force is the mechanism, which necessitates active state, the fourth mechanism. Muscle force increases with an increase in active state (Hill, 1949). Bobbert and Casius (2005) argue that the traditional countermovement provides the timing necessary for greater active state to develop, which allows muscles to produced greater force and increase acceleration of the limb segments. This study, however, suggests that a well-timed, rapid stretch can remove compliance from the series elasticity and stretch the fibers such that force enhancement occurs (Hill, 1949; Hill, 1951). Force enhancement will optimize segmental accelerations.

Therefore, the SD technique should be compared to the CM and (SJ) conditions. The SD technique may result in increased vertical acceleration compared to the CM and SJ conditions.

Increased acceleration could come from exploitation of the force enhancement property.

Summary

Muscle actuates human movement. Movement is affected by the amount of force that can be produced by the muscle. Traditionally, muscle force is believed to be governed by the force-

length relation, which was first expressed by Gordon, Huxley, and Julian (1966). Because muscle length changes with joint angle, angular position of limbs affects the amount of force that the muscle may produce. Consequently, this also affects the moment produced about the joint. Thus, the force-length relation is important for understanding human movement.

However, research has exposed the force-length relation as incomplete. Muscle contractile history alters the amount of force that may be produced by the muscle. For example, active muscle stretch increased the amount of force produced above that predicted by the force-length relation (Rassier, MacIntosh, & Herzog, 1999). Consequently, active muscle stretch will lead to increased joint moments for a given angular position of the limb of interest. This phenomenon has been shown to occur in multi-articular leg extensions.

The fact that force enhancement occurs in multi-articular tests suggests that force enhancement may play a role in lower extremity SSC, particularly the CM. However, an appropriate muscle-tendon interaction is necessary to elicit force enhancement at the muscular level. In other words tendon compliance must be removed, or the compliance will allow muscles to shorten or remain isometric in SSC movements. In such a case force enhancement will not occur. On the other hand, movements involving high eccentric loads exploit the viscoelastic properties of tendons causing stiff behavior. This may result in stretch of the fibers, which causes force enhancement.

Force enhancement at the muscular level should result in increased torque about the primary joints contributing to the motion. If torque increases, segment orientation will determine the amount of endpoint force generated. The ground produces an equal force opposite in direction. This ground reaction force acts to accelerate the center of mass of the body in SSC.

Greater endpoint forces result in greater accelerations. Therefore, force enhancement may be a mechanism for SSC.

Mechanisms for SSC have been examined in the CM jump. Several mechanisms for the CM exist, but force enhancement has not been shown. However, force enhancement is implied in ultrasound studies examining drop jumps. Therefore, force enhancement may have a role in the drop jump. The goal of this study is to examine various loading conditions to determine if force enhancement is a mechanism for increased vertical acceleration in the vertical jump.

CHAPTER III

METHODS

Project objectives were, first, to establish a curve relating force and torque to leg extension and joint angles during upright, multi-articular, maximum effort leg extension. Second, force and torque during three countermovement conditions were evaluated relative to the force and torque versus leg extension curves. Third, variations in muscle-tendon behavior due to the three SSC conditions were examined by measuring EMD. Finally, performance measures such as jump height, vertical take-off velocity, rate of force development, and impulse were compared among the three SSC conditions.

To fulfill these objectives appropriate methods are necessarily prescribed. The role of this chapter is to outline and describe the methodology. Sections describing the methodology are as follows: 1] participants, 2] setting, 3] materials, 4] instrumentation, 5] design and procedures, and 6] data analysis.

Participants

Participants recruited as volunteers for this study included male and female students from Auburn University ranging from 19 to 35 years old. Participants were in good health and without lower extremity injury or surgery in the past year. Participants were required to complete a health screening questionnaire prior to inclusion (Appendix A). Exclusion criteria included: 1) any current or recent injury to the lower extremity, pelvis, low back, or trunk, within the last year, 2)

any previous injury/illness that might prevent completion of the required tasks, 3) any allergies to adhesives, and 4) any inner ear disturbance or susceptibility to loss of balance. Participants were required to affirm the voluntary nature of their participation by signing an Informed Consent document approved by the Auburn University Institutional Review Board (Appendix B).

Setting

Data collection took place in the Sports Biomechanics Laboratory (003 Kinesiology Building) at Auburn University.

Materials

Power Rack

A power rack was utilized to perform isometric leg extensions, which provided reference force and torque values necessary to evaluate force and torque enhancement (Figure 6). Racks were turned upside down to provide a mechanism that prevented upward movement of the bar. Racks were lowered or raised to adjust the height of the hip and alter ankle, knee, and hip angles. Each isometric leg extension provided force data that were plotted against the height of the hip relative to the floor. Also, each isometric leg extension allowed the calculation of joint moments at the ankle, knee and hip, which were plotted against joint angle. These data were necessary to determine if the force for a given hip height in the transition phase of a vertical jump is greater than isometric force produced at the same hip height. Similarly, torque enhancement was tested by comparing whether the torque at a given angle during the transition phase was greater than the isometric torque produced at the same angle.

Previous work has established force-leg length curves and torque-angle curves using a leg extension dynamometer (Hahn, 2008; Hahn, Olvermann, Richtberg, Seiberl, & Schwirtz, 2011, respectively). One advantage of the leg press dynamometer is increased control over leg length and joint angle change. On the other hand, it may be easier to approximate the body segment configurations of vertical jump in an upright leg extension task performed in a power rack. Furthermore, the dynamometer was set such that leg extensions were performed horizontally (Hahn, 2008; Hahn, Olvermann, Richtberg, Seiberl, & Schwirtz, 2011, respectively). In a horizontal leg extension gravitational forces accelerate the legs into extension. However, in vertical jump gravitational forces act to accelerate the legs into flexion. Therefore, the power rack has the advantage of gravitational forces working against leg extension, which more closely approximates vertical jump dynamics.



 $Figure\ 6.\ Example\ of\ a\ power\ rack.\ http://www.powerhouse-fitness.co.uk/bodymax-zenith-line-power-rack.php$

Instrumentation

Kinematics

To evaluate vertical jump dynamics a numerical description of the motion was gathered with a 10-camera Vicon® MX motion capture system (Vicon®, Los Angeles, CA, USA) with a sampling frequency of 200 Hz. Cameras were arranged to capture motion within a volume 2.5 m high, 2 m wide, and 2 m deep (Figure 4). Fifty-four, 14 mm, retroreflective markers (MKR-6.4, B&L Engineering, Tustin, California, USA) were attached to anatomical landmarks of the lower extremity, pelvis and trunk (Figure 5, Figure 6, Table 1) to enable the calculation of segment orientation in three-dimensional space. Markers were secured using double-sided tape (Duck Tape®, ShurTech Brands, Avon, OH, USA). Segment orientation and joint angles were calculated using Visual 3D (C-Motion Research Biomechanics, Germantown, Maryland, USA). Additionally, the time rate of change of segment orientation provided angular velocities of the foot, shank, and thigh. The time series of angular velocities and the times series of muscle activation allowed the determination of EMD. Segment orientations in conjunction with kinetic data provided the necessary information to obtain joint moments.

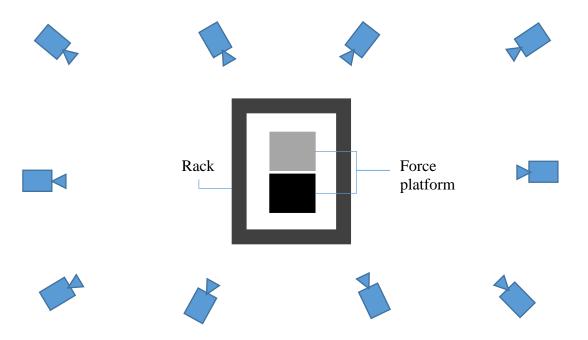


Figure 7. Motion capture system arrangement. The rack was removed for vertical jump trials.



Figure 8. Marker set from anterior view



Figure 9. Marker set from posterior view

| Marker Name | Position | Segment |
|--------------|--|-------------|
| R/LLT | Lateral Foot | Foot |
| R/LMT | Medial Foot | Foot |
| R/LLA | Right /Left Lateral Malleoli | Calibration |
| R/LMA | Right/Left Medial Malleoli | Calibration |
| R/LH | Right/Left Calcaneus | Foot |
| RSH1,2,3 | Right Lateral Shank | Right Shank |
| LSH1,2,3 | Left Lateral Shank | Left Shank |
| R/LLK | Right/Left Lateral Tibiofemoral Joint | Calibration |
| R/LMK | Right/Left Medial Tibiofemoral Joint | Calibration |
| RTH1,2,3,4 | Right Lateral Thigh | Right Thigh |
| LTH1,2,3,4 | Left Lateral Thigh | Left Thigh |
| R/LHIP | Right/Left Greater Trochanter | Pelvis |
| R/LIC | Right/Left Iliac Crest | Pelvis |
| RASIS 1,2 | Right ASIS | Pelvis |
| LASIS 1,2 | Left ASIS | Pelvis |
| R/LPSI | Right/Left Posterior Superior Iliac Spin | e Pelvis |
| SAC | Sacrum | Pelvis |
| R/LAC | Right/Left Acromion Process | Trunk |
| C7 | 7th Cervical Vertebra | Trunk |
| Head 1,2,3,4 | Head | Head |

Table 1. Retroreflective marker locations.

Kinetics

Kinetic data were obtained using two AMTI OR6-1000 force platforms (Advanced Mechanical Technology, Inc., Watertown, Massachusetts) with two MiniAmp MSA-6 amplifiers (Advanced Mechanical Technology, Inc., Watertown, Massachusetts). Each force plate was constructed with four triaxial force transducers and was embedded centrally within the floor of the capture volume. Force was converted from analog to digital data and sampled at 1000 Hz. Force data were obtained to derive relative endpoint force enhancement, relative joint torque enhancement, and vertical impulse.

Surface Electromyography

Lower extremity muscle activation was collected bilaterally at the tibialis anterior, gastrocnemius, vastus lateralis, and gluteus maximus using 8 pairs of bipolar Ag-AgCl surface electrodes (Red Dot, 3M, St. Paul, MN, USA). Leads in connection with a Noraxon® Telemyo 2400T-V2 wireless transmitter (Noraxon® U.S.A. Inc., Scottsdale, AZ, USA) relayed data to a Noraxon® Telemyo 2400R-World Wide Telemetry receiver (Noraxon® U.S.A. Inc., Scottsdale, AZ, USA). Muscle activation was lowpass filtered at the hardware (cutoff frequency 500Hz) and sampled at 1000 Hz according to standard practice (Merletti & Torino, 1999). Sampled EMG was post processed in MATLAB® (MATLAB R2012a, The Mathworks, Inc., Natick, MA). Each signal was digitally band-pass filtered with a second order, dual-pass, Butterworth filter with cutoff frequencies of 20 and 400Hz (DeLuca, Gilmore, Kuznetsov, & Roy, 2010; Merletti & Torino, 1999).

Design and Procedures

All testing occurred in three scheduled meetings with the following purposes: 1) familiarization, 2) force-leg extension protocol, and 3) vertical jump protocol without arm swing. The order of the second and third protocols was randomized. The familiarization protocol involved completion of the health screening questionnaire (Appendix A) and signing of the Institutional Review Board approved Informed Consent document (Appendix B). In addition participants received instruction on how to complete the isometric, maximum effort leg extensions and the various conditions of vertical jump. Lastly, participants were given the opportunity to practice and demonstrate their ability to perform each task accurately and successfully.

At the second meeting the force-leg extension protocol was performed. Once the participant arrived, compression clothing was donned to minimize motion of retroreflective markers. Skin over the muscles of interest (gluteus maximus, vastus lateralis, gastrocnemius, and tibialis anterior) was prepared for electromyography, and electrodes were placed according to the methods outlined by Basmajian and DeLuca (1985). Maximal voluntary isometric contractions (MVIC) of each muscle were performed according to the methods outlined by Kendall, McCreary, Provance, Rodgers, and Romani (2005). To take the MVIC of the gluteus maximus the participant lay prone on a mat with the knee flexed 90° or more and extended the hip against resistance at the distal end of the thigh. For the vastus lateralis participants sat on a table with their knee over the side and knee extended. The participant contracted the quadriceps maximally against resistance just above the ankle. To test the tibialis anterior participants lay supine on a mat with the leg extended and performed maximal effort dorsiflexion against resistance. The MVIC for the soleus was performed by having the participant lay supine with the leg extended and plantarflex with maximal effort against resistance. Forty-six retroreflective markers were placed at the anatomical locations specified in Table 1 (Figure 8, Figure 9). Next, a static calibration video was taken with the participant standing in the anatomically neutral position on the two force platforms located centrally on the floor of the capture volume and within the confines of a modified power rack. After the static capture the participant performed a series of upright, maximum-effort, isometric leg extensions underneath a bar locked in place. The bar was moved to various heights which altered the height of the hip with respect to the ground ("leg length") and the configuration of the segments such that knee angles measured ranged from 80° to 160°. Participants were asked to achieve maximum force as rapidly as possible and to hold with maximum effort until force began to decline from peak. A minimum of 3 minutes rest was

provided between each isometric leg extension (Hahn, 2008). Once this was done a third meeting was scheduled no sooner than 24 hours after the force-leg extension protocol for the completion of the vertical jump protocol.

Upon the participant's arrival to the third meeting, preparations were the same as in the force-leg extension protocol. Compression clothing was worn, skin was prepared and electrodes placed at the same locations, retroreflective markers were placed at the same anatomical locations (Table 1), and another static calibration was performed. MVICs were performed for the muscles of interest according to Kendall, McCreary, Provance, Rodgers, and Romani (2005). Participants performed a minimum of 3 maximum vertical jumps utilizing 3 different loading conditions (SD, CM, SJ). Arm swing was not allowed in any of the jumps. The order of the loading conditions was randomized. The SD required participants to lift their feet from the ground without vertical rise in the center of mass, and fall into a countermotion. The second condition was the CM in which the center of mass lowers prior to the push phase with the feet in contact with the ground. Additionally a SJ condition was performed with the knees pre-flexed to a self-selected depth. The participants remained in the pre-flexed position for two seconds prior to jumping.

Experimental Design

The trial resulting in the best performance (defined by highest vertical take-off velocity) of each condition was kept for statistical analysis. All statistical analyses were performed using SPSS software (version 20.0; SPSS Inc., Chicago, IL, USA), and an alpha level set *a priori* at $p \le 0.05$, was utilized.

To evaluate the statistical differences between loading conditions on kinematics, kinetics, and EMG, a two-way repeated measures ANOVA was utilized. SD, CM, and SJ served as the three levels of the independent variable. The dependent variables were peak concentric EMG, EMD, force enhancement, torque enhancement, impulse, rate of force development, and jump height. Pairwise comparisons were used to determine at which levels the differences occurred.

Data Analysis

Preamplified and bandpass filtered EMG signals were postprocessed in two ways. First, the signal was filtered an additional time with a moving root mean square algorithm (window length of 75 samples). Peak amplitude during the concentric phase was located from the root mean squared signal and normalized to the maximal voluntary isometric contraction (MVIC) for the respective muscles tested.

Finally, for the measurement of electromechanical delay it was necessary to locate the appropriate rise in the EMG signal. For this purpose the methods of Santello & McDonagh (1998) were used (Figure 10). To determine onset the original signal was rectified and integrated using the following equation:

$$I|x(t)| = \int_{t}^{t+T} |x(t)|dt$$

The integrated signal was normalized to the total integrated value and plotted against normalized time (curved solid line in Figure 7). As a result the average slope of the integrated signal was 1. Next a straight line with a slope of one was constructed (dashed line in Figure 10). The difference between each value in the two lines was taken, and the frame of the maximum difference (vertical solid line identified as "d") was taken to be equivalent to the frame of EMG onset. This method was developed to determine the timing of EMG onset in landing; as such it is

a relevant technique for finding onset of muscle activation in an SSC (Santello & McDonagh, 1998). Additionally, it was valuable as EMG onset was determined from non-resting EMG levels (Allison, 2003). Once the onset of muscle activation was identified, the interval between onset and a reversal in sign of angular acceleration represented the EMD.

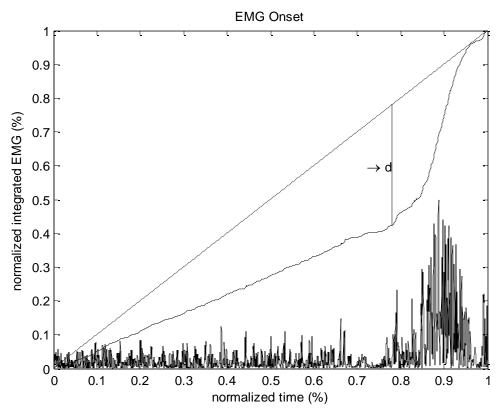


Figure 10. Graphical representation of the determination of EMG onset using the Santello and McDonagh (1998) method.

Force enhancement and torque enhancement were derived from comparing kinetic and kinematic data from jump trials to the force-leg extension and torque-angle curves established during the isometric leg extensions. Once force fell from its peak, leg extension trials were terminated. Force during the plateau was averaged, and this was paired with the height of the greater trochanter from the force platform, which represents "leg length". In this way a force-leg

extension curve was derived, and isometric references were constructed. Force and "leg length" were gathered during the transition from eccentric to concentric phases of the vertical jumps. It was expected that hip height in the isometric leg extensions would not match hip height during the transition phase of the jumps. However, force-leg length was shown to be linear by Hahn (2008), although the setup was horizontal. Furthermore, pilot data suggested that force-leg extension curves are linear in an upright setup also. Because of the linearity, force-leg extension values were interpolated to allow a comparison. Similarly, maximum torque plateau in the leg extensions was averaged and paired with the angle of the respective joint. Once the force or torque during the transition phase of the vertical jumps was extracted, the difference was taken between this value and the force or torque produced during the corresponding isometric reference position. The difference was normalized to the force of the isometric reference value to obtain relative force or torque enhancement.

Performance variables such as impulse, rate of force development, and jump height were also determined. Impulse is the integral of force and time. Output from the force platforms is numerical data. Therefore, impulse was approximated using the midpoint rule. Rate of force development was derived as the difference between maximum vertical force and minimum force divided by the time interval between the two force data points. Therefore, rate of force development was the average rate of change in force from minimum force to maximum force. Jump height was defined by the difference in maximum height achieved of the greater trochanter marker and its height during the static calibration trial.

CHAPTER IV

RESULTS

Methods set forth in Chapter III were designed to obtain kinematic, kinetic, and myoelectric data in the vertical jump for the purpose of deriving and evaluating endpoint force enhancement, torque enhancement, EMD, and performance variables (i.e. jump height, take-off velocity, rate of force development, and impulse) resulting from a novel countermovement (SD), a traditional countermovement (CM), and a no-countermovement (SJ) vertical jump. However, force enhancement, torque enhancement, and EMD values were found to be non-normally distributed. Hence, non-parametric statistical analyses were applied to these variables. The current chapter is designated for reporting results and is outlined as follows: 1] Participant Demographics, 2] Force and Torque Enhancement, 3] Electromechanical Delay, 4] Electromyography, and 5] Performance.

Participant Demographics

Forty-seven students without injury who participated in resistance training 3 times per week volunteered for the study. Of the volunteers 25 were female and 22 were male. However, due to reasons unrelated to the study, only 20 females and 17 males completed the protocol. Of the 20 females completing the study, 3 were not included in the data analysis due to data loss during collection. Hence, a pool of 17 females and 17 males was kept for analysis.

| Table 2. Participant Demographics | | | | | | | | | | | |
|--|----|----------|-----------|-----------|------------------|--|--|--|--|--|--|
| Sex N Age (years) Height (m) Mass (kg) Leg Dominance | | | | | | | | | | | |
| Female | 17 | 23.5±3.5 | 1.68±0.06 | 66.9±8.5 | 17 right | | | | | | |
| Male | 17 | 23.9±1.8 | 1.79±0.08 | 87.3±10.6 | 16 right; 1 left | | | | | | |

Force and Torque Enhancement

Force Enhancement

To determine if force enhancement occurs in the stretch-shortening cycle (SSC) and whether the amount of force enhancement can be altered through different movement histories, vertical ground reaction force during the transition phase of the traditional countermovement, novel countermovement, and at the initiation of the no-countermovement conditions was compared to force during the isometric squat. Figure 11 illustrates the change in ground reaction force with respect to change in trochanter height. The data illustrated in Figure 11 were used to yield an estimate of ground reaction force for a measured trochanter height during an isometric squat. At the end of the section Table 3 summarizes results on force enhancement.

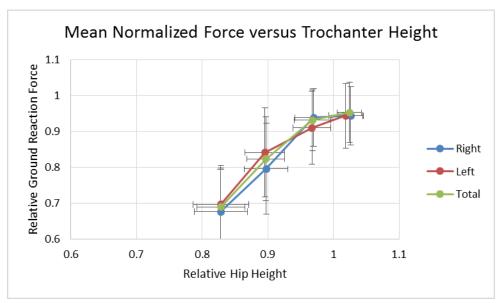


Figure 11. Illustrates the relationship between ground reaction force and trochanter height in an isometric squat. Total is combination of both right and left legs.

Force Enhancement results tended to violate the assumption of normality. Hence, nonparametric analyses were used to determine if movement history affects force enhancement. In this study, based on Friedman's test with follow-up Wilcoxon signed Rank tests and Bonferroni corrections applied, there were statistically significant differences between the SSC conditions for total force enhancement ($\chi^2(2) = 24.182, p < 0.001$). Although the means are statistically different and indicate that force enhancement occurred, not all participants exhibited force enhancement. Several exhibited force depression. In the CM jump 76% of the participants (63% of males and 88% of females) exhibited larger magnitude ground reaction force during transition than was estimated from the curve in Figure 11. Whereas in the SD and SJ condition 73% (50% males, 94% females) and 38% (31% males, 41% females) showed force enhancement, respectively. Force enhancement during transition was not-significantly greater in the SD compared to the CM (Z = -2.153, p = 0.031) but was significantly greater in the SD as well as during the CM compared to SJ (Z = -3.976, p < 0.001, Z = -3.672, p < 0.001, respectively) (Figure 12).

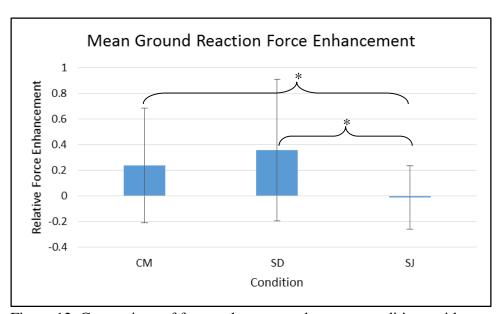


Figure 12. Comparison of force enhancement between conditions without distinction between genders. * denotes significance at the p = 0.017 level.

Force enhancement was also examined in the right leg (Figure 13). For the right leg a Friedman's test with follow-up Wilcoxon signed Rank tests and Bonferroni corrections applied revealed significant differences between vertical jump conditions ($\chi^2(2) = 19.313$, p < 0.001). Differences in condition for the right leg were between the CM and SJ (Z = -3.534, p = 0.001), SD and SJ (Z = -3.441, p < 0.001), and the SD was non-significantly greater than the CM. Additionally not all participants showed force enhancement.

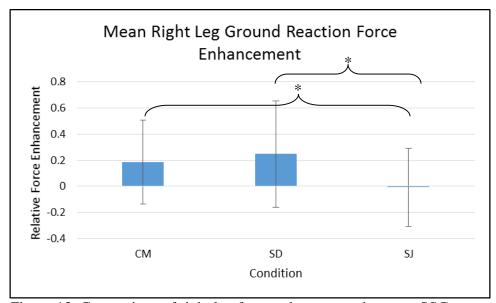


Figure 13. Comparison of right leg force enhancement between SSC conditions. No distinction is made between genders due to a lack of gender effect. * denotes significance at the p = .017 level.

The left leg had similar results (Figure 14). A Friedman's test with follow-up Wilcoxon signed Rank tests and Bonferroni corrections applied demonstrated that there were significant differences ($\chi^2(2) = 18.606$, p < 0.001). Significant differences were between the CM and SJ (Z = -3.046, p = 0.002), SD and SJ (Z = -3.136, p < 0.001), and SD was significantly greater than the CM (Z = -3.725, p = 0.002) (Figure 14).

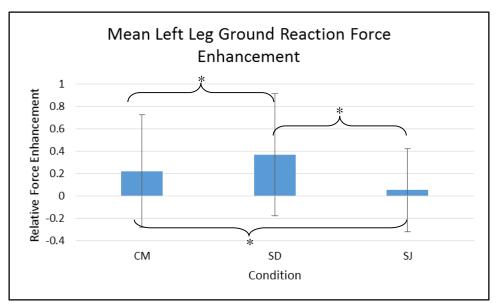


Figure 14. Comparison of left leg force enhancement between SSC conditions. No distinction is made between genders due to a lack of gender effect. * denotes significance at the p = 0.017 level.

Table 3. Summarizes the ground reaction force enhancement results.

| Force Enhancement | | | | | | | | | | | | |
|-------------------|------|-------|------------------|-------------------|-------|------------------|---------|---------------|----------------------|-------------------|--|--|
| | | Both | | | Right | | | | | | | |
| | | CM | SD | SJ | CM | SD | SJ | CM | SD | SJ | | |
| Males | | | | | | | | | | | | |
| | # | 10 | 8 | 5 | 9 | 10 | 8 | 8 | 9 | 4 | | |
| | Mean | 0.03 | 0.16 | 0.06 | 0.09 | 0.15 | -0.05 | -0.06 | 0.07 | -0.09 | | |
| | STD | 0.27 | 0.44 | 0.25 | 0.32 | 0.40 | 0.28 | 0.25 | 0.30 | 0.21 | | |
| Females | | | | | | | | | | | | |
| | # | 15 | 16 | 7 | 14 | 14 | 6 | 15 | 15 | 10 | | |
| | Mean | 0.44 | 0.55 | 0.04 | 0.28 | 0.35 | 0.04 | 0.48 | 0.65 | 0.19 | | |
| | STD | 0.50 | 0.59 | 0.24 | 0.31 | 0.41 | 0.32 | 0.55 | 0.59 | 0.44 | | |
| Total | | | | | | | | | | | | |
| | # | 25 | 24 | 12 | 23 | 24 | 14 | 23 | 24 | 14 | | |
| | Mean | 0.24* | 0.36^{\dagger} | $0.01*^{\dagger}$ | 0.19* | 0.25^{\dagger} | -0.01*† | $0.22^{*\pi}$ | $0.37^{\dagger \pi}$ | $0.05*^{\dagger}$ | | |
| | STD | 0.45 | 0.55 | 0.25 | 0.32 | 0.41 | 0.30 | 0.50 | 0.55 | 0.37 | | |

Table 3. # denotes the number of participants showing force enhancement. The mean is the average of all participants including those with force depression. STD denotes the standard deviation. *, π and † denote significance at the p = 0.017 level.

Torque enhancement was measured at the ankle, knee, and hip for both legs. Torqueangle curves were constructed for each joint, and an estimated torque was derived from each
curve to compare to the measured torque of the transition phase in each vertical jump condition.
Figures 15, 16, and 17 provide examples of ankle, knee, and hip torque-angle curves,
respectively. At the end of the section, Table 4 summarizes results on torque enhancement.

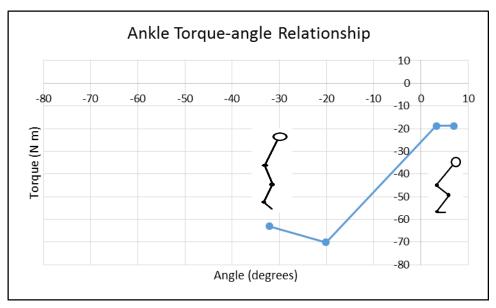


Figure 15. Example of torque-angle relationship for the ankle in an upright isometric squat. Angle is the orientation of the foot in the reference frame of the shank. As angle becomes increasingly negative the participant is increasingly plantarflexed. Negative torque represents plantarflexion torque. Stick figures provide a sense of the orientation of segments at the extreme data points.

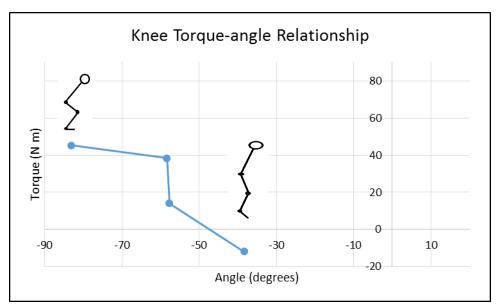


Figure 16. Example of torque-angle relationship for the knee in an upright isometric squat. Angle is the orientation of the shank in the reference frame of the thigh. As angle becomes increasingly negative the participant shows greater knee flexion. Positive torque represents extension torque. Stick figures provide a sense of the orientation of segments at the extreme data points.

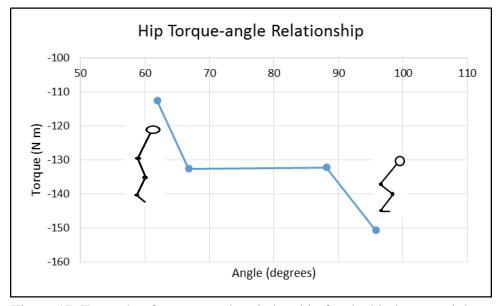


Figure 17. Example of torque-angle relationship for the hip in an upright isometric squat. Angle is the orientation of the thigh in the reference frame of the pelvis. As angle becomes increasingly positive the participant shows greater hip flexion. Negative torque represents extension torque. Stick figures provide a sense of the orientation of segments at the extreme data points.

Not all torque-angle curves provided substantial amounts of data that could be kept for analysis to estimate torque during the transition phase of the SSC conditions. This was especially true of the ankle torque-angle curves. Because many participants pushed through their heels, dorsiflexion torque was measured in the lowest squat position. As a result when torque was estimated for the vertical jump trials, a dorsiflexion torque was predicted, but plantarflexion torque occurred in the jump. Therefore, extrapolations that led to erroneous estimates were discarded. Table 4 at the end of the section summarizes the results from torque enhancement data.

Torque enhancement results tended to violate the assumption of normality. Hence, non-parametric analyses were used to determine if movement history affects torque enhancement. Of the 13 participants kept for comparing torque enhancement at the right ankle 6 were females and the remaining 7 male. 54%, 38% and 46% of the 13 participants demonstrated torque enhancement at the right ankle during the CM, SD, and SJ, respectively. To compare torque enhancement means a Friedman's test was used. The statistical analysis revealed no statistically significant difference between conditions ($\chi^2(2) = 1.077$, p = 0.584).

Results at the left ankle were similar. 11 participants were kept for comparing torque enhancement at the ankles 5 were females and the remaining 6 male. 45%, 36% and 27% of the 11 participants demonstrated torque enhancement at the left ankle during the CM, SD, and SJ, respectively. However, a Friedman's test was used to compare torque enhancement means. The analysis revealed no statistically significant difference between conditions ($\chi^2(2) = 0.182$, p = 0.913).

Torque-angle curves for the knee allowed more participants' data to be kept for analysis.

As a result data from 28 participants were kept for comparisons at the right knee. Of the 28

participants kept for knee torque enhancement comparisons 13 were female and 15 were male. Of the 28 participants kept for comparing torque enhancement at the knee 36%, 32% and 29% demonstrated torque enhancement during the CM, SD, and SJ, respectively. To compare means a Friedman's test was utilized. The analysis revealed no statistically significant difference between conditions ($\chi^2(2) = 3.714$, p = 0.156).

Results at the left knee showed significant differences (Figure 18). 28 participants were kept for comparing torque enhancement at the knee. Thirteen were females and the remaining 15 male. 29%, 42% and 29% of the 28 participants demonstrated torque enhancement at the left knee during the CM, SD, and SJ, respectively. However, to compare means a Friedman's test was used. The analysis revealed no statistically significant difference between conditions ($\chi^2(2)$) = 7.786, p = 0.020). Wilcoxon Signed Rank tests with Bonferroni correction applied were used to determine which conditions were significantly different. Differences were between CM and SD (Z = -2.778, p = 0.005).

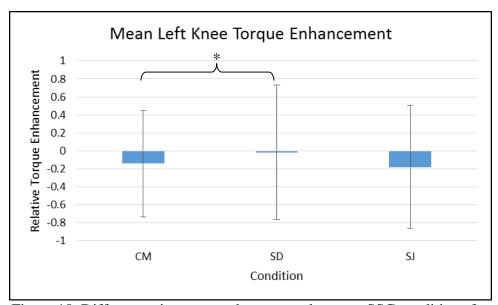


Figure 18. Differences in torque enhancement between SSC conditions for the left knee. * denotes significant differences at the p = 0.017 level.

Similar to the knee, the torque estimation allowed more data to be kept at the hip than at the ankle. Hence, data from 30 participants were kept. Of the 30 participants kept for hip torque enhancement comparisons at the right hip 15 were female and 15 were male, but at the left hip 16 were female and 14 were male.

Unlike data from from the ankles, knees, and left hip, data from the right hip was normally distributed. Hence, a repeated measures ANOVA was to compare vertical jump conditions. Of the 30 participants kept for comparing torque enhancement at the right hip 3%, 10% and 0% demonstrated torque enhancement during the CM, SD, and SJ, respectively. Mauchly's test of sphericity was significant. Therefore, the Greenhouse-Geisser adjustment to the degrees of freedom was used. A repeated measures ANOVA revealed significant differences between conditions ($F(1.663, 46.565) = 28.196, p < 0.001, \eta^2 = 0.502, Power = 1.000$). No significant interaction between sex and condition was found. Pairwise comparisons revealed significant differences between CM and SJ (p < 0.001) and SD and SJ (p < 0.001). However, the CM and SD were not significantly different, although torque enhancement in SD was on average greater than CM (Figure 19).

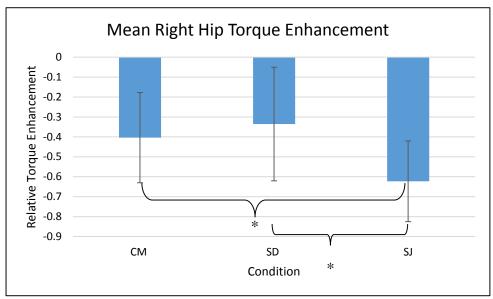


Figure 19. Differences in torque enhancement between SSC conditions for the right hip. * denotes significant differences at the p = 0.017 level.

Results at the left hip were significant (Figure 20). 7%, 7% and 0% of the 30 participants demonstrated torque enhancement at the left hip during the CM, SD, and SJ, respectively. A Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections applied revealed statistically significant difference between conditions ($\chi^2(2) = 36.867 \ p < 0.001$). Wilcoxon Signed Rank tests with Bonferroni corrections showed that significant differences were between CM and SJ (Z = -4.741, p < 0.001) and SD and SJ (Z = -4.288, p < 0.001).

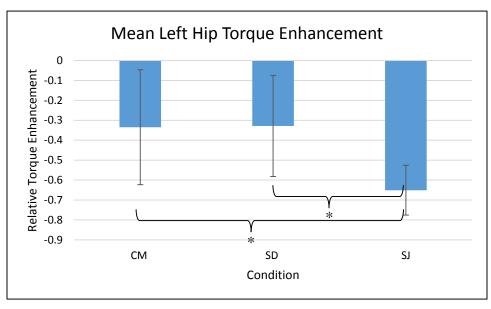


Figure 20. Differences in torque enhancement between SSC conditions for the right hip. * denotes significant differences at the p = 0.017 level.

Table 4. Torque enhancement means and standard deviations.

| Males Right # 5 4 4 3 2 3 0 0 Mean 0.65 0.71 0.68 -0.39 -0.45 -0.27 -0.49 -0.5 STD 1.44 1.86 1.85 0.43 0.33 0.77 0.21 0.1 Left # 3 3 2 2 4 2 1 1 Mean 0.10 0.24 -0.32 -0.41 -0.39 -0.43 -0.35 -0.35 -0.35 STD 0.68 1.12 0.51 0.35 0.35 0.54 0.38 0.2 Females Right # 2 1 2 7 7 5 1 3 Mean -0.06 -0.01 0.00 0.09 0.33 0.17 -0.31 -0.1 Left # 2 1 1 6 8 6 1 1 Mean <th>Hip</th> <th></th> <th>nee</th> <th>Kı</th> <th colspan="3">Ankle</th> <th></th> <th></th> <th></th> | Hip | | nee | Kı | Ankle | | | | | | | | | | | | | |
|--|---|---------|-------|-------|-------|-------|-------|------|---------------------------|---------|-------|-------|------|------|------|------|--|--|
| ## 5 4 4 4 3 2 3 0 0 0 Mean 0.65 0.71 0.68 -0.39 -0.45 -0.27 -0.49 -0.5 STD 1.44 1.86 1.85 0.43 0.33 0.77 0.21 0.1 Left | SJ CM SD SJ | SJ | SD | CM | SJ | SD | CM | | | | | | | | | | | |
| Mean 0.65 0.71 0.68 -0.39 -0.45 -0.27 -0.49 -0.5 STD 1.44 1.86 1.85 0.43 0.33 0.77 0.21 0.1 Left | | | | | | | | | Right | Males | | | | | | | | |
| Left | 3 0 0 0 | 3 | 2 | 3 | 4 | 4 | 5 | # | | | | | | | | | | |
| Left # 3 3 2 2 4 2 1 <th <="" colspan="8" td=""><td>-0.27 -0.49 -0.50 -0.69</td><td>5 -0.27</td><td>-0.45</td><td>-0.39</td><td>0.68</td><td>0.71</td><td>0.65</td><td>Mean</td><td></td><td></td></th> | <td>-0.27 -0.49 -0.50 -0.69</td> <td>5 -0.27</td> <td>-0.45</td> <td>-0.39</td> <td>0.68</td> <td>0.71</td> <td>0.65</td> <td>Mean</td> <td></td> <td></td> | | | | | | | | -0.27 -0.49 -0.50 -0.69 | 5 -0.27 | -0.45 | -0.39 | 0.68 | 0.71 | 0.65 | Mean | | |
| # 3 3 2 2 4 2 1 1 1 Mean 0.10 0.24 -0.32 -0.41 -0.39 -0.43 -0.35 -0.3 STD 0.68 1.12 0.51 0.35 0.35 0.54 0.38 0.2 Females Right | 0.77 0.21 0.18 0.17 | 0.77 | 0.33 | 0.43 | 1.85 | 1.86 | 1.44 | STD | | | | | | | | | | |
| Mean STD 0.10 0.24 0.68 -0.32 0.51 -0.41 0.35 0.35 -0.43 0.54 0.38 -0.35 0.35 0.54 -0.35 0.38 0.2 Females Right # 2 1 2 7 7 7 5 1 3 0.00 2 7 7 7 5 1 3 0.00 3 0.17 0.01 0.01 0.00 0.09 0.33 0.17 0.01 0.01 0.00 0.09 0.33 0.17 0.01 0.01 0.00 0.21 0.2 0.2 0.06 0.94 0.00 0.21 0.2 0.2 0.2 0.00 0.21 0.2 0.2 0.2 0.00 0.17 0.42 0.11 0.00 0.11 0.032 0.02 0.00 0.02 0.00 0.00 0.00 < | | | | | | | | | Left | | | | | | | | | |
| Females Right # 2 1 2 7 7 7 5 1 3 Mean -0.06 -0.01 0.00 0.09 0.33 0.17 -0.31 -0.1 STD 0.93 1.10 1.29 0.66 0.94 1.08 0.21 0.2 Left # 2 1 1 6 8 6 1 1 Mean -0.14 -0.20 0.06 0.17 0.42 0.11 -0.32 -0.2 STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | $2 \qquad 1 \qquad 1 \qquad 0$ | 2 | 4 | 2 | 2 | 3 | 3 | # | | | | | | | | | | |
| Females Right # 2 1 2 7 7 5 1 3 Mean -0.06 -0.01 0.00 0.09 0.33 0.17 -0.31 -0.1 STD 0.93 1.10 1.29 0.66 0.94 1.08 0.21 0.2 Left # 2 1 1 6 8 6 1 1 Mean -0.14 -0.20 0.06 0.17 0.42 0.11 -0.32 -0.2 STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | -0.43 -0.35 -0.38 -0.66 | 9 -0.43 | -0.39 | -0.41 | -0.32 | 0.24 | 0.10 | Mean | | | | | | | | | | |
| # 2 1 2 7 7 5 1 3 Mean | 0.54 0.38 0.25 0.14 | 0.54 | 0.35 | 0.35 | 0.51 | 1.12 | 0.68 | STD | | | | | | | | | | |
| Mean -0.06 -0.01 0.00 0.09 0.33 0.17 -0.31 -0.1 STD 0.93 1.10 1.29 0.66 0.94 1.08 0.21 0.2 Left # 2 1 1 1 6 8 6 1 1 1 Mean -0.14 -0.20 0.06 0.17 0.42 0.11 -0.32 -0.2 STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | | | | | | | | | Right | Females | | | | | | | | |
| Left # 2 1 1 6 8 6 1 1 1 Mean -0.14 -0.20 0.06 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 5 1 3 0 | 5 | 7 | 7 | 2 | 1 | 2 | # | | | | | | | | | | |
| Left # 2 1 1 6 8 6 1 1 Mean -0.14 -0.20 0.06 0.17 0.42 0.11 -0.32 -0.2 STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 0.17 -0.31 -0.17 -0.55 | 0.17 | 0.33 | 0.09 | 0.00 | -0.01 | -0.06 | Mean | | | | | | | | | | |
| # 2 1 1 6 8 6 1 1 Mean -0.14 -0.20 0.06 0.17 0.42 0.11 -0.32 -0.2 STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 1.08 0.21 0.28 0.21 | 1.08 | 0.94 | 0.66 | 1.29 | 1.10 | 0.93 | STD | | | | | | | | | | |
| Mean STD -0.14 -0.20 0.65 0.06 0.17 0.42 0.11 -0.32 -0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | | | | | | | | | Left | | | | | | | | | |
| STD 0.65 0.69 1.11 0.66 0.85 0.74 0.19 0.2 Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 6 1 1 0 | 6 | 8 | 6 | 1 | 1 | 2 | # | | | | | | | | | | |
| Total Right # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 0.11 -0.32 -0.28 -0.65 | 0.11 | 0.42 | 0.17 | 0.06 | -0.20 | -0.14 | Mean | | | | | | | | | | |
| # 7 5 6 10 9 8 1 3 Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | 0.74 0.19 0.26 0.12 | 0.74 | 0.85 | 0.66 | 1.11 | 0.69 | 0.65 | STD | | | | | | | | | | |
| Mean 0.32 0.38 0.36 -0.16 -0.09 0.06 -0.40* -0.3 | | | | | | | | | Right | Total | | | | | | | | |
| | 8 1 3 0 | 8 | 9 | 10 | 6 | 5 | 7 | # | | | | | | | | | | |
| STD 1.24 1.54 1.59 0.59 0.78 0.94 0.23 0.2 | 0.06 $-0.40*$ -0.33^{\dagger} $-0.62*^{\dagger}$ | 9 0.06 | -0.09 | -0.16 | 0.36 | 0.38 | 0.32 | Mean | | | | | | | | | | |
| | 0.94 0.23 0.29 0.20 | 0.94 | 0.78 | 0.59 | 1.59 | 1.54 | 1.24 | STD | | | | | | | | | | |
| Left | | | | | | | | | Left | | | | | | | | | |
| # 5 4 3 8 12 8 2 2 | | | | | | | | | | | | | | | | | | |
| | | | | | | | | | | | | | | | | | | |
| STD 0.64 0.93 0.82 0.59 0.75 0.69 0.28 0.2 | 0.69 0.28 0.25 0.13 | 0.69 | 0.75 | 0.59 | 0.82 | 0.93 | 0.64 | STD | | | | | | | | | | |

Table 4. # denotes the number of participants with torque enhancement. The mean includes those with torque depression. STD denotes the standard deviation. Values multiplied by 100 equal the percent torque enhancement. Negatives denote torque depression. * and † denote significance at the p = 0.017 level.

Electromechanical Delay

Electromechanical delay (EMD) was measured for the tibialis anterior, soleus, vastus lateralis, and gluteus maximus of the right and left lower limbs. EMD was taken as the time between a change in velocity of the joint and onset of muscle activity. Therefore a positive EMD indicates that EMG onset occurred prior to the change in velocity of the corresponding joint. Conversely, a negative EMD indicates that EMG onset occurred after the kinematic event. Additionally it should be noted that much of the data was non-normally distributed. Therefore, non-parametric statistics were used to analyze results.

Right and Left Tibialis Anterior

Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections applied showed statistically significant differences in EMD between conditions for the right tibialis anterior ($\chi^2(2) = 18.650$, p < 0.001). Condition effects were between CM and SJ (Z = -3.371, p = 0.001) and SD and SJ (Z = -2.910, p = 0.004) with CM and SD EMD being less than SJ EMD (Figure 21).

Friedman's Test showed statistically significant differences in EMD between conditions for the left tibialis anterior ($\chi^2(2) = 15.063$, p = 0.001). Wilcoxon Signed Rank Tests with Bonferroni corrections applied showed that condition effects were between CM and SD (Z = -2.711, p = 0.007) (Figure 22).

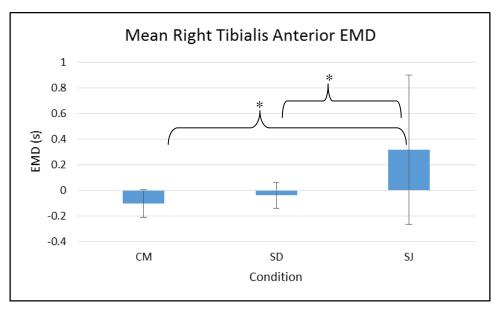


Figure 21. Differences in tibialis anterior EMD for the right leg. * denotes significant differences between conditions at the p = 0.017 level.

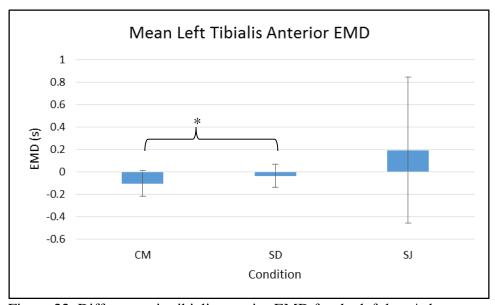


Figure 22. Differences in tibialis anterior EMD for the left leg. * denotes significant differences between conditions at the p=0.017 level.

Right and Left Soleus

A Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections showed statistically significant differences in EMD between conditions for the right soleus ($\chi^2(2) = 24.788$, p < 0.001). Condition effects were between CM and SD (Z = -3.913, p < 0.001) and CM and SJ (Z = -4.655, p < 0.001) (Figure 23).

For the left soleus a Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections showed statistically significant differences between conditions ($\chi^2(2) = 25.647$, p < 0.001). Follow up Wilcoxon Signed Rank tests with Bonferroni corrections were run. Results indicated significant differences were between CM and SD (Z = -4.163, p < 0.001) and CM and SJ (Z = -4.676, p < 0.001) (Figure 24).

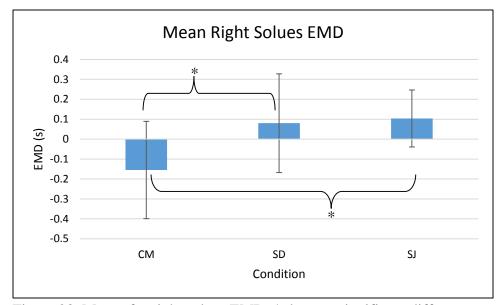


Figure 23. Means for right soleus EMD. * denotes significant difference between conditions at the p = 0.017 level.

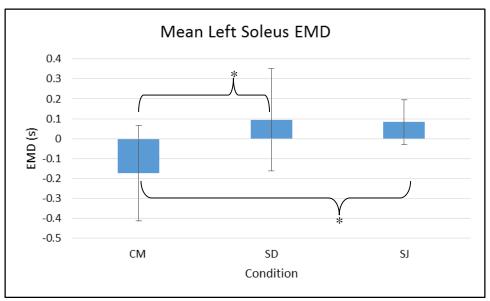


Figure 24. Differences in left soleus EMD between SSC conditions are shown. * denotes significant differences at the p = 0.017 level.

Right and Left Vastus Lateralis

A Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections showed statistically significant differences in EMD between conditions for the right vastus lateralis ($\chi^2(2) = 25.613$, p < 0.001). Condition effects were between CM and SD (Z = -4.419, p < 0.001) and CM and SJ (Z = -4.223, p < 0.001) (Figure 25).

For the left vastus lateralis a Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections indicated significant differences between conditions ($\chi^2(2) = 34.938$, p < 0.001). Similar to the right vastus lateralis, the left vastus lateralis showed differences between CM and SD (Z = -4.862, p < 0.001) and CM and SJ (Z = -4.675, p < 0.001) (Figure 26).

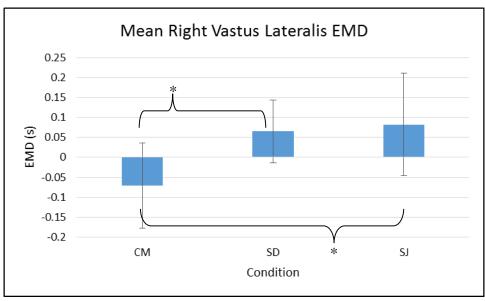


Figure 25. Demonstrates differences in EMD between SSC conditions for the right vastus lateralis. * denotes significance at the p = 0.017 level.

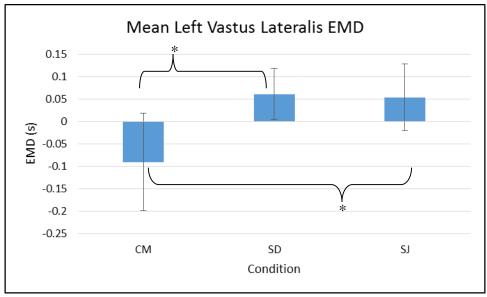


Figure 26. Demonstrates differences in EMD between SSC conditions for the left vastus lateralis. * denotes significance at the p = 0.017 level.

Right and Left Gluteus Maximus

Unlike EMD data at the tibialis anterior, soleus, vastus lateralis, and left gluteus maximus, EMD at the right gluteus maximus was normally distributed. Hence, a repeated measures ANOVA was used to compare conditions. A repeated measures ANOVA showed statistically significant differences in EMD between conditions for the right gluteus maximus $(F(2,62) = 35.647, p < 0.001, \eta^2 = .534, Power = 1.000)$. Condition effects were between CM and SD (p < 0.001) and CM and SJ (p < 0.001) (Figure 27).

Friedman's test with follow-up Wilcoxon Signed Rank tests and Bonferroni corrections showed statistically significant differences in EMD between conditions ($\chi^2(2) = 24.424$, p < 0.001). Similar to the right gluteus maximus, the left gluteus maximus showed differences between CM and SD (Z = -4.154, p < 0.001) and CM and SJ (Z = -4.271, p < 0.001) (Figure 28).

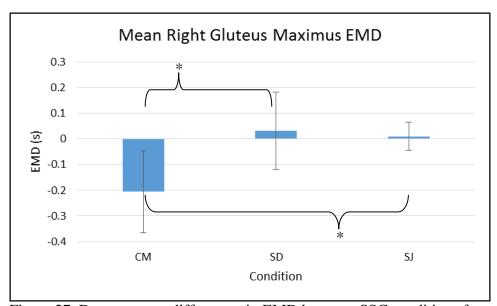


Figure 27. Demonstrates differences in EMD between SSC conditions for the right gluteus maximus. * denotes significance at the p = 0.017 level.

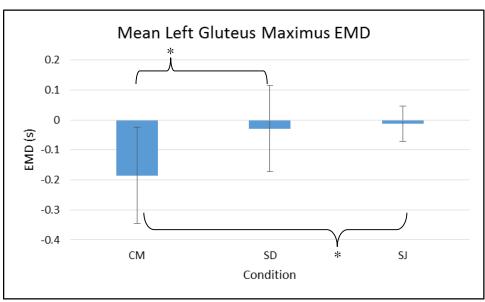


Figure 28. Demonstrates differences in EMD between SSC conditions for the left gluteus maximus. * denotes significance at the p = 0.017 level.

Table 5. Means and standard deviations of electromechanical delay data.

| | | RTA | | | LTA | | | RSOL | | | LSOL | | |
|---------|------|----------|--------------------|--------------------|----------|--------------------|-------------------|----------------------|-------------------|-------------------|----------------------|-------------------|--------------------|
| | | CM | SD | SJ | CM | SD | SJ | CM | SD | SJ | CM | SD | SJ |
| Males | | | | | | | | | | | | | |
| | Mean | -0.125 | -0.067 | 0.444 | -0.109 | -0.019 | -0.095 | -0.180 | -0.039 | 0.075 | -0.178 | -0.046 | 0.064 |
| | STD | 0.128 | 0.095 | 0.643 | 0.135 | 0.080 | 0.556 | 0.280 | 0.242 | 0.106 | 0.245 | 0.228 | 0.074 |
| Females | | | | | | | | | | | | | |
| | Mean | -0.084 | -0.017 | 0.213 | -0.097 | -0.048 | 0.483 | -0.129 | 0.206 | 0.134 | -0.168 | 0.235 | 0.104 |
| | STD | 0.088 | 0.099 | 0.519 | 0.096 | 0.122 | 0.625 | 0.206 | 0.188 | 0.172 | 0.241 | 0.206 | 0.140 |
| Total | | | | | | | | | | | | | |
| | Mean | -0.103* | -0.040^{\dagger} | $0.317^{*\dagger}$ | -0.103** | -0.033^{\dagger} | 0.194^{*} | -0.155* [†] | 0.080^{\dagger} | 0.103^{*} | -0.173* [†] | 0.095^{\dagger} | 0.084^{*} |
| | STD | 0.108 | 0.098 | 0.580 | 0.115 | 0.103 | 0.652 | 0.244 | 0.248 | 0.143 | 0.239 | 0.257 | 0.112 |
| | | | | | | | | | | | | | |
| | | RVL | | | LVL | | | RGM | | | LGM | | |
| | | CM | SD | SJ | CM | SD | SJ | CM | SD | SJ | CM | SD | SJ |
| Males | | | | | | | | | | | | | |
| | Mean | -0.125 | 0.048 | 0.062 | -0.113 | 0.040 | 0.049 | -0.236 | -0.002 | -0.006 | -0.197 | -0.085 | -0.011 |
| | STD | 0.118 | 0.084 | 0.109 | 0.127 | 0.064 | 0.083 | 0.181 | 0.152 | 0.085 | 0.188 | 0.156 | 0.068 |
| Females | | | | | | | | | | | | | |
| | Mean | -0.021 | 0.081 | 0.102 | -0.064 | 0.081 | 0.077 | -0.178 | 0.065 | 0.027 | -0.174 | 0.023 | -0.015 |
| | STD | 0.067 | 0.072 | 0.146 | 0.079 | 0.042 | 0.069 | 0.136 | 0.145 | 0.056 | 0.136 | 0.111 | 0.049 |
| Total | | | | | | | | | | | | | |
| | Mean | -0.071*† | 0.065^{*} | 0.082^{\dagger} | -0.090*† | 0.061^{*} | 0.054^{\dagger} | -0.206*† | 0.032^{*} | 0.010^{\dagger} | -0.185* [†] | -0.029* | -0.013^{\dagger} |
| | STD | 0.107 | 0.079 | 0.129 | 0.109 | 0.057 | 0.074 | 0.159 | 0.150 | 0.054 | 0.161 | 0.143 | 0.058 |

Table 5. RTA is right tibialis anterior. LTA is left tibialis anterior. RSOL is right soleus. LSOL is left soleus. RVL is right vastus lateralis. LVL is left vastus lateralis. RGM is right gluteus maximus. LGM is left gluteus maximus. *, π , and † denote significance at the p = 0.017 level.

Electromyography

EMG amplitude was measured during the vertical jump conditions. EMG amplitude was normalized to the maximal voluntary isometric contractions (MVICs). The maximum normalized EMG amplitude in the concentric phase of each SSC condition was kept for comparison.

Right and Left Tibialis Anterior

To compare EMG amplitude for the right tibialis anterior a two-way repeated measures ANOVA was utilized. Results from the statistical analysis indicated that there were significant differences between the SSC conditions (F(2,64) = 4.176, p = 0.020, $\eta^2 = 0.115$, Power = 0.716). However, there was not a significant sex interaction (F(2,64) = 0.579, p = 0.573, $\eta^2 = 0.018$, Power = 0.142). Maximum concentric EMG amplitude was significantly less in the CM compared to SD (p = 0.025) conditions.

In contrast to the right tibialis anterior, the left tibialis anterior showed no significant differences between conditions ($F(2,64)=0.962,\,p=0.388,\,\eta^2=0.029,\,Power=0.210$) and no sex interactions ($F(2,64)=0.548,\,p=0.581,\,\eta^2=0.017,\,Power=0.137$). Right and Left Soleus

A two-way repeated measures ANOVA was utilized for comparing right soleus normalized EMG amplitude across conditions and sex. Results from the statistical analysis indicated that there were no significant differences between the SSC conditions (F(1.292,41.334)) = 0.483, p = 0.538, $\eta^2 = 0.015$, Power = 0.111). Additionally, there was no significant sex interaction (F(1.292,41.334) = 2.124, p = 0.148, $\eta^2 = 0.062$, Power = 0.333). Similar to the right soleus, the left soleus showed no significant differences between conditions (F(1.494,47.797) = 1.666, p = 0.204, $\eta^2 = 0.049$, Power = 0.291) and no sex interactions (F(1.494,47.797) = 0.758, p = 0.438, $\eta^2 = 0.023$, Power = 0.155).

Right and Left Vastus Lateralis

A two-way repeated measures ANOVA was utilized for comparing right vastus lateralis normalized EMG amplitude across conditions and sex. Results from the statistical analysis indicated that there were no significant differences between the SSC conditions (F(2,64) = 0.682, p = 0.509, $\eta^2 = 0.021$, Power = 0.160). Additionally, there was no significant sex interaction (F(2,64) = 0.982, p = 0.380, $\eta^2 = 0.030$, Power = 0.214). For the left vastus lateralis the sphericity assumption was violated. The Greenhouse-Geisser adjustment was utilized. Nonetheless, the analysis showed no significant differences between conditions (F(1.057,33.820) = 0.454, p = 0.516, $\eta^2 = 0.014$, Power = 0.102) and no sex interactions (F(1.057,33.820) = 0.949, p = 0.342, $\eta^2 = 0.029$, Power = 0.160). No pairwise comparisons were necessary. *Right and Left Gluteus Maximus*

A two-way repeated measures ANOVA was utilized for comparing right gluteus maximus normalized EMG amplitude across conditions and sex. Right gluteus maximus EMG data violated the assumption of sphericity, necessitating the use of the Greenhouse-Geisser adjustment. Results from the statistical analysis indicated that there were no significant differences between the SSC conditions (F(1.378,44.102) = 0.496, p = 0.544, $\eta^2 = 0.015$, *Power* = 0.114). Additionally, there was no significant sex interaction (F(1.378,44.102) = 3.297, p = 0.063, $\eta^2 = 0.093$, *Power* = 0.498).

Results for the right and left gluteus maximi were similar. The sphericity assumption was violated, and the Greenhouse-Geisser adjustment was utilized. Nonetheless, the analysis showed no significant differences between conditions (F(1.514,48.432) = 1.517, p = 0.230, $\eta^2 = 0.045$, Power = 0.270) and no sex interactions (F(1.514,48.432) = 1.145, p = 0.314, $\eta^2 = 0.035$, Power = 0.214).

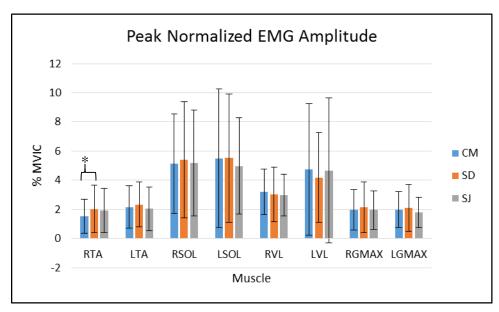


Figure 29. RTA and LTA refer to the right and left tibialis anterior. RSOL and LSOL refer to the right and left soleus. RVL and LVL refer to the right and left vastus lateralis. RGMAX and LGMAX refer to the right and left gluteus maximus. * denotes a significant difference between conditions at the p = 0.05 level.

Performance

A number of performance variables were measured. This was to determine the consequence of altered EMD and force enhancement.

Jump Height

A two-way repeated measures ANOVA was used to analyze differences in jump height. The results of the analysis indicated significant differences among conditions (F(2,64) = 33.738, p < 0.001, $\eta^2 = 0.513$, Power = 1.000) and both sexes had the same response to the imposed conditions (F(2,64) = 2.512, p = 0.089, $\eta^2 = 0.073$, Power = 0.486). Pairwise comparisons demonstrated that jump height in the CM was significantly greater than the SD (p = 0.014) and the SJ (p < 0.001). Additionally, height in the SD was significantly greater than the SJ (p < 0.001).

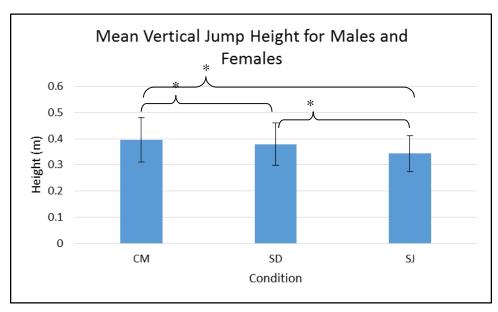


Figure 30. Mean vertical jump height for each SSC condition. * denotes significance at the p = 0.05 level.

Take-off Velocity

A two-way repeated measures ANOVA was used to analyze differences in take-off velocity. The results of the analysis indicated significant differences among conditions (F(2,64)) = 15.792, p < 0.001, $\eta^2 = 0.330$, Power = 0.999) and both sexes had the same response to the imposed conditions (F(2,64) = 0.248, p = 0.781, $\eta^2 = 0.008$, Power = 0.087). Pairwise comparisons demonstrated that take-off velocity in the CM was significantly greater than the SD (p = 0.012) and the SJ (p < 0.001). Additionally, take-off velocity in the SD was significantly greater than the SJ (p = 0.019).

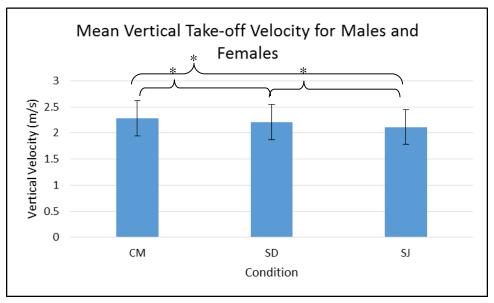


Figure 31. Mean vertical jump take-off velocity for each SSC condition. * denotes significance at the p = 0.05 level.

Impulse

A two-way repeated measures ANOVA was used to analyze differences in impulse. The results of the analysis indicated significant differences among conditions (F(2,64) = 5.601, p = 0.006, $\eta^2 = 0.149$, Power = 0.842), and both sexes had the same response to the imposed conditions (F(2,64) = 1.099, p = 0.339, $\eta^2 = 0.033$, Power = 0.235). Pairwise comparisons demonstrated that impulse in the CM was significantly greater than the SD (p = 0.018) and the SJ (p = 0.013). There was no significant difference between SD and SJ in impulse.

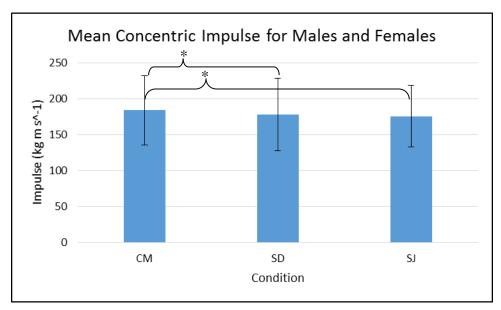


Figure 32. Mean vertical jump impulse for each SSC condition. * denotes significance at the p = 0.05 level.

Rate of Force Development

A two-way repeated measures ANOVA was used to analyze differences in RFD. RFD results violated the sphericity assumption requiring the Greenhouse-Geisser adjustment to the degrees of freedom. The results of the analysis indicated significant differences among conditions (F(1.297,41.491) = 15.104, p < 0.001, $\eta^2 = 0.321$, Power = 0.987) and both sexes had the same response to the imposed conditions (F(1.297,41.491) = 1.875, p = 0.177, $\eta^2 = 0.055$, Power = 0.300). Pairwise comparisons demonstrated that RFD in the SD was significantly greater than the CM (p < 0.001) and the SJ (p = 0.025). Additionally, RFD in the SJ was significantly greater than the CM (p = 0.009).

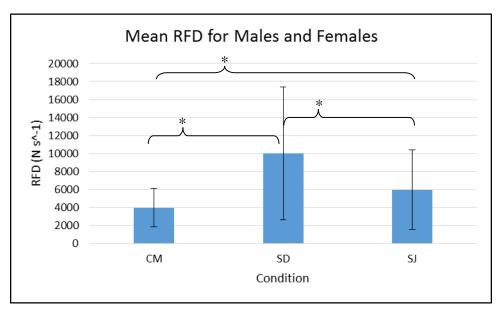


Figure 33. Mean vertical rate of force development (RFD) for each SSC condition. * denotes significance at the p = 0.05 level.

CHAPTER V

DISCUSSION

The purpose of this study was to determine the relevance of force enhancement to human movement, specifically with regard to the stretch-shortening cycle (SSC). This was done by altering the movement history of the SSC during the vertical jump. In other words the countermotion was varied among three conditions: 1) traditional countermovement (CM), 2) self-induced drop (SD), and 3) no-countermovement or squat jump (SJ). For each of the three movement histories, this study investigated: 1) Endpoint force enhancement during the transition phase; 2) Torque enhancement at the ankle, knee, and hip; 3) Electromechanical delay in the tibialis anterior, soleus, vastus lateralis, and gluteus maximus; 4) Normalized peak EMG during the concentric phase; and 5) Performance variables (i.e. jump height, take-off velocity, impulse, and rate of force development (RFD)). The discussion is divided into six sections. The first section discusses force and torque enhancement during the transition phase of the SSC conditions. The second section discusses the effect of SSC conditions on EMD. The third section discusses the influence of SSC on normalized peak EMG. The fourth section discusses the influence of various movement histories on performance. The fifth section makes an attempt to relate the outcomes to one another. Finally, the sixth section sets forth conclusions and proposes future inquiry.

Force and Torque Enhancement

This study aimed to examine the effect of movement history on force enhancement. Force enhancement is a phenomenon that occurs in muscle as a result of active stretch (Herzog, 2004). If a muscle is actively stretched to an isometric reference length, it has the capacity to produce more force than its length-tension relation predicts. The reason is that length-tension curves are constructed from completely isometric contractions. No previous contraction is involved, and mechanisms for force enhancement are activated during active stretch (Herzog, 2004). Here no direct measurement of the muscle could be made. However, countermotions certainly lead to length changes in the muscle-tendon unit and could lead to length changes in the muscle fibers, which affects muscle force (Ishikawa, Komi, Finni, & Kuitunen, 2006). Hence, endpoint force and torque were measured in an attempt to infer force enhancement due to various movement histories. This is not a new idea. Muscle force enhancement has been inferred in single joint (Oskouei, & Herzog, 2009) and multi-joint studies (Seiberl, Paternoster, Achatz, Schwirtz, & Hahn, 2013). This study expands upon such research by measuring force and torque enhancement in an everyday, sport applicable, multi-joint task. In order to determine force and torque enhancement ground reaction force (GRF) to greater trochanter height as well all torque-angle relationships were established for each participant. Based on these relationships (Figure 11, 42, 43 and 44) force could be estimated from trochanter height or joint angle in a vertical jump trial using the equation for a line:

$$v = mx + b$$

In the equation above y is the estimated force or torque; m is the slope obtained from each participant's force-trochanter height relationship or torque-angle relationship; and x is the trochanter height or angle obtained during the transition phase of the vertical jump trial. The

transition phase is a necessary point of comparison as this phase approximates isometric conditions (Kieran & Wallace, 2007) and provides for the comparison between the isometric strength curve to the isometric-like condition of the transition phase of the vertical jump (Figure 33). The idea was that a countermotion prior to a vertical jump necessitates a length change in the muscle-tendon unit. However, the muscle-tendon interaction during the countermotion is unclear. That is whether the muscle fibers are producing tension eccentrically or concentrically is not known. In the event that muscle fibers act eccentrically during the countermotion, a state of force enhancement should be achieved during the transition phase. If force enhancement occurs at the muscle level, it would be indirectly revealed through external force and joint moments. External force and joint moments above the values indicated by a multi-joint, isometric leg extension strength curve would suggest force and torque enhancement.

Phases of the Vertical Jump

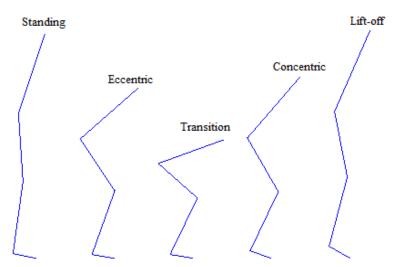


Figure 34. Phases of the vertical jump. Muscle stretch necessary for force enhancement may occur in the eccentric phase. Force and torque enhancement are measured in the transition phase, since there is a brief period of isometric muscle activity.

In this study the hypothesis (H01) was that endpoint force enhancement would occur in SD but not in CM and SJ. External, endpoint force enhancement was demonstrated for both legs combined, the right leg, and the left leg in all three conditions (Figures 12, 13, 14). Significant differences were between CM versus SJ and SD versus SJ for both legs combined and the right leg. However, in the left leg significant differences were between CM and SJ, SD and SJ, and CM and SD. The general pattern shows force enhancement in the SSC conditions to be greater compared to movements without a countermotion. This demonstrates that certain movement histories, those that elicit muscle-tendon unit stretch (i.e. SSC), are a prerequisite for endpoint force production that is above what can be expected based on extrapolation of a multi-joint isometric strength curve. Furthermore, because the SD condition yields greater average endpoint force enhancement compared to CM, it is evident that popular movement histories may be manipulated to increase force enhancement. Additionally it should be noted that participants in this study were not trained to perform the SD. Hence, movement history manipulations can be utilized to produce force enhancement acutely or without training. Further, these findings provide support for additional study into the SD movement history. Perhaps, training in the SD preparatory movement may yield enhanced "explosiveness". In summary, H01 is rejected as endpoint force enhancement was found to occur in all three conditions, CM, SJ and SD.

Similar to force enhancement, it was hypothesized (H02) that torque enhancement, like endpoint force enhancement, was expected to be seen in the SD but not in the CM and SJ. However, torque results did not follow force enhancement results. At the ankle all measures of torque enhancement were on average positive. This means that on average torque enhancement occurred. At the right ankle the largest torque enhancement occurred in the SD condition (SD > SJ > CM). In contrast at the left ankle the greatest torque enhancement occurred in the CM (CM

> SJ > SD). Additionally torque enhancement at the left ankle during the CM was very high (Mean = 14.68, std. = 48.92). This indicates that at the left ankle during the CM participants produced 1,468 times the torque estimated from the strength curve. Attention should also be drawn to the large standard deviation. As a result torque enhancement measures should be interpreted with caution, particularly at the ankle. One reason is that the participant pool is small. Data from only thirteen participants was kept due to a failure in the torque estimation. Torque estimation failures occurred primarily due to the fact that participants pushed through the heel in the squat protocol, but participants pushed through the forefoot in the jump protocol. As a result not all torque-angle curves turned out like the ankle torque-angle curve shown in Figure 15. When participants pushed through the heel, plantarflexion torque became very small, and occasionally a dorsiflexion torque was present. Dorsiflexion torque is a positive torque. Hence if plantarflexion torque was present in the transition phase of the vertical jump at the ankle a negative torque was divided by a positive torque resulting in a nonsense value. Therefore, it is difficult to evaluate torque enhancement in everyday movements without taking into account other parameters that contribute to torque. Since pushing through the heel in the strength curve results in a small moment arm at the ankle, moment arms should be added into the torque estimation in addition to joint angle. While overall the ankle showed torque enhancement, the data warrants a cautious interpretation. Furthermore, evaluating torque enhancement in everyday movements may require more mathematical approaches (i.e. modeling) as applied research does not always allow for adequate control over the studied movement.

The knee provided a more data to analyze torque enhancement compared to the ankle. At the right knee, an average torque enhancement occurred in the SD and SJ conditions but not in the CM condition. At the left knee, torque depression occurred in all conditions, but the SD

condition demonstrated the least torque depression compared to SJ and CM, respectively. Furthermore at the left knee the torque depression was significantly less in SD compared to CM. Hence, it appears that due to higher magnitude GRF, the SD condition resulted in near or greater than the torque estimated from the isometric squat. Similarly, at the hip for both the right and left leg no torque enhancement occurred. Torque depression in CM and SD was significantly less compared to SJ. However, the least torque depression occurred in the SD compared to the CM and SJ, respectively. The SD condition results in larger torques at the hip during the hip transition phase. It is clear that the SD condition involves greater torque production at a given joint. However, the active stretch in the SD and CM does not always result in torque enhancement. Despite the occurrence of torque depression, it is evident from these results that movement history in the vertical jump has a greater influence on the hip compared to other joints, since the hip is where significance was found. A possible anatomical explanation is provided by the architecture of the gluteus maximus (Alexander & Ker, 1990). The gluteus maximus is characterized by a relatively short tendon and relatively long fascicles, which makes the muscle useful for performing work. The relatively short tendon necessitates that the muscletendon unit length change occur in the muscle fibers, which increases the chance of force enhancement. However, given that most participants showed torque depression, a more likely explanation of this is the timing of the transition phases for each joint and the timing of the maximum force immediately prior to propulsion.

A cluster analysis was performed on the top five female and top five male jumpers to determine if the joint transition phases were simultaneous. Also cluster analysis was used to determine which of the joints had transition phases that were simultaneous with maximum force prior to propulsion. The cluster analysis was similar to the methods of Krouchev, Kalaska and

Drew (2006). Results of the analysis revealed trends in the timing of the joint transitions relative to one another and relative to maximum force prior to propulsion (Figure 35, 36, 37). Cluster analysis showed that for both legs all joint transitions were on average simultaneous in the SJ condition (Figure 35). Similarly, in the SD condition all joint transitions were simultaneous, but the left hip on average was less simultaneous with the ankle compared to the hip with the knee (Figure 36). In the CM conditions for both legs the ankle and knee and knee and hip were simultaneous. However, the hips were less simultaneous with the ankle compared to the knee with the ankle (Figure 37).

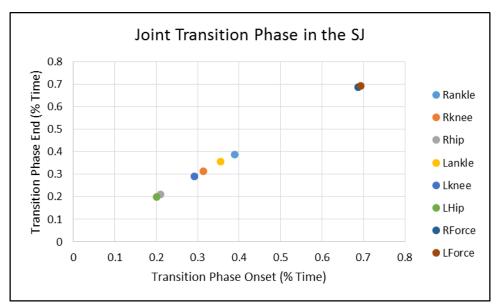


Figure 35. Shows the average relative timing of the joint transition phases and maximum force prior to propulsion. The abscissa represents the beginning of an event, while the ordinate represents the ending of an event. "R" and "L" are used as prefixes to denote right versus left.



Figure 36. Shows the average relative timing of the joint transition phases and maximum force prior to propulsion. The abscissa represents the beginning of an event, while the ordinate represents the ending of an event. "R" and "L" are used as prefixes to denote right versus left.

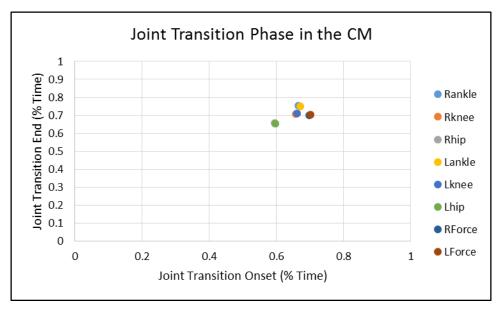


Figure 37. Shows the average relative timing of the joint transition phases and maximum force prior to propulsion. The abscissa represents the beginning of an event, while the ordinate represents the ending of an event. "R" and "L" are used as prefixes to denote right versus left.

Additionally, cluster analysis showed that in the SJ condition none of the joint transitions were near the maximum force. Because force approximates body weight in SJ condition just prior to motion and there is not pre-stretch, force and torque enhancement are not expected. In the CM condition (Figure 37) shows that on average the knee and ankle in transition share in handling the maximum force prior to propulsion. However, in the SD condition the hip and the knee transition encompass maximum force prior to propulsion (Figure 36). Given the apparent coupling between at least two joints in the CM and SD, it seems that this could prevent the occurrence of torque enhancement. It is possible that due to greater rates of force development, participants coordinated the jump such that the more proximal joints with larger musculature contributed to the motion sooner. As the body is a series of rigid links the motion of a joint neighboring one in transition must affect torque output. Hence, torque enhancement must be derived and interpreted with caution. It appears from this study that force enhancement cannot be explained by torque enhancement and that torque enhancement does not play a major role in the SSC. Furthermore, H02 is rejected as torque depression occurred more frequently than torque enhancement in all conditions.

Figures 11, 15, 16, and 17 demonstrate the relationships between vertical force and hip height and torque and joint angle, respectively. For individuals the force-hip height relationship was not always linear. However, after averaging the data from all participants the force-hip height relationship approximates linearity. Torque-angle curves were always non-linear. Hence H03 is accepted.

Electromechanical Delay

Electromechanical delay has been suggested to represent the compliance of the series elastic component of muscle (Cavanagh & Komi, 1979) and therefore is an indirect indicator of

tendon stiffness. Muscle-tendon units with stiff tendons are more likely to exhibit force enhancement (Biewener & Blickhan, 1988). Furthermore, high rates of force development have been shown to stiffen the tendon due to its viscoelastic nature (Earp, Newton, Cormie, & Blazevich, 2013) and lead to lengthening of muscle fibers even during the concentric phase of a jump (Ishikawa, Komi, Finni, & Kuitunen, 2006). Hence it seemed possible that imposing an SSC condition that greatly increased RFD would likely lead to a shorter EMD and further to force enhancement.

To measure EMD the onset of EMG was determined using methods outlined by McDonagh and Santello (1998). Next a kinematic event was identified to represent the completion of EMD. Norman and Komi (1979) used a change in acceleration as this event. Similarly, in the present study a change in acceleration was identified, specifically the extensor muscles peak flexion velocity was selected. More specifically, for the soleus, peak dorsiflexion velocity marked the end of EMD. For the vastus lateralis and gluteus maximus peak knee flexion velocity and hip flexion velocity marked the completion of EMD, respectively. Positive EMD values indicate that EMG onset occurred prior to the kinematic event. Conversely, negative EMD values indicate that EMG onset occurred after the kinematic event.

Utilizing these methods, significant differences between conditions were found. For the right and left tibialis anterior the mean EMD in SJ was significantly longer than in the CM and SD. Furthermore, the SJ condition presented a positive value EMD, whereas the CM and SD showed a negative value EMD (Figure 21, Table 5). This means that in the CM and SD conditions, EMG onset occurred after the kinematic event. Additionally the large EMD value in the SJ condition can be attributed to maintaining dorsiflexion. In other words in the SJ the tibialis anterior does not exhibit true EMD because it is active to maintain dorsiflexion. In contrast, the

tibialis anterior should have EMD in the CM and SD, since some musculature must cause dorsiflexion. However, the shorter and negative EMD values in the CM and SD suggest that dorsiflexion may be initiated by more proximal musculature or torque at a more proximal joint (Kepple, Siegel, & Stanhope, 1997).

EMD in the right soleus was different between the CM versus SJ and CM versus SD. Similarly in the left soleus differences were between the CM and SJ and CM and SD. Without applying the Bonferroni correction CM and SD would also be significantly different in the right soleus. Such a result adds to the developing patter of CM and SD being significantly different. The fact that EMD in the CM condition is primarily negative indicates that muscle activity began after the kinematic event used to timing. First this highlights the possibility that more proximal musculature initiated the change in joint velocity. Additionally, it is possible that due to the large RFD seen in SD but not in CM, earlier onsets of muscle activity may have been necessary for muscle tuning (Wakeling & Nigg, 2001).

Significant EMD differences between conditions were also found for the right and left vastus lateralis muscles. For both the right and left vasti EMD differences were between the CM versus SD and the CM versus SJ conditions. Again the general pattern of negative EMD in CM but positive in the SD and SJ was found. This shows that knee flexion can be reversed without contribution of the vastus lateralis in the CM. Whether or not this suggests that the CM is optimized for minimizing energetic cost by relying on more proximal musculature is unclear. In the SD condition the vasti have an earlier onset relative to the movement history time, which may be necessary to withstand the large RFD in SD compared to CM.

EMD results from the right and left gluteus maximus muscles revealed significant differences between conditions. Differences were between CM versus SD and CM versus SJ. EMD

in the CM was significantly longer than in SD and SJ. Again EMD in the CM was negative, but in the SD and SJ it was positive. It seems less likely that more proximal musculature was necessary to initiate a change in joint velocity of the hip. Therefore, it is possible that a better kinematic event for measuring EMD should be selected. Finally, results at the hip, similar to the other joints, fails to support the hypothesis that EMD is shortened in the SD compared to the CM and SJ. However, a clear pattern has been established: the timing of muscle activity in the CM is different from SD and SJ relative to the kinematics of the movement.

Electromyography

Peak normalized electromyography was also examined in this study, specifically during the concentric phase. Significant differences were found at the right tibialis anterior between the CM versus SD conditions. Given that the tibialis anterior muscle provides dorsiflexion, which is a motion that does not contribute to propulsion, this difference is irrelevant. The fact that the other musculature does not show significant differences is consistent with the literature (Walshe, Wilson, & Ettema, 1996). This indicates that other factors besides active state contribute to the concentric phase of the vertical jump.

Performance

Many factors associated with vertical jump performance were evaluated in this study. It was suggested that measures such as jump height, take-off velocity, impulse, and RFD would be increased in the SD conditions compared to CM and SJ. Results of jump height demonstrated that the CM outperformed SD and SJ, respectively. Take-off velocity was also highest in the CM followed by SD and SJ, respectively. Again CM led SD and SJ in concentric impulse. In contrast SD showed the highest RFD followed by CM and SJ, respectively. Altogether the performance results point to the CM as being the superior technique for jumping, despite having the lowest

eccentric load or RFD. While increased eccentric load tends to lead to improved concentric propulsion (McCarthy, Wood, Bolding, Roy & Hunter, 2012) it is possible that the eccentric load was too great for improved performance. Furthermore, the population was not trained in performing the SD condition. Hence, coordination could have been an issue as shown in Figures 31-33. Therefore, H05 is rejected due to the CM outperforming the SD, but given the lack of training and the high RFD the SD should be studied farther. Developing better coordination may allow for better performances in SD condition.

Synthesis

Overall, the results of the study were unexpected. The original logic behind the study was formulated based on the force enhancement property of muscle and the viscoelasticity of tendons. That is, a force production of a muscle is increased with active stretch, and active stretch is most probable given a stiff tendon. For example, a compliant tendon will stretch more easily due to muscle fiber tension and changes in joint angle. Thus, a compliant tendon allows the muscle fibers to remain isometric or even shorten in SSC. Furthermore because of the viscoelasticity of tendons, stiffness can be modulated through rate of load (Wren, Yerby, Beaupré, & Carter, 2001). Rate of tendon loading can be increased in a SSC (Earp, Newton, Cormie, Blazevich, 2013). Hence, it was hypothesized that by increasing the RFD in an SSC (accomplished through the SD condition) one could acutely increase tendon stiffness. This could be inferred from a shorter EMD. Due to the increased stiffness of the tendon some of the length change in the muscle due to joint angle change (movement history) would be taken up by the muscle fibers. In other words the muscle fiber might undergo active stretch, inducing muscle force enhancement. Provided that enough muscles experience muscle force enhancement this could be indicated by ground reaction force enhancement and joint torque enhancement. Finally,

given the results of Biewener and Blickhan (1988), the occurrence of force and torque enhancement was expected to result in improved jump performance.

However, the recurring pattern in EMD does not support such logic. The recurring pattern was that the CM was significantly different from the SD and SJ conditions. Additionally EMD in the CM was negative, whereas EMD tended to be positive in the SD and SJ conditions. One suggestion is that a different kinematic variable for measuring EMD is necessary. A better kinematic event might be a change from flexion velocity to extension velocity of the joint. This would provide positive average EMD for each condition. However, the results still demonstrate that by changing movement history the onset of muscle activity changes relative to the certain kinematic events. The logical consequence is that movement history may affect the time available for active state development prior to maximum force at the onset of the concentric portion of the motion (Bobbert & Casius, 2005). Additionally because the pattern of EMD differences (i.e. differences between CM and SD) varies from the pattern of differences in force and torque enhancement (i.e. CM and SD are not different), this measure of EMD cannot provide any further insight into changes in force and torque enhancement in the conditions involving movement history.

Therefore, an analysis of the interval from EMG onset to joint extension (i.e. plantarflexion, knee extension, and hip extension, which yields positive EMD values for CM) might provide a better analysis. The results of such an analysis demonstrated that the time from EMG onset to joint extensions is no different in the SD condition compared to the CM, but CM and SD have significantly longer EMD than the SJ condition. Hence this measure of EMD shows a pattern of statistical differences similar to force and torque enhancement. However, contrary to the hypothesis, EMD was longer in CM and SD compared to SJ. Hence, shorter EMD occurred

in the conditions with the least endpoint force enhancement. As a result this measure of EMD may be better understood as active state. The results of this study would then indicate that conditions involving SSC allow for greater active state development at the onset of concentric motion compared to SJ, which confirms the view of Bobbert and Casius (2005). Hence, EMD did not decrease with an increase in eccentric load or RFD, which was significantly greater in SD. Finally, H04 is rejected as the SD condition showed longer or similar EMD values to the CM and SJ.

However, the expectations for torque enhancement did not occur either, despite the fact that ground reaction force enhancement did. Nonetheless torque depression during the SD condition was most often smaller compared to CM and SJ. This demonstrates that ground reaction force enhancement was not simply an increase in the force capacity and torque application of any one muscle. Instead, the participant may prefer to take advantage of motor redundancy and coordination to avoid muscle force enhancement. Additionally, EMD (onset of EMG to beginning of concentric joint motion as opposed to the original measure shown in Chapter 4) tended to be longer in the SD condition compared to the CM and SJ, which goes against the original logic of the study. Therefore, in addition to coordinating motions differently, movement histories that create larger rates of force development may be dealt with by relatively earlier onsets of muscle activity to increase active state. This would also explain the increase in EMD of the CM over the SJ condition. Furthermore, Bobbert and Casius (2005) demonstrated that the CM provides the movement history for greater active state development over the SJ, which leads to improved performance. Given that EMD was greater in the SD compared to the CM, although this was not always significant, it seems that this indicates a potentially greater active state in the SD condition. This greater active state resulted in a trend toward decreased

torque depression, but not improved performance (i.e., jump height, velocity, and impulse). However, the onset of EMG in SD tends to be earlier relative to the movement time (beginning of movement to take-off). This suggests that active state may be "used up" by the development of force and therefore not available for propulsion. Overall, the coordination of the joints relative to maximum force prior to propulsion and the earlier development of active state may not be beneficial for optimal muscle mechanics but may prevent inducing muscle force enhancement. Preventing muscle force enhancement in everyday movements may be of safety concern, as Lieber and Friden (1993) showed that muscle damage occurs due to active muscle strain. In other words, the active stretch necessary for force enhancement may be dangerous, if it occurs often. Therefore, humans may avoid force enhancement by different coordination strategies and altered timing of active state development.

In summary, altering movement history did not change force enhancement, torque enhancement or EMD between SSC conditions. Some possible explanations are movement coordination (Figure 35, 36, 37). It is possible that participants coordinated the movements to avoid the development of torque enhancement and maintain performance. Furthermore coordination patterns may allow the appropriate amount of active state to withstand the rise in force that occurred with each movement.

Future Research

More understanding is needed of how movement history affects the movement and force capacity of the muscle in everyday movements. This study found that participants utilized different strategies to accomplish the different conditions. Hence it is difficult to gain an understanding of muscle contributions without seeing the muscle itself. As a result it is suggested that ultrasound techniques be used to gain a better understanding of muscle behavior during

motions utilizing different movement histories. Furthermore, the use of ultrasound in conjunction with motion capture and EMG may allow for correlations to be made between different movement histories and muscle motion. The author believes that timing of EMG and changes in joint velocity may provide insight into muscle mechanics *in vivo*. If adequate relationships can be found, these relationships can be employed by applied research laboratories and clinicians to better understand muscle actions involved in different movements without being invasive. Furthermore, this could provide insight into muscle strain and injury in various circumstances.

REFERENCES

- Abbott, B. C. & Aubert, X. M. (1952). The force exerted by active striated muscle during and after change of length. *Journal of Physiology*, 117, 77-86.
- Alexander, R. M. (2002). Tendon elasticity and muscle function. *Comparative Biochemistry and Physiology Part A*, 133, 1001-1011.
- Alexander, R. M., & Ker, R. F. (1990). The architecture of leg muscles. In S. L. Woo & J. M Winters (Eds.), *Multiple muscle systems* (pp. 568-577). New York, NY: Springer.
- Anderson, D. E., Madigan, M. L., & Nussbaum, M. A. (2007). Maximum voluntary joint torque as a function of joint angle and angular velocity: Model development and application to the lower limb. *Journal of Biomechanics*, 40, 3105-3113.
- Anderson, F. C. & Pandy, M. G. (1993). Storage and utilization of elastic strain energy during jumping. *Journal of Biomechanics*, 26(12), 1413-1427.
- Angle, T. C., Gillette, R. L., & Weimar, W. H. (2012). Kinematic analysis of maximal movement initiation in Greyhounds. *Australian veterinary journal*, 90(3), 60-68.
- Arakawa, H., Nagano, A., Yoshioka, S., & Fukashiro, S. (2010). Interaction between elastic energy utilization and active state development within the work enhancing mechanism during countermovement. *Journal of Electromyography and Kinesiology*, 20, 340-347.
- Arampatzis, A., Stafilidis, S., DeMonte, G., Karamanidis, K., Morey-Klapsing, G., & Bruggeman, G. P. (2005). Strain and elongation of the human gastrocnemius tendon and aponeurosis during maximal plantarflexion effort. *Journal of Biomechanics*, *38*, 833-841.
- Azizi, E. & Roberts, T. J. (2010). Muscle performance during frog jumping: influence of elasticity on muscle operating lengths. *Proceedings of the Royal Society: Biological Sciences*, 277, 1523-1530.doi: 10.1098/rspb.2009.2051
- Basmajian, J. V. & Deluca, C. J. (1985). Muscles alive: Their functions revealed by electromyography. 5th ed. Baltimore, MA: Williams and Wilkins.
- Biewener, A. A. & Blickhan, R. (1988). Kangaroo rat locomotion: Design for elastic energy storage or acceleration? *Journal of Experimental Biology*, 140, 243-255.
- Bobbert MF & Casius LJ. (2005). Is the effect of a countermovement on jump height due to active state development? *Medicine & Science in Sports & Exercise*, 37(3): 440-446.

- Bobbert, M. F., Gerritsen, M. C., Litjens, M.C.A., and Van Soest, A. J. (1996). Why is countermovement jump height greater than squat jump height? *Medicine & Science in Sports & Exercise*, 28, 1402 1412.
- Bobbert, M. F., Huijing, P. A., & Van Ingen Schenau, G. J. (1987). Drop jumping. 1. The influence of jumping technique on the biomechanics of jumping. *Medicine & Science in Sports & Exercise*, 19(4), 332-338.
- Bobbert, M. F., Mackay, M., Schinkelshoek, D., Huijing, P. A., & van Ingen Schenau, G. J. (1986). Biomechanical analysis of drop and countermovement jumps. *European Journal of Applied Physiology*, *54*, 566-573.
- Brown, I.E. & Loeb, G. E. (2000). Measured and modeled properties of mammalian skeletal muscle: III. the effects of stimulus frequency on stretch-induced force enhancement and shortening-induced force depression. *Journal of Muscle Research and Cell Motility*, 21, 21-31.
- Cavagna, G. A., and Citterio, G. (1974). Effect of stretching on the elastic characteristics and the contractile component of frog striated muscle. *Journal of Physiology*, 239, 1-14.
- Cavagna, G. A., Dusman, B., & Margaria, R. (1968). Positive work done by a previously stretched muscle. *Journal of Applied Physiology*, 24(1), 21-32.
- Cavanagh, P. R., & Komi, P. V. (1979). Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. *European journal of applied physiology and occupational physiology*, 42(3), 159-163.
- Costa, Pablo B., Ryan, Eric D., Herda, Trent J., Walter, Ashley A., Hoge, Katherine M., Cramer, Joel T. (2010). Acute effects of passive stretching on the electromechanical delay and evoked twitch properties. *European Journal of Applied Physiology*, 108, 301-310.
- De Luca C. J., Gilmore L. D., Kuznetsov, M., & Roy S. H. (2010). Filtering the Surface EMG signal: Movement artifact and baseline noise contamination. *Journal of Biomechanics*, 43 (8): 1573- 1579.
- De Monte, G. & Arampatzis, A. (2008). Influence of different shortening velocities preceding stretch on human triceps surae moment generation in vivo. *Journal of Biomechanics*, 41, 2272-2278.
- De Monte, G. & Arampatzis, A. (2009). In vivo moment generation and architecture of the human platar flexors after different shortening-stretch cycles velocities. *Journal of Electromyography and Kinesiology*, 19, 322-330.
- Diamant, J., Keller, A., Baer, E., Litt, M., & Arridge, R. G. C. (1972). Collagen: Ultrastructure and its relation to mechanical properties as a function of ageing. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, 180(1060), 293-315.

- Earp, J. E., Newton, R. U., Cormie, P., & Blazevich, A. J. (2013). The influence of loading intensity on muscle-tendon unit behavior during maximal knee extensor stretch shortening cycle exercise. *European Journal of Applied Physiology*, 114(1), 59-69.
- Edman, K. A. P. (2012). Residual force enhancement after stretch in striated muscle. *Journal of Physiology*, 590(6), 1339-1345.
- Edman, K. A. P. & Tsuchiya, T. (1996). Strain of passive elements during force enhancement by stretch in frog muscle fibres. *Journal of Physiology*, 490(1), 191-205.
- Edman, K. A. P., Elzinga, G., & Noble, M. I. (1978). Enhancement of performance by stretch during tetanic contractions of vertebrate skeletal muscle fibres. *Journal of Physiology*, 281, 139-155.
- Edman, K. A. P., Elzinga, G., & Noble. M. I. (1982). Residual force enhancement after stretch of contracting frog single muscle fibers. *Journal of General Physiology*, 80, 769-784.
- Epstein, M., Wong, M., & Herzog, W. (2006). Should tendon and aponeurosis be considered in series?. *Journal of biomechanics*, 39(11), 2020-2025.
- Esposito, F., Limonta, E., & Ce, E. (2010). Passive stretching effects on electromechanical delay and time course of recovery in human skeletal muscle: new insights from an electromyographic and mechanomyographic combined approach. *European Journal of Applied Physiology*, 111, 485-495.
- Gordon, A. M., Huxley, A. F., & Julian, F. J. (1966). The variation in isometric tension with sarcomere length in vertebrate muscle fibres. *Journal of Physiology*, *184*, 170-192.
- Griffiths, R. I. (1991). Shortening of muscle fibres during stretch of the active cat medial gastrocnemius muscle: the role of tendon compliance. *Journal of Physiology*, *436*(1): 219-236.
- Hahn, D. (2008). Force-length properties of leg extension and their implications for strength diagnostics. In *The XXVI International Conference on Biomechanics in Sports*. Korean Society of Biomechanics in Sports, Seoul/Korea (pp. 120-123).
- Hahn, D., Olvermann, M., Richtberg, J., Seiberl, W., & Schwirtz, A. (2011). Knee and ankle joint torque-angle relationships of multi-joint leg extension. *Journal of Biomechanics*, 44, 2059-2065.
- Hahn, D., Seiberl, W, Schmidt, S., Schweizer, K., Schwirtz, A. (2010). Evidence of residual force enhancement for multi-joint leg extension. *Journal of Biomechanics*, 43, 1503-1508.
- Hansen, P., Bojsen-Moller, J., Aagaard, P., Kjaer, M., & Magnusson, S. P. (2006). Mechanical properties of the human patellar tendon, in vivo. *Clinical Biomechanics*, 21, 54-58.

- Herzog, W. (2007). Muscle. In B. M. Nigg & W. Herzog (Eds.), *Biomechanics of the musculo-skeletal system* (pp. 169-225). West Sussex, England: John Wiley & Sons Ltd.
- Herzog, W. (2004). History dependence of skeletal muscle force production: Implications for movement control. *Human Movement Science*, 23, 591-604.
- Herzog, W. & Leonard, T. R. (2000). The history dependence of force production in mammalian skeletal muscle following stretch-shortening and shortening-stretch cycles. *Journal of Biomechanics*, *33*, 531-542.
- Herzog, W., Joumaa, V., & Leonard, T. R. (2010). The force-length relationship of mechanically isolated sarcomeres. In D. L. Rassier (Ed.), *Muscle Biophysics* (pp. 141-161). New York, NY: Springer.
- Herzog, W., & ter Keurs, H. E. (1988). A method for the determination of the force-length relation of selected in-vivo human skeletal muscles. *Pflügers Archiv*, 411(6), 637-641.
- Herzog, W., & ter Keurs, H. E. (1988). Force-length relation of in-vivo human rectus femoris muscles. *Pflügers Archiv*, 411(6), 642-647.
- Hill, A. V. (1938). The heat of shortening and the dynamic constants of muscle. *Proceedings of the Royal Society of London, Series B-Biological Sciences, 126*, 136-195.
- Hill, A. V. (1949). The abrupt transition from rest to activity in muscle. *Proceedings of the Royal Society of London, Series B-Biological Sciences*, 136(884), 399-420.
- Hill, A. V. (1951). The effect of series compliance on the tension developed in a muscle twitch. *Proceedings of the Royal Society of London. Series B-Biological Sciences*, *138*(892), 325-329.
- Hirayama, K., Yanai, T., Kanehisa, H., Fukunaga, T., & Kawakami, Y. (2012). Neural modulation of muscle-tendon control strategy after a single practice session. *Medicine & Science in Sports & Exercise*, 44(8), 1512-1518.
- Hisey, B., Leonard, T. R., Herzog, W. (2009). Does residual force enhancement increase with increasing stretch magnitudes? *Journal of Biomechanics*, 42, 1488-1492.
- Hopkins, J. T., Brown, T. N., Christensen, L., & Palmieri-Smith, R. M. (2009). Deficits in peroneal latency and electromechanical delay in patients with functional ankle instability. *Journal of Orthopaedic Research*, 27(12), 1541-1546.
- Howatson, G., Glaister, M., Brouner, J., & van Someren, K. A. (2009). The reliability of electromechanical delay and torque during isometric and concentric isokinetic contractions. *Journal of Electromyography and Kinesiology*, 19, 975-979.
- Howatson, Glyn. (2010). The impact of damaging exercise on electromechanical delay in biceps brachii. *Journal of Electromyography and Kinesiology*, 20, 477-481.

- Huxley, H. & Hanson, J. (1954). Changes in the cross-striations of muscle during contraction and stretch and their structural inpterpretation. *Nature*, *173*, 973-976.
- Huxley, A. & Niedergerke, R. (1954). Structural changes in muscle during contraction: Interference microscopy of living fibres. *Nature*, *173*, 171-173.
- Huxley, A. F. & Simmons, R. M. (1971). Proposed mechanism of force generation in striated muscle. *Nature*, 233, 533-538.
- Ishikawa, M, Komi, P. V., Finni, T., & Kuitunen, S. (2006). Contribution of the tendinous tissue to force enhancement during stretch-shortening cycle exercise depends on the prestretch and concentric phase intensities. *Journal of Electromyography and Kinesiology, 16*, 423-431.
- Ishikawa, M., Niemelä, E., & Komi, P. V. (2005). Interaction between fascicle and tendinous tissues in short-contact stretch-shortening cycle exercise with varying eccentric intensities. *Journal of Applied Physiology*, 99(1), 217-223.
- Journaa, V. & Herzog, W. (2013). Energy cost of force production is reduced after active stretch in skinned muscle fibers. *Journal of Biomechanics*, 46, 1135-1139.
- Joumaa, V., Rassier, D. E., Leonard, T. R., & Herzog, W. (2007). The origin of passive force enhancement in skeletal muscle. *American Journal of Physiology Cell Physiology*, 294, C74-C78.
- Katz, B. (1939). The relation between force and speed in muscular contraction. *Journal of Physiology*, *96*, 45-64.
- Kawakami, Y. & Lieber, R. L. (2000). Interaction between series compliance and sarcomere kinetics determines internal sarcomere shortening during fixed-end contraction. *Journal of Biomechanics*, *33*, 1249-1255.
- Kawakami, Y., Muraoka, T., Ito, S., Kanehisa, H., & Fukunaga, T. (2002). In vivo muscle fibre behaviour during counter-movement exercise in humans reveals a significant role for tendon elasticity. *Journal of Physiology*, *540*(2), 635-646.
- Kendall, F. P., McCreary, E. K., Provance, P. G., Rodgers, M. M., & Romani, W. A. (2005). Muscles: Testing and function with posture and pain. 5th Ed. Baltimore, MD: Lippincott Williams & Wilkins.
- Kepple, T. M., Siegel, K. L., & Stanhope, S. J. (1997). Relative contributions of the lower extremity joint moments to forward progression and support during gait. *Gait & Posture*, 6(1), 1-8.
- Ker, R. F. (1981). Dynamic tensile properties of the plantaris tendon of sheep (ovis aries). *Journal of Experimental Biology*, 93, 283-302.

- Kieran, A. M., & Wallace, E. S. (2007). Eccentric loading and range of knee joint motion effects on performance enhancement in vertical jumping. *Human Movement Science*, 26(6), 824-840. http://dx.doi.org/10.1016/j.humov.2007.05.001.
- Koppes, R. A., Herzog, W., & Corr, D. T. (2013). Force enhancement in lengthening contractions of cat soleus muscle in situ: transient and steady-state aspects. *Physiological Reports*, *1*(2), 1-10. doi: 10.1002/phy2.17
- Kubo, K., Kanehisa, H., & Fukunaga, T. (2003). Gender differences in the viscoelastic properties of tendon structures. *European Journal of Applied Physiology*, 88, 520-526.
- Krouchev, N., Kalaska, J. F., & Drew, T. (2006). Sequential activation of muscle synergies during locomotion in the intact cat as revealed by cluster analysis and direct decomposition. *Journal of Neurophysiology*, *96*(4), 1991-2010.
- Kurokawa, S., Fukunaga, T., & Fukashiro, S. (2001). Behavior of fascicles and tendinous structures of human gastrocnemius during vertical jumping. *Journal of Applied Physiology*, *90*, 1349-1358.
- Kurokawa, S., Fukunaga, T., Nagano, A., & Fukashiro, S. (2003). Interaction between fascicles and tendinous structures during counter movement jumping investigated in vivo. Journal of Applied Physiology, 95, 2306-2314.
- Lacourpaille, L., Nordez, A., & Hug, F. (2013). Influence of stimulus intensity on electromechanical delay and its mechanisms. *Journal of Electromyography and Kinesiology*, 23(1), 51-55.
- Leonard, T. R. & Herzog, W. (2010). Regulation of muscle force in the absence of actin-myosin-based cross-bridge interaction. *American Journal of Cell Physiology*, 299, C14-C20.
- Lieber, R. L., Brown, C. G., & Trestik, C. L. (1992). Model of muscle-tendon interaction during frog semitendinosis fixed-end contractions. *Journal of Biomechanics*, 25(4), 421-428.
- Lieber, R. L., & Friden, J. (1993). Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology*, 74, 520-520.
- Linari, M., Lucii, L., Reconditi, M., Casoni, M. E. V., Amenitsch, H., Bernstorff, S., Piazzesi, G., & Lombardii, V. (2000). A combined mechanical and x-ray diffraction study of stretch potentiation in single frog muscle fibres. *Journal of Physiology*, *526*(3), 589-596.
- MacIntosh, B. R., Gardiner, P. F., & McComas, A. J. (2006). Skeletal muscle: Form and Function. Champagne, IL: Human Kinetics.
- Mackey, D. C., & Robinovitch, S. N. (2006). Mechanisms underlying age-related differences in ability to recover balance with the ankle strategy. *Gait & posture*, 23(1), 59-68.
- Maganaris, C. N. (2001). Force-length characteristics of in vivo human skeletal muscle. *Acta Physiological Scandinavica*, 172, 279-285.

- Maganaris, C. N. & Paul, J. P. (2000). Hysteresis measurements in intact human tendon. *Journal of Biomechanics*, 33, 1723-1727.
- McGowan, C. P., Neptune, R. R., & Herzog, W. (2013). A phenomenological muscle model to assess history dependent effects in human movement. *Journal of Biomechanics*, 46, 151-157.
- Merletti, R., & Di Torino, P. (1999). Standards for reporting EMG data. *Journal of Electromyography and Kinesiology*, 9(1), 3-4.
- McCarthy, J. P., Wood, D. S., Bolding, M. S., Roy, J. L., & Hunter, G. R. (2012). Potentiation of Concentric Force and Acceleration Only Occurs Early During the Stretch-Shortening Cycle. *The Journal of Strength & Conditioning Research*, 26(9), 2345-2355.
- Minozzo, F. C., Baroni, B. M., Correa, J. A., Vaz, M. A., & Rassier, D. E. (2013). Force produced after stretch in sarcomeres and half-sarcomeres isolated from skeletal muscles. *Scientific Reports*, *3*(2320), DOI: 10.1038/srep02320.
- Morse, C. L., Thom, J. M., Birch, K. M., & Narici, M. V. (2005). Tendon elongation influences the amplitude of interpolated doublets in the assessment of activation in elderly men. *Journal of Applied Physiology*, 98, 221-226.
- Muraoka, T., Muramatsu, T., Fukunaga, T., & Kanehisa, H. (2003). Influence of tendon slack on electromechanical delay in the human medial gastrocnemius in vivo. *Journal of Applied Physiology*, *96*, 540 544.
- Nieminen, M. J. J., Piirainen, J. M., Salmi, J. A., & Linnamo, V. (2013). Effects of neuromuscular function and split step on reaction speed in simulated tennis response. *European Journal of Sport Science*, DOI: 10.1080/17461391.2013.785598
- Nordez, A., Gallot, T., Catheline, S., Guevel, A., Cornu, C., & Hug, F. (2009). Electromechanical delay revisited using very high frame rate ultrasound. *Journal of Applied Physiology*, *106*, 1970-1975.
- Norman, R. W., & Komi, P. V. (1979). Electromechanical delay in skeletal muscle under normal movement conditions. *Acta Physiologica Scandinavica*, 106(3), 241-248.
- Osigo, K., McBride, J.M., & Komi, P.V. (2002). Stretch-reflex mechanical response to varying types of previous muscle activities. *Journal of Electromyography and Kinesiology*, 12, 27-36.
- Oskouei, A. E. & Herzog, W. (2009). Activation-induced force enhancement in human adductor pollicis. *Journal of Electromyography and Kinesiology*, 19, 821-828.
- Peterson, D. R., Rassier, D. E., & Herzog, W. (2004). Force enhancement in single skeletal muscle fibres on the ascending limb of the force-length relationship. *The Journal of Experimental Biology*, 207, 2787-2791.

- Rassier, D. E. & Herzog, W. (2004). Active force inhibition and stretch-induced force enhancement in frog muscle treated with BDM. *Journal of Applied Physiology*, 97, 1395-1400.
- Rassier, D. E., MacIntosh, B. R., & Herzog, W. (1999). Length dependence of active force production in skeletal muscle, *Journal of Applied Physiology*, 86(5), 1445-1457.
- Rigby, B. J., Hirai, N., Spikes, J. D., & Eyring, H. (1959). The mechanical properties of rat tail tendon. *The Journal of General Physiology*, 43, 265-284.
- Roberts, T. J., Marsh, R. L., Weyand, P. G., & Taylor, C. R. (1997). Muscular force in running turkeys: The economy of minimizing work. *Science*, 275, 1113-1115.
- Rubenson, J., Pires, N. J., Loi, H. O., Pinniger, G. J., & Shannon, D. G. (2012). On the ascent: the soleus operating length is conserved to the ascending limb of the force-length curve across gait mechanics in humans. *The Journal of Experimental Biology*, 215, 3539-3551.
- Sasaki, K., Sasaki, T., & Ishii, N. (2011). Acceleration and force reveal different mechanisms of electromechanical delay. *Medicine & Science in Sports & Exercise*, 43, 1200-1206.
- Schachar, R., Herzog, W., & Leonard, T. R. (2004). The effects of muscle stretching and shortening on isometric forces on the descending limb of the force-length relationship. *Journal of Biomechanics*, *37*, 917-926.
- Seiberl, W., Hahn, D., Herzog, W., & Schwirtz, A. (2012). Feedback controlled force enhancement and activation reduction of voluntarily activated quadriceps femoris during sub-maximal muscle action. *Journal of Electromyography and Kinesiology*, 22, 117-123.
- Seiberl, W., Paternoster, F., Achatz, F., Schwirtz, A., & Hahn, D. (2013). On the relevance of residual force enhancement for everyday movement. *Journal of Biomechanics*, 46, 1996-2001.
- Shadwick, R. E. (1990). Elastic energy storage in tendons: mechanical differences related to function and age. *Journal of Applied Physiology*, 68(3), 1033-1040.
- Shim, J. & Garner, B. (2012). Residual force enhancement during voluntary contractions of knee extensors and flexors at short and long muscle lengths. *Journal of Biomechanics*, 45, 913-918.
- Sugi, H. & Tsuchiya, T. (1988). Stiffness changes during enhancement and deficit of isometric force by slow length changes in frog skeletal muscle fibres. *Journal of Physiology*, 407, 215-229.
- Uzu, R., Shinya, M., & Oda, S. (2009). A split-step shortens the time to perform a choice reaction step-and-reach movement in a simulated tennis task. *Journal of Sport Sciences*, 27(12), 1233-1240. DOI: 10.1080/02640410903233222

- Van Ingen Schenau, G. J. (1989). From rotation to translation: constraints on multi-joint movements and the unique action of bi-articular muscles. *Human Movement Science*, 8, 301-337.
- Wakeling, J. M., & Nigg, B. M. (2001). Modification of soft tissue vibrations in the leg by muscular activity. *Journal of Applied Physiology*, 90(2), 412-420.
- Walshe, A. D., Wilson, G. J., & Ettema, G. J. C. (1998). Stretch-shorten cycle compared with isometric preload: contributions to enhanced muscular performance. *Journal of Applied Physiology*, 84, 97-106.
- Wang, X. T., Ker, R. F., & Alexander, R. M. (1995). Fatigue rupture of wallaby tail tendons. The *Journal of Experimental Biology*, 198, 847-852.
- Whiting, C. W. & Rugg, S. (2012). Dynatomy: Dynamic Human Anatomy. Champagne, IL: Human Kinetics.
- William, G. P. (1997). Statistical Analysis Methods for Chemists: A Software Based Approach. Cambridge, UK: The Royal Society of Chemistry.
- Wren, T. A. L., Yerby, S. A., Beaupré, G. S., & Carter, D. R. (2001). Mechanical properties of the human achilles tendon. *Clinical Biomechanics*, 16, 245-251.
- Zatsiorsky, V. M. & Prilutsky, B. I. (2012). Biomechanics of skeletal muscles. Champaign, IL: Human Kinetics.
- Zatsiorsky, V. M. (2003). Biomechanics of strength and strength training. In P. V. Komi (Ed.), Strength and power in sport, (pp. 439-487). Oxford: Blackwell Science.

Appendix A

Participant Screening Questionnaire

| quest | ion, pl | each question carefully and answer honestly. If you do not understand the lease ask the investigator for clarification. Check the appropriate answer. Number: |
|--------|-----------|--|
| Yes | <u>No</u> | |
| | | 1. Are you between the ages of 19 to 30? |
| | | 2. Within the past year have you had, or do you currently have a lower extremity injury that prevents you from performing lower extremity ballistic exercise? |
| | | 3. Within the past year have you had, or are you currently recovering from lower extremity surgery that prevents you from performing lower extremity ballistic exercise? |
| | | 4. Within the past year have you had, or do you currently have a trunk injury that prevents you from performing lower extremity ballistic exercise? |
| | | 5. Do you currently have an illness that prevents you from performing lower extremity ballistic exercise? |
| | | 6. Do you have any reason to believe that your participation in this investigation may put your health or well-being at risk? |
| | | 7. Are you allergic to adhesives? |
| 8. Do | you cı | urrently weight train at least 3 times per week? |
| 9. Hov | w long | have you been consistently weight training 3 times per week? |
| Signa | ture of | `participant Date |

Appendix B

****DO NOT AGREE TO PARTICIPATE UNLESS AN IRB APPROVAL STAMP HAS BEEN APPLIED TO THIS DOCUMENT****

INFORMED CONSENT FOR

Does force enhancement occur with preparatory movement?

You are invited to participate in a research study of "Does force enhancement occur with preparatory movement?" With your help it is hoped that the influence of loading configuration in a lower extremity ballistic exercise can be better understood. You were selected as a potential participant because you are between the ages 19 and 30 and your current health status permits you to perform the test safely and successfully. The results of the study will be used to investigate the development of force and utilization of muscle elasticity during variations of the countermovement.

Purpose: The purpose of this investigation is to evaluate force enhancement and the time delay between the onset of muscle activity and force development during various loading configurations. Hence, kinematic data will be gathered using a 10 camera motion capture system. Force will be measured from two force plates. In addition electromyography data will be collected to evaluate muscle activity. Combined this will allow us to answer the following questions: (1) Does movement history alter tendon compliance? (2) Does a less compliant tendon result in force/torque enhancement? (3) Does movement history influence performance?

Methodology: Three separate meetings will be arranged at the Auburn University Sports Biomechanics Laboratory (003 Kinesiology Building, 301 Wire Rd, Auburn, Al). At the first meeting, in addition to signing this form, you will complete a screening questionnaire that confirms your physical preparedness and lack of injury or fear of injury. Next, anthropometric data will be collected (height, weight, and limb measurements), and you will be familiarized with both the isometric squat and vertical jump protocols by practicing each condition. The isometric squat protocol involves eight isometric squats performed against an immovable bar. The vertical jump protocol involves vertical jumps with three different loading conditions. The no-countermovement will be completed by starting lowered to a preset knee angle. The countermovement condition involves beginning with the legs extended then lowering prior to propelling, similar to a vertical jump test. The self-induced drop is performed by beginning with the legs extended. You then quickly lift your feet from the ground, fall into a countermovement, and push maximally. This session will last approximately 30 minutes.

Participant Initials

A second meeting time will be arranged for vertical jump testing. You will return to the Auburn University Sports Biomechanics Laboratory. The second meeting will begin with preparation for the experiment. Compression clothing will be worn. We will begin careful skin preparation (shaving and cleaning with alcohol) and electrode placement over the muscle belly of the gluteus maximus, vastus lateralis, soleus, and tibialis anterior. After electrode placement is complete you will be allowed to warm-up. Next, you will perform a maximal voluntary contraction for each of the muscles of interest during which muscle activity will be recorded. In this way the muscle activity during performance of the experimental conditions can be compared to muscle activity during maximum effort. Retro-reflective markers will be placed at the feet, lower leg, upper leg, hips and trunk for motion capture data. Next, the experimental protocol begins. A static motion capture trial will be performed for calibration. The order of jump conditions will be randomized. Three trials of each condition will be performed. The protocol involves vertical jumps with three different loading conditions. The no-countermovement will be completed by starting lowered to a preferred preset knee angle. The countermovement condition involves beginning with the legs extended then lowering prior to propelling, similar to a vertical jump test. The self-induced drop is performed by beginning with the legs extended. You then quickly lift your feet from the ground, fall into a countermovement, and push maximally. Unsuccessful trials will be repeated. The second session will last approximately 1 hour.

A third meeting time will be arranged for the isometric squat protocol. This protocol defines a strength curve for leg extension. Again, you will return to the Auburn University Sports Biomechanics Laboratory. Preparations will be the same as in the vertical jump protocol. You will be given time to perform your normal warm-up routine. Compression clothing will be worn, skin will be prepared and electrodes placed at the same locations, retro-reflective markers will be placed at the same anatomical locations. MVICs will be performed for the muscles of interest. Another static calibration will be performed. After the static capture you will perform eight, maximum effort, isometric leg extensions underneath a bar locked in place. The bar will be moved to various heights corresponding to shoulder height during the vertical jumps. You will be asked to achieve maximum force as rapidly as possible and to hold with maximum effort until a plateau or drop is seen in the force trace (approximately 2 seconds). A minimum of 2 minutes rest will be provided between each isometric leg extension. The third meeting will take approximately 1 hour. Hence, a total time of 2.5 hours is required for the study. Once the third meeting is complete you will be thanked for your participation.

Participant Initials

Risk: While participating in this study it is possible that you might incur a joint sprain, muscle strain, or muscle soreness. Furthermore, these factors may lead to serious injury or death. However, injuries are unlikely due to care taken to warm-up. It should be noted that you will be responsible for any and all medical cost resulting from injury during or related to this study.

Benefit: There is no direct benefit to you other than the opportunity to learn about loading configurations and their effect on power development.

Confidentiality: Any information obtained in connection with this study that can be identified with you will remain confidential. Your decision whether or not to participate will not jeopardize your relation with Auburn University or the School of Kinesiology. If you decide later to withdraw from the study you may also withdraw any identifiable information, which has been collected about you in this study.

Contact/Questions: If you have any questions now or later, please feel free to contact John Fox (jwf0007@tigermail.auburn.edu) or Wendi H. Weimar (weimawh@auburn.edu) at (334) 844-1468. Additionally you may contact the IRB Chair at hsubjec@auburn.edu or IRBchair@auburn.edu or you may call them at (334) 844-5966. You will be given a copy of this form to keep.

YOU ARE MAKING A DECISION WHETHER OR NOT TO PARTICIPATE. YOUR

| READ THE INFORMATION | PROVIDED ABOVE. | | |
|-------------------------------|-------------------------|------|--|
| Participant's Printed Name | Participant's Signature | Date | |
| Investigator Conducting Conse | ent | | |

SIGNATURE INDICATES THAT YOU HAVE DECIDED TO PARTICIPATE HAVING

Appendix C

Free Body Diagram and Equations for Force and Moment Calculations

Foot

$$F_{Ax}$$
 GRF_x
 GRF_y

$$\sum F_x = m_{foot} a_{foot_x}$$

$$\sum F_x = GRF_x + F_{Ax}$$

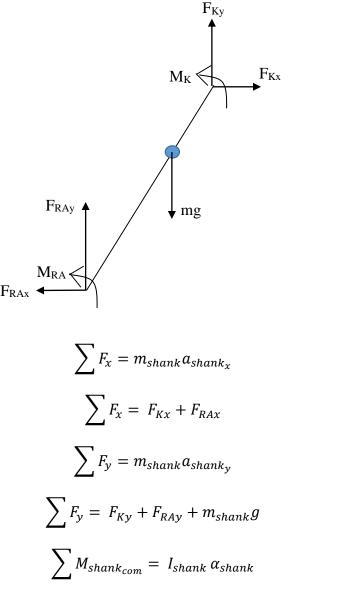
$$\sum F_y = m_{foot} a_{foot_y}$$

$$\sum F_y = GRF_y + F_{Ay} + m_{foot}g$$

$$\sum M_{foot_{com}} = I_{foot} \alpha_{foot}$$

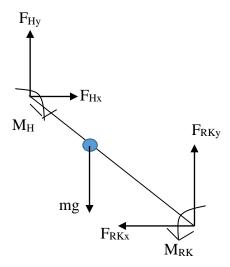
$$\sum M_{foot_{com}} = GRF_y l_{GRF_y} + GRF_x l_{GRF_x} + F_{Ay} l_{F_{Ay}} + F_{Ax} l_{F_{ax}} + M_A = I_{foot} \alpha_{foot}$$

Shank



$$\sum M_{shank_{com}} = F_{Ky}l_{F_{Ky}} + F_{Kx}l_{F_{Kx}} + F_{RAy}l_{F_{RAy}} + F_{RAx}l_{F_{RAx}} + M_{RA} + M_K = I_{shank} \alpha_{shank}$$

Thigh



$$\sum F_{x} = m_{thigh} a_{thigh_{x}}$$

$$\sum F_{x} = F_{Hx} + F_{RKx}$$

$$\sum F_{y} = m_{thigh} a_{thigh_{y}}$$

$$\sum F_{y} = F_{Hy} + F_{RKy} + m_{thigh} g$$

$$\sum M_{thigh_{com}} = I_{thigh} \alpha_{thigh}$$

$$\sum M_{thigh_{com}} = F_{Hy}l_{F_{Hy}} + F_{Hx}l_{F_{Hx}} + F_{RKy}l_{F_{RKy}} + F_{RKx}l_{F_{RKx}} + M_{RK} + M_H = I_{thigh} \alpha_{thigh}$$