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MUSCULAR AND NEURAL CONTRIBUTIONS TO POSTACTIVATION POTENTIATION

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MUSCULAR AND NEURAL CONTRIBUTIONS TO POSTACTIVATION POTENTIATION

DISSERTATION

A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy in the
College of Education at the University of Kentucky

By
Brian Joseph Wallace

Lexington, Kentucky

Director: Dr. Robert Shapiro, Professor of Kinesiology and Health Promotion

Lexington, Kentucky

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ABSTRACT OF DISSERTATION

MUSCULAR AND NEURAL CONTRIBUTIONS TO POSTACTIVATION POTENTIATION

Muscle performance is partially a consequence of its recent contractile history. Postactivation potentiation (PAP) can occur after muscle contractions and leads to enhanced neuromuscular performance. The purpose of this dissertation was to explain the relationship between muscle factors (twitch potentiation, TP) and neural factors (reflex potentiation, RP) contributing to overall PAP following a non-fatiguing volitional muscle contraction. The tibial nerves of fifteen resistance trained volunteers (eleven men, four women) were stimulated intermittently at supramaximal (Mmax) and submaximal (Hmax) intensities for 20 minutes on separate days under three conditions: rest (Control); after a after a 10 second maximum voluntary isometric contraction (MVIC) of the plantarflexors; and after a low frequency fatigue protocol prior to the MVIC. Plantarflexion isometric torque and rate of force development (RFD), and soleus and gastrocnemius EMG Hmax/Mmax ratios, were analyzed. Both experimental conditions resulted in TP at 10 seconds post-MVIC compared to the control condition. The two experimental conditions were not different for any measure. Torque and RFD at Hmax (overall PAP) were highest at 3 and 4.5 minutes post MVIC, respectively, but were not significantly different from the control condition. EMG values generally were insignificantly increased in the experimental conditions versus the control condition. Mmax torque and RFD significantly contributed to Hmax torque and RFD at 20 seconds, Hmax peak, and 20 minute post-MVIC time points. The soleus significantly contributed to Hmax torque at 20 seconds and 20 minutes post-MVIC, and Hmax RFD at 20 seconds, 4.5 minutes, and 20 minutes post-MVIC. The results of this study suggest that both muscle and neural factors play a significant role in overall PAP, and that neural factors may play a more meaningful role in RFD potentiation than torque potentiation.

KEYWORDS: Postactivation Potentiation, Twitch Potentiation, Reflex Potentiation, H-reflex, Rate of Force Development

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February 25, 2015_____
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MUSCULAR AND NEURAL CONTRIBUTIONS TO POSTACTIVATION POTENTIATION

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This dissertation is dedicated to my wife, Kelly, and our two four-legged companions, Samson and Lucky. Kelly, you have been a source of support at every turn throughout the process of pursuing my Ph.D., even moving to Kentucky and staying for longer than expected. Samson and Lucky have been a source of companionship and joy from the day we brought them home off the streets. I would also like to dedicate this project to my parents, Patricia and Dan. Thank you for instilling in me the characteristics that are required to earn a doctorate, and for all of your support through the process.

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Chapter 1: Introduction

Background

It is well established in the scientific literature that the performance of skeletal muscle is partially affected by its recent contractile history. The most obvious example of this is the attenuation of muscle force output with fatigue. However, muscle contractions can also elicit muscle performance enhancing postactivation potentiation (PAP). PAP is a phenomenon where a muscle's contraction characteristics, such as force or rate of force development (RFD), are increased as a result of recent contractions (Robbins 2005; Sale 2002). A conditioning activity is necessary to elicit PAP. Usually this activity takes the form of either an electrically induced contraction or a maximum voluntary contraction (MVC), which is often performed isometrically (MVIC). When the conditioning activity is elicited by evoked high frequency electrical stimulation it is termed posttetanic potentiation (PTP). For clarity, in this paper PAP will be used to describe either phenomenon, as has been done previously (Tillin and Bishop 2009; Gossen and Sale 2000). The nature of the conditioning activity determines the PAP response among other factors (i.e.: training status, strength/power ratios, etc.). PAP measured in response to a maximal intensity muscle twitch is highest immediately after conditioning activities of short duration (i.e.: MVIC $\leq$ 10 seconds), while long duration (i.e.: MVIC $\geq$ 60 seconds) conditioning activities elicit both peripheral fatigue and PAP (Vandervoort et al. 1983; Houston and Grange 1990; Grange and Houston 1991). Other studies have investigated PAP by measuring reflexes after a conditioning activity, accounting for the nervous system's ability to recruit motor units. In these studies the

PAP response is delayed, even with short conditioning activities (Enoka et al. 1980; Gossen and Sale 2000; Gullich and Schmidtbleicher 1996; Folland et al. 2008). PAP is relatively long lasting, having been reported to last up to 16 minutes after an electrically evoked conditioning activity, and up to 18 minutes after a 10 second MVIC (Kitago et al. 2004; Folland et al. 2008).

PAP is commonly thought of as a muscle property that is only apparent at low stimulation frequencies, most notably with a muscle twitch (Sale 2004; Sale 2002). Although the magnitude of the PAP effect on maximal force output diminishes with increasing activation frequencies, it is still present at high, but sub-maximal, shortening velocities and frequencies (Sale 2002; Abbate et al. 2000; MacIntosh and Willis 2000; Baudry et al. 2008). Additionally, both muscle twitches and dynamic movement increase RFD after conditioning contractions (Abbate et al. 2000; Baudry and Duchateau 2007; Baudry et al. 2005, 2008; MacIntosh et al. 2008; Sale 2002; Vandenboom et al. 1993; Rixon et al. 2007; Requena et al. 2005a; Requena et al. 2011; Paasuke et al. 2007). The most commonly described mechanism behind these PAP responses is phosphorylation of the myosin regulatory light chains (RLC) (Houston et al. 1985; Moore and Stull 1984; Rassier and Macintosh 2000; Sweeney et al. 1993; Grange et al. 1993). The phosphorylation of the RLCs is triggered by the enzyme myosin light chain kinase (MLCK) (Moore and Stull 1984; Stuart et al. 1988; Sweeney et al. 1993). When calcium is released from the sarcoplasmic reticulum during a muscle contraction, MLCK is activated (Bowman et al. 1992; Blumenthal and Stull 1980). Calcium binds to calmodulin, and this complex attaches to MLCK, initiating RLC phosphorylation (Klug et al. 1982). It was first hypothesized that RLC phosphorylation causes a conformational

change of the myosin head by moving it closer to the thin filament, increasing force at low calcium concentrations (Persechini et al. 1985; Sweeney and Stull 1986). This hypothesis was later supported by Sweeney and Stull (1990), who reported that the rate constant describing this transition from the non-force binding to the force binding state was increased with RLC phosphorylation. Such a change would increase the number of cross bridge attachments at a given calcium concentration since the probability of myosin attaching to actin increases as their separation distance decreases (Abbate et al. 2000; MacIntosh et al. 2008; Sweeney and Stull 1990; Huxley 1969). The mechanism by which RLC phosphorylation acts appears to be via a conformational change of the myosin head as described by Persechini et al. (1985) and Sweeney and Stull (1986), and supported by subsequent research (Levine et al. 1996). This may also be a mechanism by which RFD is increased at high calcium concentrations following a conditioning activity (Sale 2004; Sale 2002; Baudry and Duchateau 2007).

Early studies using fast twitch muscle with animal models concluded that twitch potentiation is regulated mostly or exclusively by phosphorylation of the RLCs (Manning and Stull 1979; Klug et al. 1982; Manning and Stull 1982; Persechini et al. 1985; Moore et al. 1985; Moore and Stull 1984). An early study reported similar conclusions in mixed human muscle (Houston et al. 1985). More recent studies, however, have mostly suggested that PAP may be largely due to non-myogenic phenomenon(s) (Baudry and Duchateau 2007; Chiu et al. 2003; Hodgson et al. 2005; MacIntosh et al. 1993; Stuart et al. 1988; Tubman et al. 1996; Tubman et al. 1997; Vandenboom et al. 1995; Vandenboom and Houston 1996; MacIntosh et al. 2008; Hamada et al. 2000a). In fast twitch rat muscle (gastrocnemius) and mixed human muscle (quadriceps femoris) an

uncoupling of RLC phosphorylation and potentiation has been reported (MacIntosh et al. 1993; Tubman et al. 1996; Houston and Grange 1990; Stuart et al. 1988). In the animal studies there was more PAP following calcium attenuating low frequency fatigue (LFF) than what would have been expected based on RLC phosphate content. In investigations using human participants the opposite was true; likely because of the different conditioning stimuli between studies, the time courses over which PAP was measured, and the neural influence on the measures of muscle contractile performance.

The other primary mechanism besides RLC phosphorylation behind PAP is likely neurogenic (Gullich and Schmidtbleicher 1996; Hodgson et al. 2005; Trimble and Harp 1998; Tubman et al. 1996; Chiu et al. 2003). The Hoffmann reflex (H-reflex) response is widely recognized as a means of measuring motor neuron pool excitability during isometric muscle activation by applying a single electrical stimulus to a nerve and measuring the associated electrical (H-wave) and mechanical (twitch force or torque) response of the muscle (Enoka et al. 1980; Kitago et al. 2004; Macdonell et al. 1989; Moritani and Shibata 1994; Trimble and Harp 1998; Zehr 2002). An H-wave of greater amplitude can be attributed to more and/or larger motor units being recruited (Tillin and Bishop 2009; Gullich and Schmidtbleicher 1996). Only three studies have investigated the time course of H-reflex amplitude in response to a conditioning activity (Folland et al. 2008; Gullich and Schmidtbleicher 1996; Trimble and Harp 1998). Each of these studies reported a reduction in H-reflex amplitude after a conditioning activity, known as postactivation depression (PAD), followed by PAP of the H-reflex 3+ minutes thereafter. Specifically, Gullich and Schmidtbleicher (1996) reported a high ($r = .90$) temporal relationship between H-wave amplitude and plantarflexion force during maximal effort

voluntary contractions. Unlike the aforementioned experiments that measured only twitch potentiation, which may have a different mechanism (i.e.: RLC phosphorylation) than reflex potentiation, the timing of reflex PAP relates temporally to when PAP has been reported with dynamic movement (Chiu et al. 2003; Crewther et al. 2011; Hamada et al. 2000a; Young et al. 1998; Miyamoto et al. 2011; Mitchell and Sale 2011; Esformes et al. 2010; Judge et al. 2010; McBride et al. 2005; Tsimahidis et al. 2010; Weber et al. 2008; Kilduff et al. 2008).

Statement of the Problem

In the presence of a non-fatiguing MVIC (i.e., duration $\leq$ 10 seconds) the amount of twitch potentiation in response to a maximal muscle twitch (i.e.: an electrical stimulation that elicits a maximal M-wave, Mmax) is highest immediately after the conditioning activity (Folland et al. 2008; Stuart et al. 1988; Tubman et al. 1996; Vandervoort et al. 1983; Hamada et al. 2000b). However, postactivation twitch potentiation is a consequence of altered actin-myosin cross-bridge kinetics, and possibly increased stimulus efficacy (i.e.: sodium-potassium pump activity), and fails to account for the contribution of nervous system mediated reflex potentiation to the overall PAP response (Hamada et al. 2000b; Tillin and Bishop 2009; Folland et al. 2008; Klakowicz et al. 2006; Hicks et al. 1989). The mechanical isometric torque associated with an elicited maximal H-wave (Hmax) combines the contractile and reflex contributions to PAP, and represents the overall PAP response (Folland et al. 2008; Hodgson et al. 2005). RLC phosphorylation is highest immediately after a MVC, and declines steadily over an approximately 10 minute time period, even though overall PAP can last much longer

(Manning and Stull 1979, 1982; Moore and Stull 1984; Houston and Grange 1990; Zhi et al. 2005; Folland et al. 2008; Kitago et al. 2004). On the other hand, PAD has been shown to last for several minutes following a MVIC as short as 10 seconds in duration, before being potentiated for up to 18 minutes post-MVIC (Folland et al. 2008; Gullich and Schmidtbleicher 1996; Trimble and Harp 1998). The magnitude and timing of the myogenic and neurogenic contributions to the overall PAP response for a long duration following a non-fatiguing MVIC have yet to be described.

Purpose

The overall purpose of this dissertation was to explain the relationship between myogenic and neurogenic factors contributing to overall PAP following a conditioning activity. This included determining the time course and magnitude of these factors. The H-wave and its associated isometric mechanical torque are measures of motor neuron pool excitability, and the combined contractile and neurological statuses of the neuromuscular complex, respectively (Folland et al. 2008; Corrie and Hardin 1964; Kitago et al. 2004; Moritani and Shibata 1994; Zehr 2002). Torque potentiation resulting from RLC phosphorylation in response to a muscle twitch producing a maximal M-wave may be the prominent mechanism behind PAP shortly after a conditioning activity, however during volitional movement this may not be enough to overcome the reduced acute motor unit recruitment resulting from the conditioning activity. This may explain why PAP with volitional dynamic movement is not apparent for several minutes after conditioning activities. Further, the nervous system may be primarily, or solely, responsible for PAP beyond approximately 10 minutes after the conditioning activity due

to RLC phosphorylation returning to near baseline levels (Houston and Grange 1990). To test the association between RLC phosphorylation dependent twitch potentiation and neural factors, one condition of this investigation involved a LFF protocol prior to a MVIC. RLC phosphorylation is dependent on the intracellular calcium concentration of muscle (Blumenthal and Stull 1980; Bowman et al. 1992; Klug et al. 1982). Theoretically, by reducing the calcium concentration the amount of RLC phosphorylation will be reduced (Tubman et al. 1997; MacIntosh et al. 1993). The LFF protocol sought to reduce the amount of RLC phosphorylation by reducing the amount of calcium released from the sarcoplasmic reticulum, and thus the intracellular calcium concentration (Allen et al. 2008; Bruton et al. 1998; Westerblad et al. 1993).

Significance of the Study

This study will lead to a further understanding of the myogenic and neurogenic contributions to PAP. If practitioners have a better understanding of these mechanisms and the timing of these mechanisms that have been suggested to contribute to PAP, acute utilizations of PAP (i.e.: performing a conditioning activity before a ballistic athletic performance) and training programs utilizing PAP (i.e.: complex training) can be designed more effectively (Hodgson et al. 2005; Tillin and Bishop 2009; Robbins 2005; Chiu et al. 2003; Docherty et al. 2004). Acute athletic performance may be improved with the utilization of an appropriate conditioning activity. A more effective utilization of complex training in physical training programs may not only improve performance, but may also decrease the risk of incurring musculoskeletal injury as a result of

preferential chronic adaptations of the musculoskeletal system (Hubscher et al. 2010; Staron et al. 1994; Staron et al. 1991; Cornu et al. 1997).

Hypotheses

The experimental hypothesis for this investigation was that differences would be observed between muscle twitch potentiation torque and reflex potentiation (e.g.: H-wave amplitude) over the time period that overall PAP is evident. Specifically, it was hypothesized that:

1) Twitch potentiation torque and RFD would be highest immediately after the 10 second MVIC conditioning activity during the non-fatigue condition, and decline in an exponential manner until testing concluded 20 minutes post-MVIC.
2) H-wave amplitude (normalized to M-wave amplitude) would be depressed for several minutes post-MVIC during both experimental conditions.
3) Overall PAP torque (e.g.: H-wave associated twitch torque) would be closely related to both twitch potentiation torque and the H-wave. However, this relationship would be time dependent. Maximal twitch torque would relate closely to overall PAP for several minutes beginning immediately post-MVIC. H-wave would be closely related to overall PAP beginning several minutes post-MVIC until the termination of testing.
4) Overall PAP RFD (i.e.: PAP at Hmax) would show the same temporal profile as torque at Hmax, being suppressed for several minutes post-MVIC before increasing.

5) Measures of potentiation would be more pronounced in the gastrocnemius than the soleus because of the greater proportion of type II muscle fibers.
6) Both RP and TP would significantly contribute to overall PAP.

Delimitations

This study was delimited to 15 resistance trained persons between the ages of 18 and 35 years residing in central Kentucky. PAP is more apparent in trained persons (Chiu et al. 2003; Jo et al. 2010; Gullich and Schmidtbleicher 1996; Rixon et al. 2007; Smith and Fry 2007). Therefore, it was required that subjects were currently engaged in a whole-body resistance training program at least three times weekly, and that they had been engaged in this program continuously for at least one year (Wallace et al. 2008). As a performance measure of training status, male participants confirmed that they are able to back squat ≥1.5 times their bodyweight, and female subjects ≥1.0 times their bodyweight (Chiu et al. 2003). Persons were excluded from participation if they answered one or more questions "yes" on the PAR-Q fitness questionnaire (Appendix B). Prospective subjects were also excluded if they, at or prior to the time of testing, had a neurological disorder or major musculoskeletal lower extremity injury.

The type of contractions in the protocol were delimited to isometric contractions because our primary measure, the H-reflex, is most reliable with this type of contraction (Zehr 2002). We also delimited the MVIC within our protocol to 10 seconds because this has been shown to produce muscle potentiation but not muscle fatigue (Vandervoort et al. 1983).

Assumptions

Assumptions that were made in conducting this project include:

1) Subjects were truthful regarding their training and health history.
2) Subjects abided by not participating in rigorous physical activity, or consuming alcohol or caffeine, for 24 hours prior to each testing session.

Limitations

Limitations involved with this dissertation include:

1) Some subjects exerted force from muscles other than just their plantarflexors during the MVICs. However, in these instances it was apparent this was in addition to, not in place of, maximal plantarflexion effort.
2) The position of the stimulating and EMG electrodes was marked on subjects' skin to help ensure consistent placement during subsequent testing days. However, there is the possibility that small inter-day differences in placement occurred.
3) Some subjects moved their lower leg or foot slightly on occasion during the testing sessions. This could have minimally changed the contractile performance of the muscles during testing by inducing a small amount of potentiation or fatigue.

Definition of Terms

The following terms used in this dissertation are defined below:

Complex training: Complex training is accomplished by the execution of a maximal or near maximal voluntary effort designed to induce PAP, followed several minutes later by plyometric exercise (Young et al. 1998; Docherty et al. 2004). Usually

the voluntary effort is a weight training exercise, such as a barbell back squat. Theoretically, increased force or RFD will be obtained during the plyometric performance as a consequence of the voluntary effort, resulting in enhanced physiological adaptations beneficial to athletic performance.

Conditioning activity: A conditioning activity involves muscle contractions performed for the purpose of inducing PAP. It may be induced via electrical stimulations, or the result of volitional effort. Common forms of volitional conditioning activities are MVICs or resistance training exercises, most commonly the barbell back squat.

Hoffmann reflex (H-reflex): Electrically stimulating a muscle elicits a response that can be measured by electromyography (EMG). One of these responses is the Hoffmann reflex, or H-reflex. It arises from monosynaptic excitation of motor neurons via Ia afferent sensory nerve fibers from muscle spindles (Stein and Thompson 2006). The EMG signal recorded as a result of this reflex is termed the H-wave.

Hmax: The maximum H-wave amplitude response to electrical stimuli is termed Hmax. This is elicited in response to an electrical stimulation of relatively low intensity. Hmax amplitude, normalized to Mmax, is an estimate of the number of motor units that is capable of being activated in a given physiological state (Palmieri et al. 2004). This representation of PAP is the part of overall PAP that can be contributed to neurogenic mechanisms (Folland et al. 2008).

Low frequency fatigue (LFF): Fatigue is associated with a reduction in contractile force following prior muscle contractions. A reduction in muscle force in response to

low stimulation frequencies, most notably a twitch, following previous volitional or electrically induced contractions is termed LFF (Rassier and Macintosh 2000).

Mmax: The M-wave is the direct muscle response to an electrical stimulation, as measured by EMG. Mmax is the highest amplitude M-wave that can be elicited under the given conditions. It occurs in response to a maximal or supramaximal electrical twitch designed to recruit all of the motor units associated with the particular nerve being stimulated.

Overall PAP: Overall PAP is a result of the myogenic and neurogenic contributions to PAP. It is measured as the mechanical torque generated from an electrical twitch at Hmax intensity (Folland et al. 2008).

Phosphorylation: The process by which an enzyme transfers a phosphate group from ATP to another protein molecule is termed phosphorylation (Grange et al. 1993). In this dissertation MLCK is the transferring enzyme of interest, and it phosphorylates the RLCs of myosin.

Postactivation potentiation (PAP): When a muscle is activated via a conditioning activity it can acutely produce more force than would be possible in the absence of the activity. The difference between the amount of force with, and without, the activity is termed PAP.

Rate of force development (RFD): RFD refers to how quickly force increases. In this dissertation the origin of force will be the neuromuscular system.

Mathematically: $RFD = \frac{\Delta Force}{\Delta Time}$

Twitch potentiation: A conditioning activity can cause an increase in muscular force in response to a muscle twitch, compared to the force value before the conditioning

activity. The difference between these twitches is the amount of twitch potentiation. This representation of PAP is the part of overall PAP that can be contributed to myogenic mechanisms (Folland et al. 2008).

Twitch torque: The mechanical torque generated by muscle in response to an electrically evoked twitch of a given intensity is the twitch torque. In this project torques were measured at Hmax and supramaximal Mmax (Mmax plus 20%) intensities.

Chapter 2: Review of Literature

Introduction

The purpose of this study is to determine the magnitude and timing of myogenic and neurogenic factors to the overall PAP response. This chapter will analyze the literature related to PAP. Section 1 will review the literature related to the two main possible practical applications of PAP: 1) performing a MVC prior to an athletic performance, and 2) complex training as a training modality. Section 2 will include a discussion of the factors known to influence PAP. These factors include age, fiber type, muscle length, training status, and strength level. Lastly, Section 3 will review the methodologies of studies that have investigated PAP. Methods of investigating both muscular and neural PAP will be reviewed.

Section 1: Practical uses of PAP in sports and training

PAP and posttetanic potentiation (PTP) refers to phenomenon where muscle performance characteristics are enhanced as a result of recent contractile history. Muscle contraction(s) are required to elicit this acute enhanced muscle performance. These contractions are referred to as a conditioning activity. When the conditioning activity is elicited by evoked high frequency electrical stimulation it is termed PTP (Tillin and Bishop 2009; Sale 2002). The conditioning activity can also be in the form of a maximal or near-maximal voluntary contraction, termed PAP (Tillin and Bishop 2009; Sale 2002) . For clarity, in this section PAP will refer to both voluntary and involuntarily stimulated conditioning activities.

It has been reported that throwing, sprinting, and jumping have force production durations between 100 and 300 ms (Tidow 1990). Movements such as these where force is required to be produced in 250 ms or less have been termed "explosive" movements (Haff et al. 1997). It has been suggested that RFD is the most important factor in athletic feats that have a force production duration between 100 and 300 ms, and maximum force production is the most important factor in performance for movements that take longer than 300 ms (Wilson et al. 1995). The performance of both "explosive" and non-explosive volitional movements may be enhanced by a conditioning activity, as they have been shown to potentiate both peak force and RFD during dynamic activity (French et al. 2003).

Although muscle performance has been shown to be enhanced by a conditioning activity, the role that PAP may have in enhancing sport performance is not well understood. There are two principal theories regarding how PAP may be utilized to enhance performance. The first is that a conditioning activity performed before a sporting event requiring high force or RFD may acutely enhance performance in that event. MVCs performed prior to activity have been shown to acutely increase jumping, running, throwing, and bench press performance (Gullich and Schmidtbleicher 1996; Judge et al. 2010; Requena et al. 2005b; Chiu et al. 2003; Crewther et al. 2011; Esformes et al. 2010; Hamada et al. 2000a; Kilduff et al. 2008; Mitchell and Sale 2011; Miyamoto et al. 2011; Morana and Perrey 2009; Till and Cooke 2009; Tsimahidis et al. 2010; Young et al. 1998). To utilize PAP in this manner, an athlete would perform a conditioning activity (i.e.: a MVC approximately 10 seconds in duration utilizing the primary movers involved in the sporting activity) several minutes prior to their

competitive performance. The increased ability to produce force and RFD could lead to a better physical performance. The second way that a conditioning activity may improve athletic performance is through the use of complex training (Figure 2.1) (Docherty and Hodgson 2007; Docherty et al. 2004; Tsimahidis et al. 2010). Complex training involves performing a weight training movement, such as a back squat, using a relatively heavy load (i.e.: 1-5 repetition maximum (RM)), followed by the performance of a plyometric exercise. Taken together, the weight training and plyometric exercises are called a "complex pair." According to the theory, greater force and RFD are elicited during the plyometric exercise after performing the weight training activity than what would be achieved in absence of the conditioning activity (Docherty and Hodgson 2007; Docherty et al. 2004). The acute enhancement of these performance variables over many sessions may result in superior long-term training adaptations.

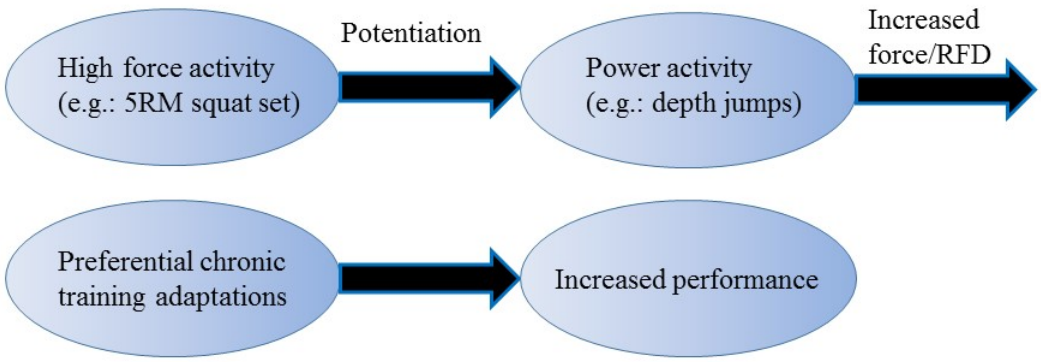

Figure 2.1: Theoretical construct of complex training.

Although the theory related to complex training is not new, to date there is only one study that has investigated the chronic adaptations to this type of training. Tsimahidis et al. (2010) divided 26 junior basketball players into 2 groups of 13

participants. The control group trained technical skills only, while the experimental group completed a resistance and sprint training program, for 10 weeks. The experimental group performed 5 sets of half squats at their 8RM for the first 5 weeks; during the second 5 weeks the intensity was increased to their 5RM. Ninety seconds after each set subjects maximally sprinted a distance of 30 meters. Ninety seconds after the completion of the sprint they performed the next set of back squats. Jump performance and 1RM strength were significantly increased in the experimental but not control group at 5 and 10 weeks after training began, while 0-10 meter and 0-30 meter sprint times were significantly decreased at these time points in the experimental, but not control, group. This study supports the notion that complex training can result in preferential long-term training results. However, because this study used sprinting as the second component of their complex pair it is difficult to relate these results to the more commonly utilized form of complex training that uses plyometrics as the second component of the complex pair. Additionally, while this study supports that complex training can improve performance, the lack of a second experimental group that performed the same exercise protocol, but not as part of a complex pair, makes it impossible to determine the contribution of complex training itself to the results. Further research needs to be conducted to determine the long-term practical results of complex training as compared to similarly prescribed non-complex paired exercises.

Section 2: Factors known to influence PAP

Different characteristics of the human subjects involved in experiments can influence PAP results. These factors include age, fiber type, muscle length, training

status, and strength level. This section will discuss the relationship between these subject characteristics and PAP.

Age:

There are few studies that have investigated PAP with ageing. One study compared myogenic PAP of the ankle plantarflexors between 11 year old pre-pubertal boys, 16 year old post-pubertal boys, and young men (ages 19-23 years) (Paasuke et al. 2000). Subjects performed a 5 second MVIC, after which twitch contractile properties were recorded and compared to pre-MVIC values. It was reported that pre-pubertal boys had lower peak twitch forces, but similar potentiation, than post-pubertal boys and young men. There were no differences between the post-pubescent boys and young men in peak twitch force or potentiation. No differences in body mass normalized muscle contractile performance characteristics were found between groups. The authors suggested that the additional muscle mass gained during puberty increases the ability to produce force, but that the ability for PAP is not age-related within the age ranges tested. This suggests that even children may benefit from methodologies designed to induce PAP.

Other studies have compared myogenic PAP between younger and older adults and found that PAP is significantly influenced by age. Potentiation still occurs with increased age, however, the magnitude is reduced (Baudry et al. 2005; Paasuke et al. 2002a; Paasuke et al. 2002b; Petrella et al. 1989). Lower limb twitch potentiation force, time to peak force, and RFD all are present in aged individuals, but have been reported to increase more in younger individuals; while potentiated responses for contraction time and half relaxation time are reduced more in younger compared to older adults (Baudry et al. 2005; Petrella et al. 1989; Paasuke et al. 2002a; Paasuke et al. 2002b). The decline in

muscle contractile performance with ageing in response to a condition activity also appears to occur similarly regardless of gender (Paasuke et al. 2002b; Baudry et al. 2005). The decline may be due to sarcopenia, specifically the reduction in size and number of type II fibers that occurs with ageing (Petrella et al. 1989).

Fiber type:

Fiber type appears to have a meaningful role in the PAP response in both rats and humans. Moore and Stull (1984) studied potentiation of the rat gastrocnemius and soleus muscles following neural stimulation at different frequencies. They reported that the fast-twitch gastrocnemius was phosphorylated over the range of evoked stimulation frequencies (0.5-100 Hz). In contrast, only stimulation frequencies of 30 Hz or more caused RLC phosphorylation in the slow-twitch soleus muscle. Fast-twitch muscle also had more than double the amount of RLC phosphorylation, and the rate of dephosphorylation was four times slower than in slow-twitch muscle. Due to the greater amount of RLC phosphorylation and slower rate of dephosphorylation, the authors concluded that PAP is likely to be greater in muscles that contain more fast-twitch fibers.

One study reported that no RLC phosphorylation occurs in slow-twitch human muscle fibers of the vastus lateralis following a MVC of unspecified duration (Houston et al. 1985). According to these results any PAP associated with RLC phosphorylation must be due exclusively to fast-twitch muscle fibers being phosphorylated. However, subsequent studies by the same research group using different muscle analysis techniques have shown that slow-twitch muscle is phosphorylated in humans after a MVC (Houston and Grange 1990, 1991; Houston et al. 1987; Stuart et al. 1988). Slow-twitch fibers do appear to have some role in PAP.

Significant negative correlations ($r \geq -.71$) have been found between muscle contraction time and twitch potentiation in whole human muscle (Belanger and Mccomas 1985; Hamada et al. 2000b). That is, muscles that had faster contraction times exhibited greater twitch potentiation. This suggests that type II fibers are strongly associated with twitch potentiation since muscles with faster contraction times have been shown to have more type II fibers (Belanger et al. 1983). Hamada et al. (2000b) examined knee extensor twitch contraction time to peak torque in 20 young men before and after a 10 second MVIC. They found that pre-MVIC contraction time to peak torque had a significant negative correlation ($r = -.73$) to post-MVIC twitch time to peak torque. They then performed biopsies of the vastus lateralis on the four subjects who had the longest and shortest pre-MVIC twitch times to peak torque. Subjects who exhibited the shortest contraction times had a much higher percentage of type II fibers than those who exhibited the longest contraction times (72 ± 9 vs. $39 \pm 7\%$). This is similar to what was reported in a subsequent study by the same author (Hamada et al. 2003). It appears that type II fibers contribute more to PAP as evoked by supramaximal stimulations than do type I fibers. This also appears to be the case with reflex potentiation (Gullich and Schmidtbleicher 1996). However, as the studies that measured RLC phosphorylation show, type I fibers still probably play a role in the potentiated response. It is likely that fast-twitch fibers are more responsible for PAP because of their larger size and number of cross-bridge attachments.

Muscle length:

The influence of muscle length on PAP has been investigated in both human and rodent skeletal muscle (Stuart et al. 1988; Rassier and MacIntosh 2002; Rassier and

Tubman 1997; Place et al. 2005; Rassier 2000). The two rodent studies, both conducted by Rassier and colleagues (1997; 2000), measured staircase potentiation elicited by 10 seconds of 10 Hz stimulation. Staircase potentiation is defined as the enhancement of twitch torque following low frequency potentiation (Rassier and MacIntosh 2002). Both of these studies reported that potentiation was greater at short vs. long muscle lengths. Rassier and Tubman (1997) studied *in situ* rat gastrocnemius muscle at optimal length, and ± 10% optimal length. The percent of twitch torque was increased by 118.5 ± 7.8, 63.1 ± 3.9, and 45.6 ± 4.1 (mean ± SE) for optimal length minus 10%, optimal length, and optimal length plus 10%, respectively. The authors also reported that these differences were in spite of RLC phosphorylation being similar between the three conditions, signifying that one or more other mechanisms are also responsible for PAP. In *in vitro* mouse extensor digitorum longus muscle Rassier and MacIntosh (2002) reported a negative correlation between muscle length and PAP. That is, there was more potentiation at shorter muscle lengths than at longer muscle lengths. They found this in not only staircase, but also posttetanic potentiation. Linear regression yielded R^2 values of 0.74 and 0.80 for staircase and posttetanic potentiation, respectively.

In human skeletal muscle the general findings are similar to the aforementioned rodent experiments. That is, PAP is greater at short vs. long muscle lengths. After a 10 second MVIC of the knee extensors in 14 healthy physical education students, Rassier (2000) reported that the amount of PAP was 68 ± 5%, 47 ± 2%, and 39 ± 4% at knee flexion angles of 30, 60, and 90 degrees (0 degrees equated to full extension), respectively. Stuart et al. (1988) also reported that twitch potentiation after a 10 second MVIC of the quadriceps was greater at a 90 degree than a 135 degree knee angle (180

degrees equated to full extension). In a study designed to compare central and peripheral mechanisms of fatigue, subjects performed an isometric contraction of the quadriceps for as long as possible while maintaining 20% MVC force (Place et al. 2005). After performing three 5 second MVICs, subjects were able to maintain 20% force for 974 ± 457 seconds vs. 398 ± 144 seconds at the short (35 degree) vs. long (75 degree) muscle lengths (0 degrees equated to full extension). Additionally, they reported that the amount of twitch potentiation after the fatiguing contraction was only present at the short, and not long, muscle length (31.8 ± 17.6% vs. 6.4 ± 1.3%). In both animals and humans PAP is more prominent at shorter muscle lengths.

Training status:

PAP appears to occur regardless of training status when measured by muscle twitch characteristics (Paasuke et al. 2002c; Paasuke et al. 2007; Hamada et al. 2000a). The magnitude of twitch PAP appears to be higher, however, in more trained individuals. Paasuke and colleagues conducted two studies comparing twitch peak torque in power-trained and untrained women (Paasuke et al. 2002c; Paasuke et al. 2007). In plantarflexors power-trained female athletes exhibited greater potentiation of twitch force than untrained college-age women (Paasuke et al. 2002c). In another study, power-trained athletes exhibited a greater potentiation of force, reduction in contraction time, and increase in RFD in knee extensors, despite pre-MVIC twitch contraction time and RFD being similar between the trained and untrained groups (Paasuke et al. 2007). PAP may manifest itself differently in response to distinct modes of training due to training-specific neural adaptations. It may also be more prominent in muscles that are trained more regularly or at a greater intensity (Paasuke et al. 2007; Hamada et al. 2000a).

Smith and Fry (2007) measured RLC phosphorylation via muscle biopsies in 11 recreationally active men with at least 1 year of weight training experience after a 10 second dynamic MVC of the knee extensors. They found that the 7 positive responders increased phosphorylation by an average of 23%, while 4 subjects were negative responders and did not experience phosphorylation in response to the MVC. Although all participants had a minimum level of training experience, the authors hypothesized that more trained individuals have a greater ability to induce PAP. Two other studies compared PAP in trained vs. untrained individuals. Chiu et al. (2003) compared pre and post-conditioning stimulus (5 sets of 1 repetition back squats at 90% 1RM) vertical jump parameters in explosive sport athletes and recreationally trained subjects. The authors reported that force and power were potentiated more in the athletes group than the recreationally trained group. More recently, Berning et al. (2010) reported similar results following a 3 second isometric leg press in resistance trained and untrained men. The authors found that potentiation, as defined by an increase in vertical jump height following the conditioning activity, was not present in the untrained group, but was significantly higher in the trained group. Similar results have been found with potentiation of the H-reflex. Trimble and Harp (1998) had difficulty finding a potentiated H-reflex after their conditioning activity in several subjects in their sample of untrained college students. In contrast, Gullich and Schmidtbleicher (1996) did not report difficulties eliciting the H-reflex from the untrained participants in their study. They also reported that potentiation of the H-reflex was greater, and longer lasting, in their sample of anaerobically trained athletes. Similar findings have been found with twitch potentiation byPaasuke et al. (2007).

Strength level:

The effect of strength level on PAP is more equivocal (Table 2.1). No difference in vertical jump height after a 5 second MVIC in the leg press was observed between power track and field athletes (n=8), bodybuilders (n=7), and physically active subjects (n=8) (Batista et al. 2011). The strength level of male soccer players was not related to their sprint and jump performance following several different types of conditioning trials (Till and Cooke 2009). Both of these authors mention that their studies show an uncoupling of strength and PAP. One additional study reported that strength ratio did not predict vertical jump increases following a conditioning activity (Magnus et al. 2006). The results of this study should be viewed with caution, since recreational weight trainers were used as subjects, and 1 squat repetition at 90% 1RM was used as the conditioning stimulus which may not have provided a sufficient stimulus for potentiation.

Other studies have found a positive relationship between strength level and PAP (Gourgoulis et al. 2003; Jensen and Ebben 2003; Jo et al. 2010; Kilduff et al. 2008; Rixon et al. 2007; Young et al. 1998). One study used the Wingate test as a measure of muscular power (Jo et al. 2010). After 1 set of 5 repetitions of the back squat at their 5RM, subjects performed a Wingate test after resting for 5, 10, 15, or 20 minutes. The authors found insignificant increases in Wingate test power characteristics after the squat set. In light of their insignificant findings, the authors wanted to determine if strength was a factor in power performance. They reported a high and significant correlation ($r = .77$, $p < .05$) between relative 1RM back squat strength and the time course of PAP in the Wingate test. That is, stronger subjects showed potentiation of power at 5 minutes, while weaker subjects took longer to show potentiation. A more homogenous group may have

led to statistically significant findings for power production because of similarly timed potentiation.

A more common experimental design to investigate functional PAP has been to study vertical jump characteristics before and after back squat protocols (Gourgoulis et al. 2003; Jensen and Ebben 2003; Kilduff et al. 2008; Rixon et al. 2007; Young et al. 1998). Stronger individuals have been shown to increase their vertical jump height more than weaker individuals after a conditioning activity, regardless of sex (Rixon et al. 2007). Young et al. (1998) reported a significant relationship between 5RM back squat strength and vertical jump height increases after a set of back squats, while Jensen and Ebben (2003) found a similar, but statistically insignificant, relationship. Kilduff et al. (2008) reported a significantly positive correlation ($r = .49$, $p = .03$) between 3 RM back squat strength and vertical jump performance in professional rugby players 8 minutes after 3 sets of 3 repetitions of back squats at 87% 1RM. Mean vertical jump height increased 4.9%, indicating that jump height would have been increased even more in the stronger subjects. Another study found that jump height improved by 2.39% in 20 physically active men 5 minutes after 2 sets of 5 repetitions of back squats (Gourgoulis et al. 2003). After obtaining mean values of the entire sample, the authors divided their sample into two groups according to their 1RM back squat strength. They discovered that the high strength group increased their vertical jump height by 4.01%, after the back squat conditioning activity, while the low strength group only increased their vertical jump height by .42%. Both of these authors suggest stronger individuals may be able to activate more motor units than weaker individuals in response to a conditioning activity, which would result in a more potentiated H-reflex. Gourgoulis et al. (2003) also

hypothesized that additional neurological adaptations, such as the ability for motor units to obtain higher activation frequencies and greater synchronization, may allow for greater PAP in stronger individuals.

Table 2.1: Summary of studies involving subjects of different training and strength statuses on PAP.

First Author (Yr.)	Subjects	Potentiating Method	Assessment	Results
Batista (2011)	All males	1 or 3 five second leg press	Countermovement	No differences
	Power track and field (n=8)	MVCs	jumps 4 minutes	between groups
	Bodybuilders (n=7)		after MVCs	
	Physically active (n=8)			
Berning (2010)	All males	3 second isometric squat	Countermovement	Trained sig. improvement
	Resistance trained (n=13)	MVC at 150% 1RM	jumps 4 minutes	in jump, no improvement
	Untrained (n=8)		after MVC	in untrained
Chiu (2003)	12 men, 12 women	1 set of 5 repetitions back	Jump squats	Only athletes group
	Explosive atheltes (n=7)	squat at 90% 1RM		showed potentiation
	Recreationally trained (n=17)			
Gourgoulis (2003)	Physically active men	5 sets of 2 repetitions of half	Countermovement	Stronger subjects
	(n=20)	squats at 20%, 40%, 60%,	jumps immediately	improved jump height
		80%, and 90% 1RM	after squats	8x more than weaker
Guellich (1996)	All males	Three 5 second isometric	Plantarflexor	Athletes reflex potentiated
	Anaerobic athletes (n=10)	MVCs	H-reflex	and lasting to 12 min.;
	Sport students (n=7)			students not potentiated
Hamada (2000)	All males	10 second isometric MVC of	Peak torque	PAP greater in regularly
	Triathletes (n=10)	elbow flexors, 10 second	from electrical	trained muscles in all
	Distance runners (n=10)	isometric MVC of plantar-	twitch	training groups
	Active controls (n=10)	flexors		
	Sedentary controls (n=10)			
Jensen (2003)	10 females, 10 males	1 set of 5 repetitions back	Countermovement	No gender or strength
	NCAA Div. 1 atheltes	squat at 5RM	jumps 10 seconds,	differences; jump height
	in power sports		1, 2, 3, and 4	depressed at 10 seconds
			minutes after squats	and recovered slowly

Table 2.1: (cont.)

First Author (Yr.)	Subjects	Potentiating Method	Assessment	Results
Jo (2010)	All males	1 set of 5 repetitions back	30 second	Timing of potentiation
	Resistance trained (n=12)	squat at 85% 1RM	Wingate	strength dependent
Kilduff (2008)	All males	3 sets of 3 repetitions in at	Countermovement	Significant positive
	Professional rugby players	87% 1RM (back squat)	jumps 15 seconds,	correlation between 3RM
	(n=20)		5, 8, 12, and 16	strength and potentiation
			minutes after squats	
Mangus (2006)	All males	1 set of 1 repetition of half or	Countermovement	No strength effect on
	Weightlifters (n=10)	quarter squats at 90% 1RM	jumps 3 minutes	potentiation
			after squats	
Paasuke (2002)	All females	5 second isometric MVC of	Peak torque	Power trained showed
	Power trained (n=11)	plantarflexors	from electrical	greater potentiation
	Recreationally trained (n=14)		twitch	
Paasuke (2007)	All females	10 second isometric MVC of	Peak torque	Power trained showed
	Power trained (n=12)	knee extensors	from electrical	greater potentiation
	Endurance trained (n=12)		twitch	
	Untrained (n=12)			
Rixon (2007)	15 males, 15 females	Three 3 second isometric	Countermovement	Experienced showed more
	(20 previous weightlifting	MVC squats	jumps 3 minutes	potentiation
	experience, 10 no experience)		after MVCs	
Smith (2007)	All males	10 second isometric MVC of	Knee extension	Potentiation not shown
	Recreationally trained (n=12)	knee extensors	kinetics	

Table 2.1: (cont.)				
First Author (Yr.)	**Subjects**	**Potentiating Method**	**Assessment**	**Results**
Till (2009)	All males	Three 3 second isometric	Spring and counter-	No strength effect on
	Professional soccer players	MVC knee extensors	movement jump	potentiation
	(n=12)			
Young (1998)	All males	One set of 5 repetitions at	Loaded counter-	Significant positive
	Resistance trained (n=10)	5RM load (back squat)	movement jump	correlation between 5RM
				strength and potentiation

Section 3: Measurement of PAP

There are two primary methods that have been used to quantify the effect of a conditioning activity on subsequent non-volitional muscle and neural performance. The first method is measuring muscle twitch force after a conditioning activity, which determines the muscle contribution to potentiation. The second method measures reflex potentiation quantified by the H-reflex peak-to-peak amplitude. This section will review these two methods in detail.

The most common method of measuring PAP is by evoking a supramaximal electrical stimulus to a nerve, or muscle directly, that has just been contracted during a conditioning activity. In human experiments usually the conditioning activity is in the form of a MVIC, although high frequency electrical stimulation has been used (Baudry and Duchateau 2007; Binder-Macleod et al. 2002; Requena et al. 2005a; O'Leary et al. 1997). In animal experiments the conditioning activity takes the form of a high frequency tetanic stimulation, or less often a lower frequency stimulation that results in unfused tetanus (MacIntosh et al. 1993; MacIntosh et al. 2008; Moore and Stull 1984; Persechini et al. 1985; Tubman et al. 1996; Tubman et al. 1997; Zhi et al. 2005).

Following the conditioning activity subjects are supramaximally twitched by an electrical stimulus. The stimulus intensity required to elicit maximum muscle force or a maximum compound muscle action potential (M-wave) is obtained prior to the conditioning activity. This stimulation intensity has been identified as the minimum intensity which evokes a maximum torque response (Hamada et al. 2000a; Hamada et al. 2000b; Paasuke et al. 2007; Requena et al. 2005a; Requena et al. 2008). Other authors have determined it as being the intensity at which neither the M-wave or its associated

twitch torque increased in response to an increased stimulation intensity (Baudry and Duchateau 2007; Baudry et al. 2005, 2008; Folland et al. 2008; Hamada et al. 2003; O'Leary et al. 1998; Place et al. 2005; O'Leary et al. 1997; Belanger and Mccomas 1985). For the evoked twitches after the conditioning activity an intensity of approximately 120% of what was determined to be maximal is typically used (Folland et al. 2008; Hamada et al. 2000a; Hamada et al. 2003; O'Leary et al. 1997; Paasuke et al. 2007; Requena et al. 2011; Requena et al. 2005a; Requena et al. 2008; Baudry et al. 2008). However, some authors have used the maximum stimulation intensity (Behm et al. 2004; Hamada et al. 2000b; Houston et al. 1985; Stuart et al. 1988; Houston and Grange 1991). The first twitch is usually conducted in close temporal proximity to the MVC, with subsequent twitches evoked intermittently over the time period the researchers investigated the potentiated response. The first twitch has been evoked as quickly as 1 second after the MVC, although 5-20 seconds or more is commonplace (Gossen and Sale 2000; Paasuke et al. 2000).

The experimental set-up is relatively consistent among studies that have investigated knee extensor twitch potentiation in humans. Typically the subject is positioned in a supportive chair with relative hip and knee angles of 100° and 90° respectively (Hamada et al. 2000b, 2003; Mitchell and Sale 2011; Alway et al. 1987; Paasuke et al. 2007; Requena et al. 2011; Requena et al. 2005a; Requena et al. 2008). Studies that have investigated the effect of muscle length on twitch potentiation have used other knee angles (Place et al. 2005; Rassier 2000; Stuart et al. 1988). A cuff is placed around the ankle to record knee extension torque. Strain gauge force transducers and isokinetic dynamometers have both been used to record twitch force (Folland et al.

2008; French et al. 2003; Gossen and Sale 2000; Paasuke et al. 2007; Place et al. 2005; Rassier 2000; Requena et al. 2011; Requena et al. 2005a; Requena et al. 2008; Stuart et al. 1988; Houston et al. 1985). One research team has designed and built a custom isometric dynamometer apparatus to measure knee extension torque in response to stimulations in their studies (Hamada et al. 2000b, 2003; Mitchell and Sale 2011).

The H-reflex is another tool that has been utilized by researchers to study the effects of contractile history on neuromuscular performance following a conditioning activity. It measures the efficacy of synaptic transmission at a given time, and thus has been widely used as a measure of α motoneuron excitability (Palmieri et al. 2004). Once thought to be a purely monosynaptic reflex, it is now believed to be influenced by presynaptic inhibition during dynamic movements (Hodgson et al. 2005). With isometric muscle actions it is still considered a monosynaptic reflex, however (Zehr 2002). The H-reflex is analogous to the spinal stretch reflex; only the spinal stretch reflex is elicited after a muscle stretch, while the H-reflex is elicited by an electrical stimulation.

The H-reflex has been most often studied in the soleus and quadriceps (Palmieri et al. 2004). Figure 2.2 depicts the process by which the H-reflex is produced. Briefly, a single indirect percutaneous electrical impulse to the relevant peripheral nerve is used to elicit the reflex. The muscle spindles are bypassed because of the involuntary nature of the stimulus. The reflex begins when a sufficiently intense electrical stimulus generates action potentials in Group 1a afferents. These action potentials are propagated to the spinal cord, where the afferent terminals will be depolarized (Palmieri et al. 2004). If the terminals are depolarized enough to cause neurotransmitter release at the Ia afferent-α motoneuron synapse, postsynaptic depolarization of the α motoneuron will occur (Zehr

2002). If the postsynaptic depolarization is above threshold an efferent action potential will be generated and cause neurotransmitter release across the neuromuscular junction, resulting in a muscle contraction. Because the stimulation is a single impulse, the response is a muscle twitch. It is important to note that many afferents and motor units associated with the stimulated nerve are involved. Thus, the measured EMG or force values are of the net muscle response to the stimulation. Increasing the intensity of the electrical stimulation will result in a progressively larger H-reflex until Hmax is achieved.

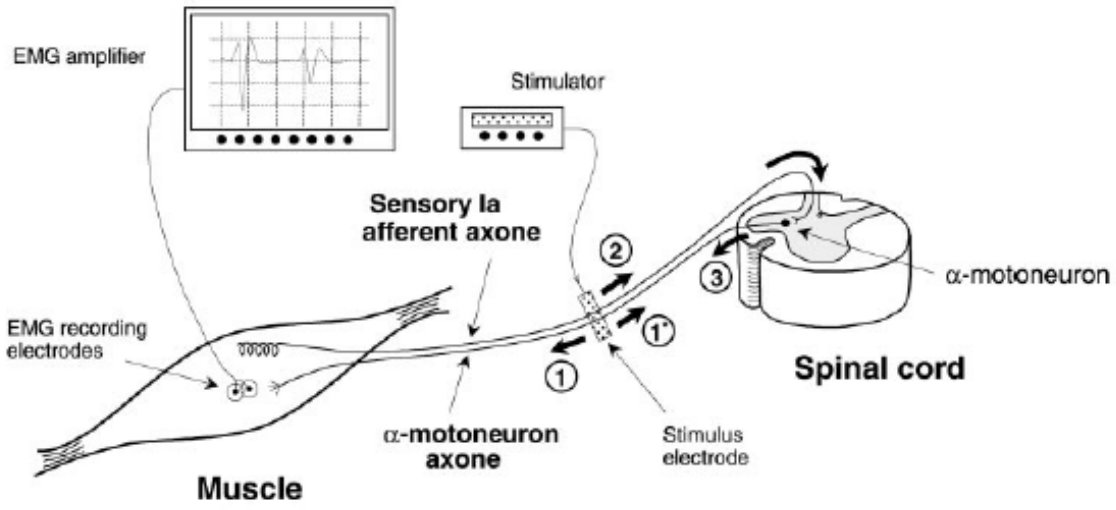

Figure 2.2: H-reflex pathway. See text for pathway details. Adapted from Aagaard et al. (2002); publisher does not require permission for use.

The experimental set-up for measuring the H-reflex in the quadriceps is similar to what was previously described for measuring twitch potentiation. The H-reflex associated muscle contraction is recorded using surface EMG on the muscle of interest.

The isometric force associated with the H-reflex muscle contraction can also be measured with a force transducer or isokinetic dynamometer. When EMG is used to quantify the reflex response the peak-to-peak amplitude is most often reported (Christie et al. 2004; Enoka et al. 1980; Folland et al. 2008; Gullich and Schmidtbleicher 1996; Kitago et al. 2004; Klakowicz et al. 2006; Palmieri et al. 2002; Trimble and Harp 1998). However, integrated EMG has shown a high correlation to the peak-to-peak value ($r \geq .93$) and has also been reported (Gollhofer et al. 1998). The H-wave is most often quantified by normalizing it to the Mmax peak-to-peak value (Palmieri et al. 2004). Mmax has been shown to be very reliable between days (ICC $\geq .96$) (Calder et al. 2005; Christie et al. 2004; Chen et al. 2010). The Hmax/Mmax ratio can be interpreted as the proportion of the entire motoneuron pool that is capable of being recruited at a given point in time, and is the preferred method of normalizing Hmax in studies where data are collected over multiple days (Palmieri et al. 2004).

Several studies have investigated the reliability of the H-reflex. The H-reflex at rest elicited by a stimulation intensity equivalent to 5% of that required to produce Mmax has been reported to have high reliability over 5 testing sessions (ICC = .85) (Christie et al. 2004). Chen et al. (2010) measured the test-retest reliability of the soleus H-reflex amplitude at rest. Two testing sessions were conducted on separate days within a one week time period. The authors reported Hmax ICCs of .75-.96 between days depending on ankle joint position (neutral, plantarflexed, or dorsiflexed). Studies have also reported reliability related to the Hmax/Mmax ratio. The aforementioned Chen et al. (2010) study found inter-day reliability of the Hmax/Mmax ratio to be high at rest (ICCs .48-.96). For both Hmax and Hmax/Mmax ratio, reliability was lowest when the ankle was in a

dorsiflexed position. Another study by Palmieri et al. (2002) measured Hmax and Hmax/Mmax reliability over two testing days in the soleus, peroneal, and tibialis anterior muscles while at rest. Intersession reliability for Hmax and Hmax/Mmax were ≥ .96 for the soleus and peroneal muscles, while the reliability was slightly lower for the tibialis anterior (ICC = .85 and .78 for Hmax and Hmax/Mmax, respectively). In resting quadriceps muscle Hmax/Mmax measured in the vastus medialis has been reported to have intersession reliability of .96 between days, and .91 over four weeks (Hopkins and Wagie 2003). Although it has been suggested that the H-reflex should be measured in muscles undergoing voluntary submaximal background muscle activity, these results show that the H-reflex is a reliable measure when measured at rest (Brinkworth et al. 2007). This coincides with the recent work of Grospretre and Martin (2012), who reported that the ascending and peak portions of the H-reflex stimulus response curve did not change when elicited at rest when compared with the stimulus response curve with background muscle contractions at an intensity of 50% MVIC.

Summary

This chapter discussed three main areas of literature related to PAP: 1) the two possible practical applications of PAP, 2) a review of the factors known to influence PAP, and 3) a review of the methodologies of studies that have investigated PAP. Sporting performance may be improved through acute utilizations of PAP, or through chronic utilizations (i.e.: complex training). A variety of factors contribute to the effect of PAP, including age, fiber type, muscle length, training status, and strength level. A

discussion of the methodologies used to assess different types of PAP was included to facilitate the understanding of some of the methods used in the current investigation.

Chapter 3: Detailed Methods

The purpose of this study was to determine the magnitude and timing of myogenic and neurogenic factors contributing to the overall PAP response. The electrical and mechanical responses to Mmax and Hmax were measured for Day 1 (control), LFF+MVIC, and MVIC conditions. This chapter provides detailed methods related to the study population, experimental design, experimental set-up, and procedures. The EMG and stimulation parameters, data analysis, and statistical analysis are also included.

Subjects

Eleven males (age 24.2 ± 2.9 years, height 182.2 ± 9.3 cm, mass 94.2 ± 14.3 kg) and four females (age 28.0 ± 4.1 years, height 164.5 ± 13.0 cm, mass 61.2 ± 12.6 kg) participated. All subjects were healthy resistance trained volunteers between the ages of 18 and 35 years. All subjects were engaged in a whole-body resistance training program at least three times per week continuously for a duration of at least one year prior to participating (Wallace et al. 2008). Males were required to be able to maximally back squat a weight ≥1.5 times their bodyweight, and females were required to be able to maximally back squat a weight ≥1.0 times their bodyweight (Chiu et al. 2003; Wallace et al. 2008). Subjects confirmed through a Training and Health Questionnaire (THQ) that they are able to meet these strength criteria (Appendix B). Subjects were excluded from participation if they answered "yes" to one or more questions on the PAR-Q fitness questionnaire; however, no one did so (Appendix B). Prospective subjects were excluded

if they had any history of a neurological disorder or major neuromuscular lower extremity injury (examples include: a broken leg bone, or ACL tear). Race, sex, and ethnic background were not considered as inclusion or exclusion criteria.

Subjects provided written Informed Consent on a University of Kentucky Institutional Review Board (IRB) approved Informed Consent form prior to answering any questionnaires or performing any testing (Appendix A). Subjects were asked to refrain from strenuous exercise and avoid the consumption of alcohol or caffeine for at least 24 hours prior to each testing session.

Experimental Design

A quasi-experimental repeated measures design was used in this study. All procedures were approved by the University of Kentucky IRB prior to any subjects being recruited. Participants performed three testing sessions on separate days. Day 1 acted as a control, and LFF+MVIC and MVIC were the experimental conditions. The control and LFF+MVIC sessions each lasted approximately 2 hours in duration. The MVIC session lasted approximately 1.5 hours. On Day 1 the temporal profile of supramaximal (120% of the stimulation intensity determined to result in Mmax) muscle twitch and reflexive responses without a conditioning activity were recorded to establish a baseline for potentiated responses during the experimental conditions. The LFF+MVIC and MVIC conditions examined the muscle twitch and reflex potentiation responses after a 10 second MVIC. The LFF+MVIC condition was designed to induce LFF prior to the MVIC to reduce the amount of RLC phosphorylation from the conditioning activity. Supramaximal twitch and reflex responses were measured for 20 minutes in each

condition (control, LFF+MVIC, and MVIC) (Enoka et al. 1980; Folland et al. 2008; Kitago et al. 2004). The LFF+MVIC and MVIC experimental conditions were performed during the second and third testing sessions. The order of these two sessions were randomly counterbalanced between subjects in an effort to negate any potential order effect. The within-subject testing sessions were conducted at approximately the same time of day and were separated by at least 96 hours. All testing took place in a quiet laboratory housed within the Graduate Center for Gerontology at the University of Kentucky, USA.

Experimental Set-up

Subjects were positioned on a Biodex Quick Set System 4 isokinetic dynamometer (Rev. 1, Biodex Medical Systems, Shirley, NY) for each testing session. The dominant leg of each subject, defined as the one they would use to kick a ball for maximum distance, was used (Ford et al. 2003). All subjects reported that they were right leg dominant. The dynamometer's isometric mode was used to measure MVIC torque and evoked muscle twitch and reflex torques. Subjects sat in the "chair" portion of the Biodex, which provides back and thigh support. The dynamometer was adjusted such that subjects were positioned at a relative anterior hip angle of 100°, relative posterior knee angle of 120°, and relative ankle angle of 90° (Bergquist et al. 2011; Enoka et al. 1980). Hip and knee angles were determined using a handheld goniometer (Fred Sammons, Inc., Brookfield, IL). The ankle angle was determined using a standard steel construction square. The lateral malleolus was aligned with the dynamometer's armature axis of rotation. Two straps secured the foot to the foot plate. Additionally, the

foot was wrapped using a heel-lock technique to prevent the heel from coming off the foot plate during isometric plantarflexions (Ank-L Wrap, Cramer Products Inc., Gardner, KS). The experimental setup of the ankle and foot is shown in Figure 3.1. The settings associated with subject positioning on the Biodex dynamometer established on the first day of testing were recorded and used on subsequent testing days. Subjects were asked to keep their lower extremity musculature relaxed for the duration of the testing, except for during the MVICs.

Figure 3.1: Setup of the foot on the Biodex dynamometer.

Stimulation and Electromyogram Recording

To reduce impedance the skin was shaved, abraded with a gauze pad, and cleaned with isopropyl alcohol before the stimulating or EMG electrodes were placed on the skin. Impedance was required to be less than 10k Ω after skin preparation (Grass Electrode Impedance Meter, model E2M5, Grass Instruments, Grass Warrick, RI) (Camen and Gabriel 2010). The impedance of 10 preparations was tested to ensure the procedure used met the requirements. These sample preparations resulted in an impedance of 5.8 ± 3.0k Ω.

The lateral gastrocnemius and soleus were evoked by surface stimulation of the tibial nerve delivered by square-wave impulses via a constant current stimulator (DS7AH, Digitimer Ltd., Welvyn Garden City, UK) using two custom sized 2 cm x 3 cm reusable rubber electrodes. Pulse durations were 1 ms for all twitches (Kitago et al. 2004). All stimulations were controlled using custom written Matlab code (Mathworks, Inc., Natick, MA) (Appendix D). The cathode (positive lead) was placed on the skin over the tibial nerve in the popliteal fossa. The anode (negative lead) was placed over the mid-portion of the thigh approximately 2 cm proximal to the superior border of the patella (Hamada et al. 2000b, 2003; Mitchell and Sale 2011). Conductor gel was placed on the stimulating electrodes prior to them being placed and taped on the skin using surgical tape. Additionally, after the electrodes were taped to the skin an elastic bandage was wrapped around the subject's thigh, knee, and upper shank to keep the stimulating electrodes in place for the duration of each session.

A Delsys Bagnoli-8 EMG system (Delsys, Inc., Boston, MA) was used to record EMG signals. Model DE-2.1 single differential surface electrodes (Delsys, Inc., Boston,

MA) with a bipolar configuration were used to record the muscle action potentials. The Ag electrode sensors on the electrodes were 1 mm wide with an inter-sensor distance of 1 cm. The electrode housing was internally shielded and contained a pre-amplifier. The EMG electrodes were placed over the longitudinal axis of the soleus muscle belly, approximately 4 cm below the inferior margin of the gastrocnemius, and over the muscle belly of the lateral gastrocnemius at 1/3 the distance from the fibular head to the calcaneus (Figure 3.2) (Kitago et al. 2004; Trimble and Harp 1998). The electrodes were placed parallel to the predicted path of the muscle fibers and held in place with surgical tape. The ground electrode was placed on the ipsilateral patella, which for all subjects was on their right side (Bergquist et al. 2011). The placement of the stimulating and EMG electrodes was marked on the subject's skin with a permanent marker to ensure their consistent placement for each testing session. Subjects were asked to re-mark the placement of the electrodes between testing days as necessary. During testing all cables were taped to subjects to help avoid movement artifacts in the signal. EMG signals were amplified (1,000×) and band pass filtered (20 Hz-450 Hz).

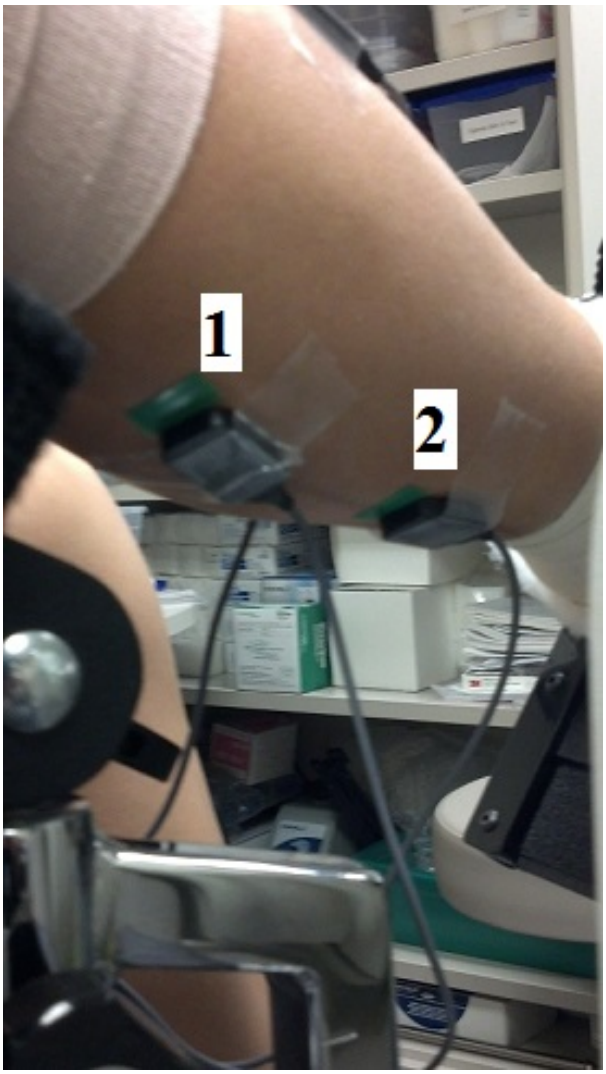

Figure 3.2: EMG electrode placements. 1) Lateral gastrocnemius, 2) Soleus.

Procedures

Day 1

After providing written Informed Consent and filling out the PAR-Q and THQ questionnaires, the stimulating and EMG electrodes were applied to subjects prior to them being positioned on the Biodex dynamometer. To remove any lingering potentiation from prior movement subjects sat passively for 20 minutes prior to testing

(Enoka et al. 1980; Folland et al. 2008; Kitago et al. 2004). Several twitches of the tibial nerve were evoked at progressively greater intensities to accustom subjects to being stimulated (Kitago et al. 2004; Folland et al. 2008; Enoka et al. 1980). Thereafter, Hmax and Mmax stimulus response curves were determined (Figure 3.3). To determine Hmax and Mmax a series of single electrical stimuli of increasing intensity were delivered to the tibial nerve. Appendix C depicts a representative H and M wave. Stimuli were delivered from 2-60 mA in 2 mA increments, 10 seconds apart (Papaiordanidou et al. 2010). Five minutes thereafter the 5 intensities in the region of Hmax were repeated to verify the previous maximum value (Folland et al. 2008). In the rare instances where the stimulation intensity required to elicit Hmax was not consistent, the higher intensity was used for the testing session. The intensity difference between the initial and secondary determinations of Hmax did not exceed one increment (2 mA).

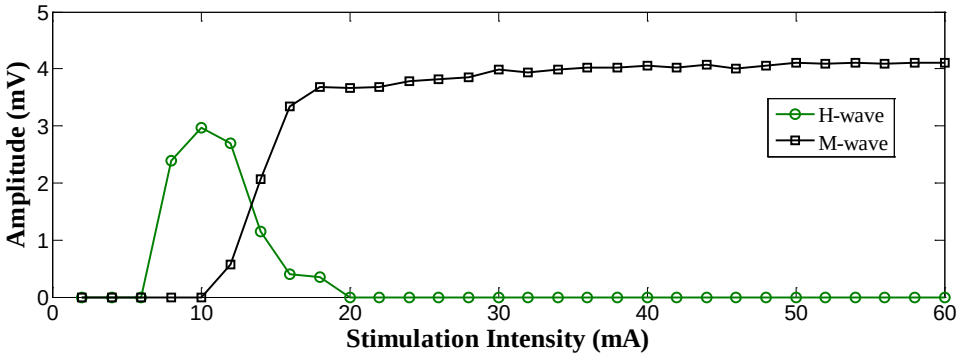

Figure 3.3: A representative H and M wave recruitment curve for one subject. Each subsequent data point along the x-axis, from left to right, represents a 2 mA increase in stimulus intensity.

Control EMG and torque values for twitches at Mmax and Hmax were established 10 minutes after Hmax and Mmax were determined (Baudry and Duchateau

2007; Requena et al. 2008). The time points of these impulses are shown in Figure 3.4. Briefly, Mmax stimulations were elicited at: 10 and 30 seconds, and 1, 2.5, 4, 6, 8, 10, 12, 14, 16, and 18 minutes, and Hmax stimulations at: 20 seconds, 1.5, 3, 4.5, 7, 9, 11, 13, 15, 17, and 20 minutes.

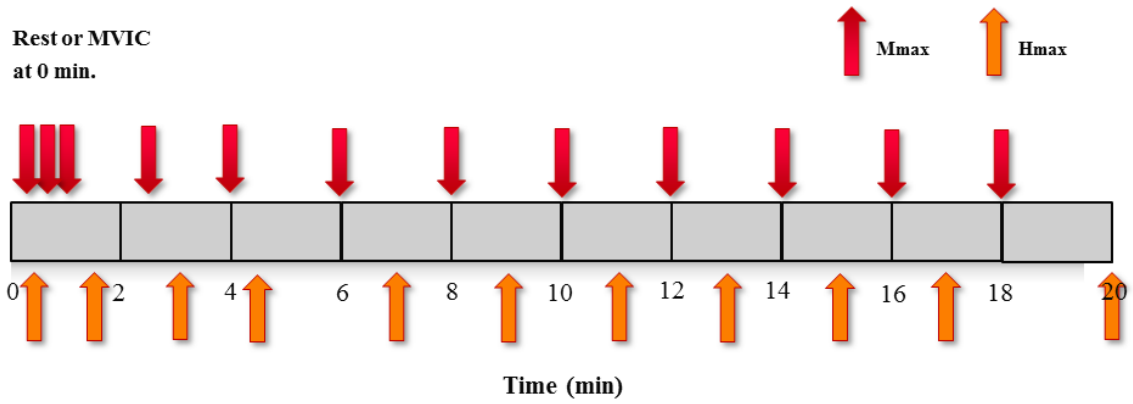

Figure 3.4: Timing of Hmax and Mmax stimulations. See text for the specific time points of stimulations.

Five minutes after baseline values were established subjects performed 3-5 10 second MVICs, separated by 1 minute, to familiarize them with the procedures they would undergo during the experimental conditions. Several minutes after the MVICs subjects experienced the LFF stimulation protocol, described below, using an estimate of the described intensity.

LFF+MVIC and MVIC Conditions

The procedures for Days 2 and 3 were identical, except that one day involved the introduction of a LFF protocol prior to the 10 second MVIC (LFF+MVIC condition). Subjects were positioned on the Biodex dynamometer as described for Day 1. They then

rested passively for 20 minutes. For both experimental conditions Hmax and Mmax were determined as described for Day 1.

The LFF protocol consisted of 7 contractions elicited at a frequency of 20 Hz with a duty cycle of 300 ms on and 1.2 seconds off at 60% of Mmax intensity (Chang and Shields 2011; Shields and Chang 1997; Shields et al. 1997; Matsunaga et al. 1999). The pulse width was 200 μs. This protocol was repeated for five trains to act as a "warm-up." After 3 minutes of passive rest this protocol was repeated for 120 contractions, lasting 3 minutes.

Following a rest period (20 minutes after the LFF protocol for the LFF+MVIC condition, or 5 minutes after the establishment of Hmax and Mmax for the MVIC condition) subjects performed a 10 second MVIC. Following the MVIC, Mmax and Hmax stimulations were evoked, and their EMG and torque values recorded, as described for the control condition on Day 1.

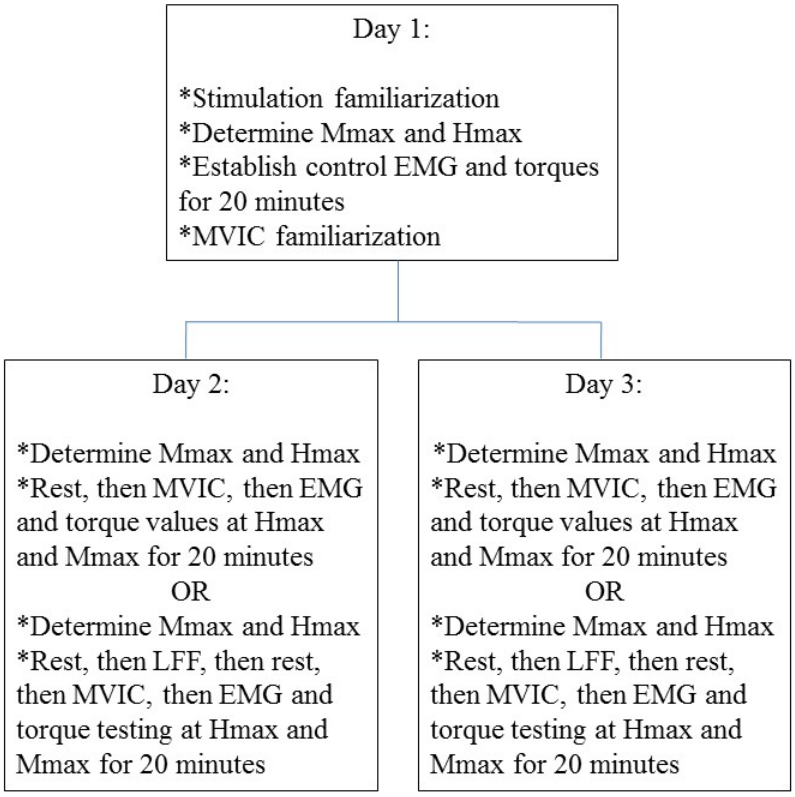

Figure 3.5: Temporal profile of study procedures.

Data Measurements and Analysis

Torque values were recorded from the analog output of the dynamometer. Analog torque and EMG data were collected through a 16-bit AD board (model USB-2659 BNC, National Instruments, Austin, TX) connected to a personal computer. Data were sampled at 4 kHz using commercially available software (EMGWorks, version 4.0, Delsys, Inc., Boston, MA). Torque was recorded during the MVIC trials for comparison between LFF+MVIC and MVIC conditions. Torques and EMG responses to stimulations at Hmax and Mmax during the three conditions were recorded. Peak twitch torque, average RFD, and peak-to-peak EMG of the gastrocnemius and soleus were calculated

for each twitch. Average RFD for each response was determined between when the signal crossed 20% of the difference between the high and low thresholds of the signal, and the peak of each response. Prior to being analyzed, torque data were filtered using a fourth order zero-lag Butterworth filter with a cutoff frequency of 24 Hz, as determined by a residual analysis (Winter 1990). Data were analyzed and compiled using standard functions and custom-written code with commercially available software (EMGWorks, version 4.0, Delsys, Inc., Boston, MA; Excel 2013, Microsoft Corporation, Redmond, WA; Matlab, version R2012b, Mathworks, Inc., Natick, MA) (Appendix E).

Statistical Analysis

Data are reported as mean ± SD. Reliability of the H-wave amplitude (Hmax/Mmax) between the three testing days were assessed for both the gastrocnemius and soleus using intraclass correlation coefficients (ICCs). During the 20 minutes of testing the EMG and torque values at Hmax were normalized to the temporally nearest Mmax EMG and torque values to determine Hmax/Mmax ratios. In situations where Hmax was elicited at the temporal midpoint between two Mmax stimulations, Hmax was normalized to the preceding Mmax stimulation.

One-way analyses of variance (ANOVA) were used to test for statistically significant differences between conditions at select time points after rest or MVICs. Specifically, torque and RFD in response to stimulations at Mmax were analyzed at 10 seconds and 18 minutes. Torque, RFD, and EMG values for stimulations at Hmax were analyzed at 20 second, inter-variable peak (i.e.: 3 minutes for torque, 4.5 minutes for RFD, and 4.5 minutes for gastrocnemius and soleus), and 20 minute time points.

Conditions were aligned for analysis where the inter-condition EMG peaks did not temporally align within a dependent variable. Holm-Sidak post-hoc analysis were used if significant differences were found with an ANOVA. The percent change between the treatment conditions vs. control condition were calculated. Linear regression analyses were performed on the MVIC condition to calculate coefficients of determination (e.g.: r^2 values) and the significance of the regression coefficients at select time points for torque at Hmax vs. torque at Mmax and EMG; and for RFD at Hmax vs. RFD at Mmax and EMG. ICCs were computed using the Statistical Package for the Social Sciences (SPSS, version 22, IBM Corporation, Armonk, NY). All other statistical analyses were performed using SigmaPlot (version 12.3, Systat Software, Inc., San Jose, CA). An $\alpha \leq .05$ was used to indicate statistical significance for all analyses.

Chapter 4: Muscular and Neural Contributions to Postactivation Potentiation

Abstract

Muscle performance after muscle contractions may be either reduced through fatigue or enhanced through postactivation potentiation (PAP). PAP can improve performance in athletics and training, particularly through complex training. The objective of this study was to explain the relationship between muscle factors (twitch potentiation, TP) and neural factors (reflex potentiation, RP) contributing to overall PAP following a non-fatiguing volitional muscle contraction. The tibial nerves of eleven men (age 24.2 ± 2.9 years, height 182.2 ± 9.3 cm, mass 94.2 ± 14.3 kg) and four women (age 28.0 ± 4.1 years, height 164.5 ± 13.0 cm, mass 61.2 ± 12.6 kg) who had at least one year of continuous resistance training experience were stimulated intermittently at supramaximal (Mmax) and submaximal (Hmax) intensities for 20 minutes on separate days under three conditions: 1) rest (Control); 2) after a after a 10 second maximum voluntary isometric contraction (MVIC) of the plantarflexors; and 3) after a low frequency fatigue protocol prior to the MVIC. Plantarflexion isometric torque and rate of force development (RFD), and soleus and gastrocnemius EMG Hmax/Mmax ratios, were analyzed. Both experimental conditions resulted in TP at 10 seconds post-MVIC compared to Control ($p \leq 0.05$). The two experimental conditions were not different for any measure ($p > 0.05$). Torque and RFD at Hmax (overall PAP) were highest at 3 and 4.5 minutes post MVIC, respectively, but were not significantly different from the control condition ($p > 0.05$). EMG values generally were non-significantly increased in the experimental conditions versus Control ($p > 0.05$). Mmax torque and RFD significantly contributed to Hmax

torque and RFD at 20 seconds, Hmax peak, and 20 minute post-MVIC time points (torque: $r^2 = 0.54$, 0.76, and 0.70, $p \leq 0.05$; RFD: $r^2 = 0.46$, 0.59, 0.53, $p \leq 0.05$). The soleus significantly contributed to Hmax torque at 20 seconds and 20 minutes post-MVIC, and Hmax RFD at 20 seconds, 4.5 minutes, and 20 minutes post-MVIC (torque: $r^2 = 0.26$, and 0.34, $p \leq 0.05$; RFD: $r^2 = 0.65$, 0.52, 0.41, $p \leq 0.05$). The results of this study suggest that both muscle and neural factors play a significant role in overall PAP, and that neural factors may play a more prominent role in RFD potentiation than torque potentiation.

Keywords

Postactivation potentiation, Twitch potentiation, Reflex potentiation, H-reflex, Postactivation depression, Rate of force development

Introduction

It is well established that the contractile performance of skeletal muscle may be either reduced or enhanced based on its recent contractile history. Sustained contractions result in performance reducing fatigue, while a smaller number of contractions may result in performance being increased through postactivation potentiation (PAP). Peak force and rate of force development (RFD) are the two performance properties that can be improved through PAP (Sale 2002). A conditioning activity, usually taking the form of a maximum voluntary isometric contraction (MVIC) or resistance training exercise, is required to elicit PAP. Short duration conditioning activities (i.e.: MVIC $\leq$ 10 seconds) elicit PAP, while long duration (i.e.: MVIC $\geq$ 60 seconds) ones have been shown to elicit

both PAP and peripheral fatigue (Vandervoort et al. 1983; Houston and Grange 1990; Grange and Houston 1991). The coupling of PAP and fatigue determines the net muscle response (Vandenboom et al. 1993).

Both central and peripheral mechanisms are responsible for muscle behavior during volitional movement (Gullich and Schmidtbleicher 1996; Hodgson et al. 2005). PAP has most often been measured as twitch potentiation (TP), defined as the muscle force or RFD produced in response to a relatively high intensity twitch that produces maximum muscle force, delivered by electrical stimulation (Gossen and Sale 2000; Vandenboom et al. 1995). However, this is a consequence of actin-myosin cross-bridge kinetics and possibly increased stimulus efficacy (i.e.: sodium-potassium pump activity), and fails to account for the influence of the nervous system in muscle recruitment during volitional effort (Hamada et al. 2000b; Tillin and Bishop 2009; Klakowicz et al. 2006; Hicks et al. 1989). The Hoffmann reflex (H-reflex) is measured as the electromyographic (EMG) signal in response to a stimulus intensity that elicits the largest H-wave divided by the EMG signal of a maximal twitch (Hmax/Mmax ratio), and is widely recognized as a means of measuring motor neuron pool excitability during isometric muscle activation (Palmieri et al. 2002; Zehr 2002). An increase in the Hmax/Mmax ratio in response to a conditioning activity is termed reflex potentiation (RP) (Trimble and Harp 1998; Folland et al. 2008). A higher ratio can be attributed to more and/or larger motor units being recruited (Tillin and Bishop 2009; Gullich and Schmidtbleicher 1996). Methods of recruiting more motor units may be important in performance training. The influence of chronic weight training on motor unit recruitment has been of interest for some time (Sale et al. 1983). More recently complex training, executed by performing a “complex pair”

of one or more sets of a resistance exercise at a relatively high percent one-repetition-maximum followed by plyometrics several minutes later, has been used to help achieve preferential physiological adaptations to training (Docherty et al. 2004). Greater force and RFD is elicited during the plyometric exercise after performing the weight training activity than what would be in its absence, resulting in superior long-term adaptations (Docherty and Hodgson 2007; Docherty et al. 2004).

Enoka et al. (1980) first reported that a reduction in H-wave amplitude (postactivation depression (PAD)) occurs after muscle contractions. Three studies have investigated the time course of the H-wave amplitude after a conditioning activity, each reporting PAD followed by RP 3+ minutes thereafter. In contrast to TP which is highest immediately after the conditioning activity (Vandervoort et al. 1983; Houston and Grange 1990; Requena et al. 2008) and quickly subsides due to myosin regulatory light chain dephosphorylation (Houston and Grange 1990), the timing of RP better relates temporally to when PAP has been reported with volitional performance-oriented movements (Wilson et al. 2013). This indicates that RP may be an important factor in force and RFD production during volitional muscle activation. It has been suggested that RFD is the most important factor in athletic feats that have a force production duration between 100 and 300 ms such as throwing, running and jumping (Tidow 1990), and maximum force production is most important in performing longer duration movements (Wilson et al. 1995). The purpose of this study was to explain the relationship between muscle and neural factors contributing to overall PAP (i.e.: torque and RFD at Hmax) (Folland et al. 2008) for 20 minutes following a non-fatiguing MVIC. No studies have investigated the contribution of TP and RP to overall PAP in the triceps surae. We hypothesized that both

TP and RP would significantly contribute to overall PAP, with TP having more of an influence soon after the MVIC and RP having more of an influence thereafter. We also hypothesized that the gastrocnemius would contribute more to overall PAP than the soleus.

Methods

2.1 Subjects

Eleven men (age 24.2 ± 2.9 years, height 182.2 ± 9.3 cm, mass 94.2 ± 14.3 kg) and four women (age 28.0 ± 4.1 years, height 164.5 ± 13.0 cm, mass 61.2 ± 12.6 kg) participated. All participants were healthy resistance trained volunteers between the ages of 18 and 35 years, and were engaged in a whole-body resistance training program at least three times per week continuously for at least one year (Wallace et al. 2008). Men were required to be able to maximally back squat a weight ≥1.5 times their bodyweight, and women were required to be able to maximally back squat a weight ≥1.0 times their bodyweight (Chiu et al. 2003; Wallace et al. 2008). Prospective participants were excluded if they had any history of neurological disorder or major neuromuscular lower extremity injury. Participants were asked to refrain from strenuous exercise and avoid the consumption of alcohol or caffeine for at least 24 hours prior to each testing session. Subjects provided written Informed Consent on an institution approved form prior to taking part in any study procedures.

2.2 Experimental Design

A repeated measures design was used in this study. Participants performed three testing sessions on separate days: Day 1, LFF+MVIC, and MVIC. Day 1 acted as a control, where the temporal profile of supramaximal (120% of the stimulation intensity determined to result in Mmax) muscle twitch and reflexive responses without a conditioning activity were recorded to establish a baseline to potentiated responses from the two experimental conditions. The LFF+MVIC and MVIC conditions examined the muscle twitch and reflex potentiation responses after a 10 second MVIC. The LFF+MVIC condition was designed to induce LFF prior to the MVIC to reduce the amount of RLC phosphorylation from the conditioning activity. Supramaximal twitch and reflex responses were measured for 20 minutes in each condition. The order of the experimental conditions was counterbalanced between subjects. Within-subject testing sessions were conducted at approximately the same time of day and were separated by at least 96 hours (Zehr 2002). All testing was conducted in a quiet laboratory (Zehr 2002).

2.3 Experimental Set-up

Subjects were positioned on a Biodex Quick Set System 4 isokinetic dynamometer (Rev. 1, Biodex Medical Systems, Shirley, NY) for each testing session. The dominant leg of each subject, defined as the one they would use to kick a ball for maximum distance (Ford et al. 2003), was used. All subjects were right leg dominant. The dynamometer's isometric mode measured MVIC torque and evoked muscle twitch and reflex torques. Subjects were positioned in the dynamometer to a relative anterior hip angle of 100°, relative posterior knee angle of 120°, and relative ankle angle of 90°.

The lateral malleolus was aligned with the dynamometer's armature axis of rotation. Two straps secured the foot to the foot plate. Additionally, the foot was wrapped using a heel-lock technique to prevent the heel from coming off the foot plate during isometric plantarflexions (Ank-L Wrap, Cramer Products Inc., Gardner, KS). The experimental setup of the ankle and foot is shown in Figure 4.1. The Biodex settings associated with subject positioning established on Day 1 were recorded and used on subsequent days. Subjects were asked to keep their lower extremity musculature relaxed during testing, except while performing MVICs.

Figure 4.1: Setup of the foot on the Biodex dynamometer.

2.4 Stimulation and Electromyogram Recording

To reduce impedance the skin was shaved, abraded with a gauze pad, and cleaned with isopropyl alcohol before the stimulating or EMG electrodes were placed on the skin. The lateral gastrocnemius and soleus were evoked by surface stimulation of the tibial nerve delivered by square-wave impulses via a constant current stimulator (DS7AH, Digitimer Ltd., Welvyn Garden City, UK) using two custom sized 2 cm x 3 cm reusable rubber electrodes. The cathode (positive lead) was placed on the skin over the tibial nerve in the popliteal fossa. The anode (negative lead) was placed over the mid-portion of the thigh approximately 2 cm proximal to the superior border of the patella (Hamada et al. 2000b, 2003; Mitchell and Sale 2011). Conductor gel was placed on the stimulating electrodes prior to them being placed and taped on the skin using surgical tape. After the electrodes were taped to the skin an elastic bandage was wrapped around the subject's thigh, knee, and upper shank to keep the stimulating electrodes in place for the session. Pulse durations were 1 ms for all twitches (Kitago et al. 2004). All stimulations were controlled using custom written Matlab code (Mathworks, Inc., Natick, MA).

A Delsys Bagnoli-8 EMG system (Delsys, Inc., Boston, MA) was used to record EMG signals. Model DE-2.1 single differential surface electrodes (Delsys, Inc., Boston, MA) with a bipolar configuration recorded muscle action potentials. The Ag electrode sensors on the electrodes were 1 mm wide with an inter-sensor distance of 1 cm. The electrode housing was internally shielded and contained a pre-amplifier. The EMG electrodes were placed over the longitudinal axis of the soleus muscle belly approximately 4 cm below the inferior margin of the gastrocnemius (Kitago et al. 2004), and over the muscle belly of the lateral gastrocnemius at 1/3 the distance from the fibular

head to the calcaneus (Trimble and Harp 1998). The ground electrode was placed on the ipsilateral patella, which for all subjects was on their right side (Bergquist et al. 2011). The placement of all electrodes were marked with a permanent marker to ensure their consistent placement for each testing session. Subjects were asked to re-mark the placements as necessary between testing sessions. EMG signals were amplified (1,000×) and band pass filtered (20 Hz-450 Hz).

2.5 Procedures

Day 1

The stimulating and EMG electrodes were applied to subjects prior to them being positioned on the Biodex dynamometer. Subjects sat passively for 20 minutes prior to testing to remove any lingering potentiation from prior movement (Enoka et al. 1980; Folland et al. 2008; Kitago et al. 2004). Several twitches of the tibial nerve were evoked at progressively greater intensities to accustom subjects to being stimulated (Kitago et al. 2004; Folland et al. 2008; Enoka et al. 1980). Thereafter, Hmax and Mmax stimulus response curves were determined (Figure 4.2). To determine Hmax and Mmax a series of single electrical stimuli of increasing intensity were delivered to the tibial nerve. Stimuli were delivered from 2-60 mA in 2 mA increments, 10 seconds apart (Papaiordanidou et al. 2010). Five minutes thereafter the 5 intensities in the region of Hmax were repeated to verify the previous maximum value (Folland et al. 2008). In the rare instances where the stimulation intensity required to elicit Hmax was not consistent, the higher intensity was used for testing. The intensity difference between the initial and secondary determinations of Hmax did not exceed one increment (2 mA).

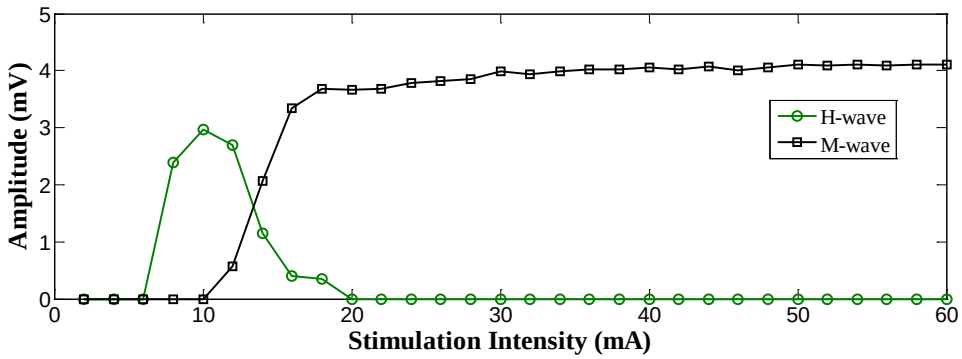

Figure 4.2: A representative H and M wave recruitment curve for one subject. Each subsequent data point along the x-axis, from left to right, represents a 2 mA increase in stimulus intensity.

Control EMG and torque values for twitches at Mmax and Hmax were established 10 minutes after Hmax and Mmax were determined (Baudry and Duchateau 2007; Requena et al. 2008). The time points of these impulses are shown in Figure 4.3. Briefly, Mmax stimulations were elicited at: 10 and 30 seconds, and 1, 2.5, 4, 6, 8, 10, 12, 14, 16, and 18 minutes, and Hmax stimulations at: 20 seconds, 1.5, 3, 4.5, 7, 9, 11, 13, 15, 17, and 20 minutes.

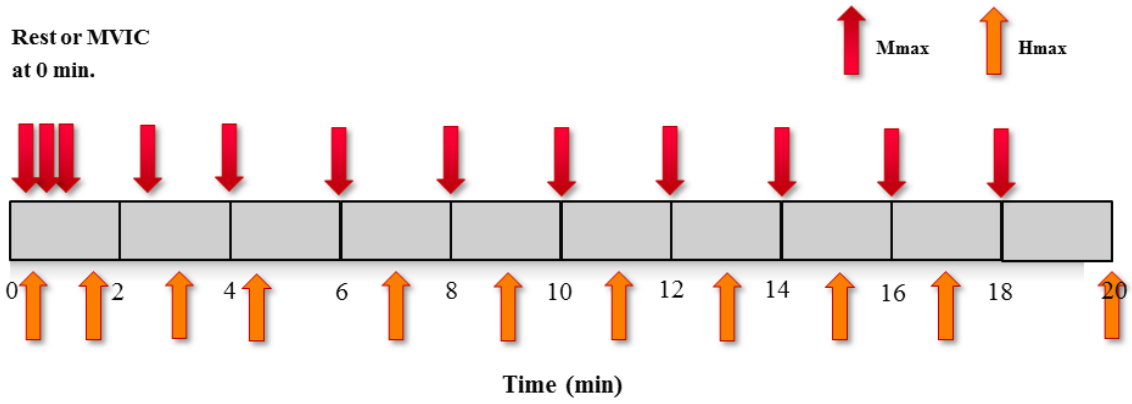

Figure 4.3: Timing of Hmax and Mmax stimulations. See text for the specific time points of stimulations.

Five minutes after baseline values were established subjects performed 3-5 10 second MVICs, separated by 1 minute, to familiarize them with the procedures they would undergo on subsequent testing days. Several minutes after the MVICs subjects experienced the LFF stimulation protocol, described below, using an estimate of the described intensity.

LFF+MVIC and MVIC Conditions

The procedures for testing the two experimental conditions were identical, except that one day involved the introduction of a LFF protocol prior to the 10 second MVIC. Subjects were positioned on the Biodex dynamometer as described for Day 1. They then rested passively for 20 minutes. For both experimental conditions Hmax and Mmax were determined as described for Day 1.

The LFF protocol consisted of 7 contractions elicited at a frequency of 20 Hz with a duty cycle of 300 ms on and 1.2 seconds off at 60% of Mmax intensity (Chang and Shields 2011; Shields and Chang 1997; Shields et al. 1997; Matsunaga et al. 1999). The

pulse width was 200 μs. Five trains acted as a "warm-up." After 3 minutes of passive rest the protocol was repeated for 120 contractions, which lasted 3 minutes.

Following a rest period of 20 minutes after the LFF protocol for the LFF+MFIC condition, or 5 minutes after the establishment of Hmax and Mmax for the MVIC condition, subjects performed a 10 second MVIC. Following the MVIC, Mmax and Hmax stimulations were evoked, and their EMG and torque values recorded, as described for the control condition on Day 1.

2.6 Data Measurements and Analysis

Torque values were recorded from the analog output of the dynamometer. Analog torque and EMG data were collected through a 16-bit AD board (model USB-2659 BNC, National Instruments, Austin, TX) connected to a personal computer. Data were sampled at 4 kHz using commercially available software (EMGWorks, version 4.0, Delsys, Inc., Boston, MA). Torque was recorded during the MVIC trials for comparison between LFF+MVIC and MVIC conditions. Torques and EMG responses to stimulations at Hmax and Mmax during the three conditions were recorded. Peak twitch torque, average RFD, and peak-to-peak EMG of the gastrocnemius and soleus were calculated for each twitch. Average RFD for each response was determined between when the signal crossed 20% of the difference between the high and low thresholds of the signal, and the peak of each response. Prior to being analyzed, torque data were filtered using a fourth order zero-lag Butterworth filter, with a cutoff frequency of 24 Hz as determined by a residual analysis (Winter 1990). Data were analyzed and compiled using standard functions and custom-written code with commercially available software (EMGWorks,

version 4.0, Delsys, Inc., Boston, MA; Excel 2013, Microsoft Corporation, Redmond, WA; Matlab, version R2012b, Mathworks, Inc., Natick, MA).

2.7 Statistical Analysis

Data are reported as mean ± SD. Reliability of the H-wave amplitude (Hmax/Mmax) between the three testing days were assessed for both the gastrocnemius and soleus using intraclass correlation coefficients (ICCs). The EMG and torque values at Hmax were normalized to the temporally nearest Mmax EMG and torque values to determine Hmax/Mmax ratios. In situations where Hmax was elicited at the temporal midpoint between two Mmax stimulations, Hmax was normalized to the preceding Mmax stimulation. One-way analyses of variance (ANOVA) were used to test for statistically significant differences between conditions at select time points. Specifically, torque and RFD in response to stimulations at Mmax were analyzed at 10 second and 18 minute post MVIC time points. Torque, RFD, and EMG values for stimulations at Hmax were analyzed at 20 second, inter-variable peak (i.e.: 3 minutes for torque, 4.5 minutes for RFD, and 4.5 minutes for gastrocnemius and soleus EMG), and 20 minute time points. Inter-condition gastrocnemius and soleus EMG peaks data were temporally aligned for analysis. Holm-Sidak post-hoc analysis was used if significant differences were found with an ANOVA. The percent change between the treatment conditions vs. control condition were calculated. Linear regression analyses were performed on the MVIC condition to calculate coefficients of determination (e.g.: r^2 values) and the significance of the regression coefficients at select time points for Hmax torque vs. Mmax torque and Hmax torque vs. EMG; and for Hmax RFD vs. Mmax RFD, and Hmax RFD vs. EMG.

All statistical analyses were performed using SigmaPlot (version 12.3, Systat Software, Inc., San Jose, CA). An $\alpha \leq .05$ was used to indicate statistical significance.

Results

3.1 Reliability of H-wave responses

H-wave amplitude (Hmax/Mmax) was shown to be reliable for both the gastrocnemius and soleus. The ICC was 0.94 (95% CI 0.87-0.98) for the gastrocnemius and 0.91 (95% CI 0.79-0.97) for the soleus (Appendix D).

3.2 Twitch and reflex responses

Both experimental conditions showed significant TP vs. the control condition for both torque and RFD at 10 seconds post-MVIC (both $p < 0.001$, Figures 4.4 and 4.5). Despite an increased trend, there were no significant differences in experimental vs. control conditions after the 10 second post-MVIC mark. At the 18 minute mark the LFF+MVIC and MVIC conditions were increased from control by 3.94 and 1.33 percent for torque and 7.51 and 5.73 percent for RFD, respectively (torque: $p = 0.902$; RFD: $p = 0.673$, Table 4.1). However, these increases were not statistically significant.

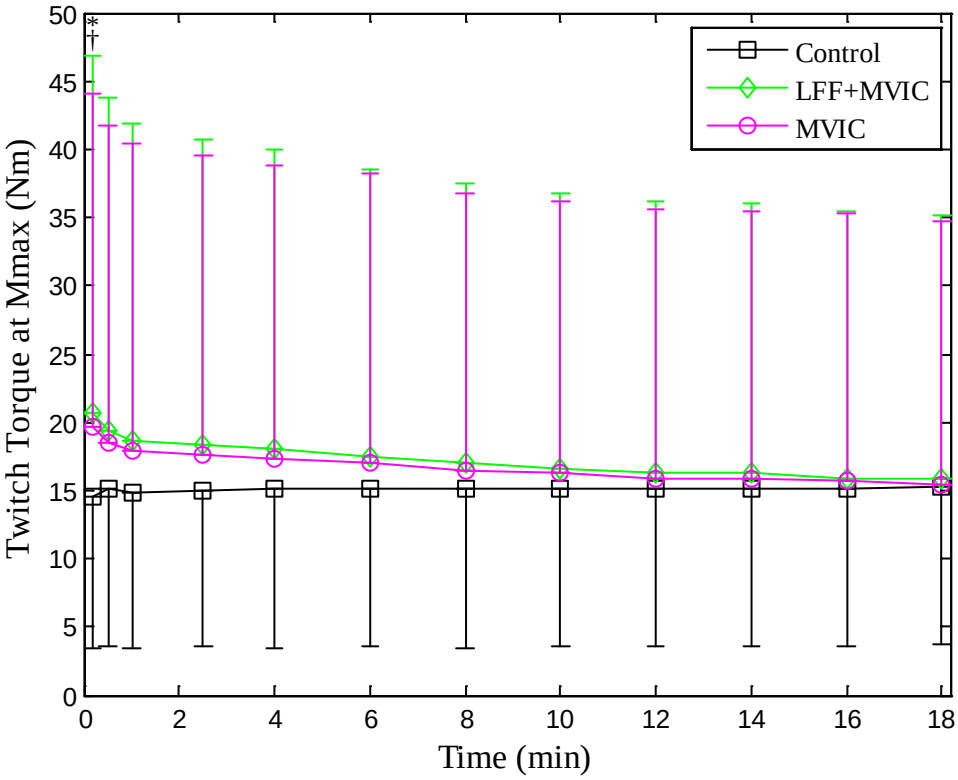

Figure 4.4: Twitch torque at Mmax stimulations vs. time. * Significant difference Control vs. LFF+MVIC. † Significant difference Control vs. MVIC. $p \leq .05$. See text for inter-condition time points compared.

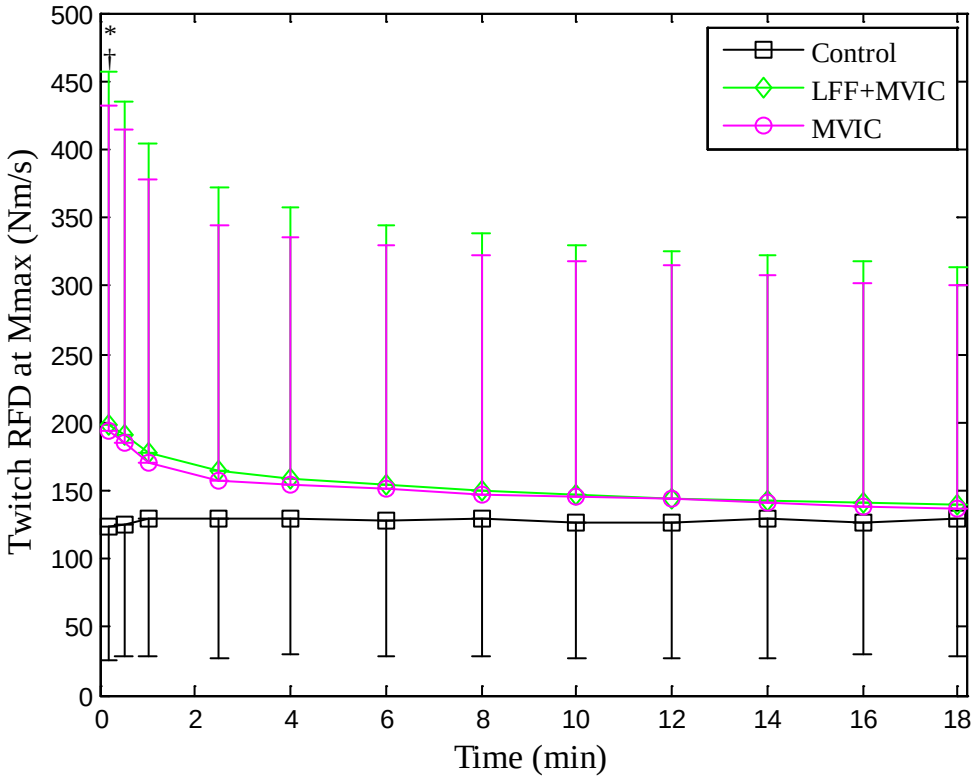

Figure 4.5: Twitch RFD at Mmax stimulations vs. time. * Significant difference Control vs. LFF+MVIC. † Significant difference Control vs. MVIC. $p \leq .05$. See text for inter-condition time points compared.

Torque at Hmax was highest at 3 minutes post-MVIC (Figure 4.6), and Hmax RFD was highest at 4.5 minutes (Figure 4.7). There were no significant differences between conditions for torque at 20 seconds, 3 minutes, or 20 minutes post-MVIC ($p = 0.264$, 0.186, and 0.387 respectively), or for RFD at 20 seconds, 4.5 minutes, or 20 minutes ($p = 0.721$, 0.482, and 0.487, respectively). Hmax percent change vs. the control condition ranged from -10.73% to 15.52% for torque and 11.38% to 22.29% for RFD (Table 4.1). The largest percent changes were observed at Hmax peak; that is, at 3 minutes post-MVIC for torque and 4.5 minutes for RFD.

Table 4.1: Percent change between Control vs. experimental conditions for torque and RFD in response to twitches at Hmax and Mmax at select time points after MVICs.

	Mmax		Hmax		
	Torque (Nm)				
	10 sec.	18 min.	20 sec.	3 min.	20 min.
LFF+MVIC	41.34	3.94	-3.36	10.04	-10.73
MVIC	34.42	1.33	7.25	15.52	1.19
	RFD (Nm/s)				
	10 sec.	18 min.	20 sec.	4.5 min.	20 min.
LFF+MVIC	60.04	7.51	17.87	19.81	11.38
MVIC	56.57	5.73	20.07	22.29	15.42

Positive values represent an increase vs. Control.

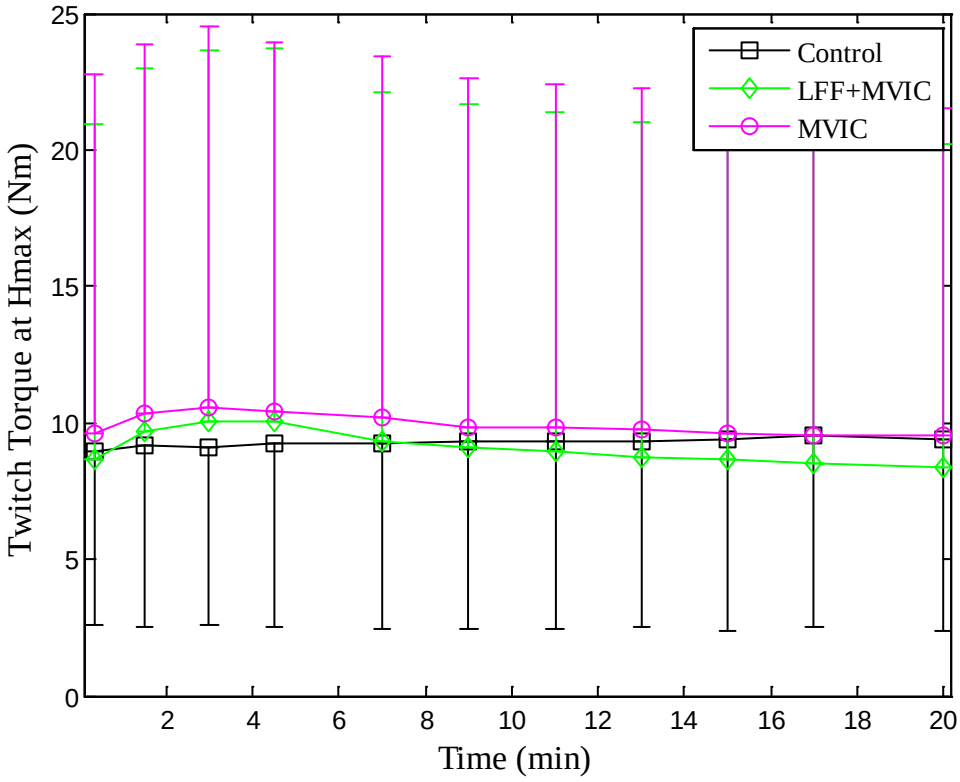

Figure 4.6: Twitch torque at Hmax stimulations vs. time. No significant differences between Control and experimental conditions. See text for inter-condition time points compared.

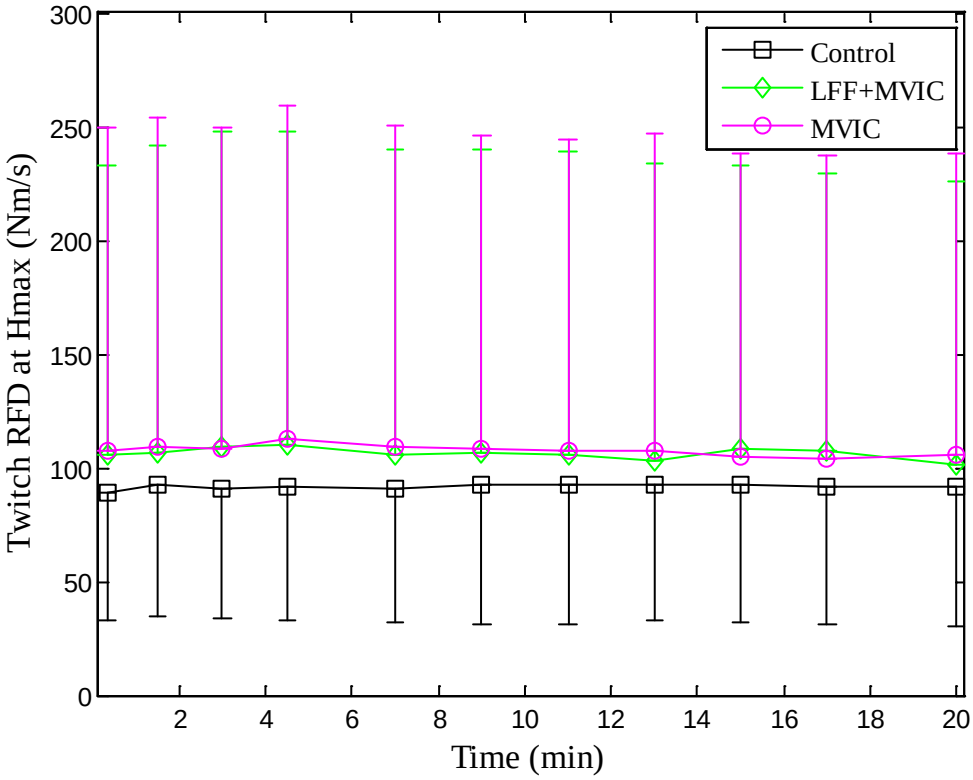

Figure 4.7: Twitch RFD at Hmax stimulations vs. time. No significant differences between Control and experimental conditions. See text for inter-condition time points compared.

Reflex potentiation was not observed in this study (Figures 4.8 and 4.9). There were no statistically significant differences between conditions at the 20 seconds, 4.5 minutes, or 20 minutes post-MVIC time points analyzed for soleus ($p = 0.552$, 0.642, and 0.404 respectively) or gastrocnemius ($p= 0.977$, 0.883, and 0.863, respectively) Hmax/Mmax ratio. The percent change was highest at the 4.5 minute time point in both experimental conditions for both muscles vs. the control condition (Table 4.2).

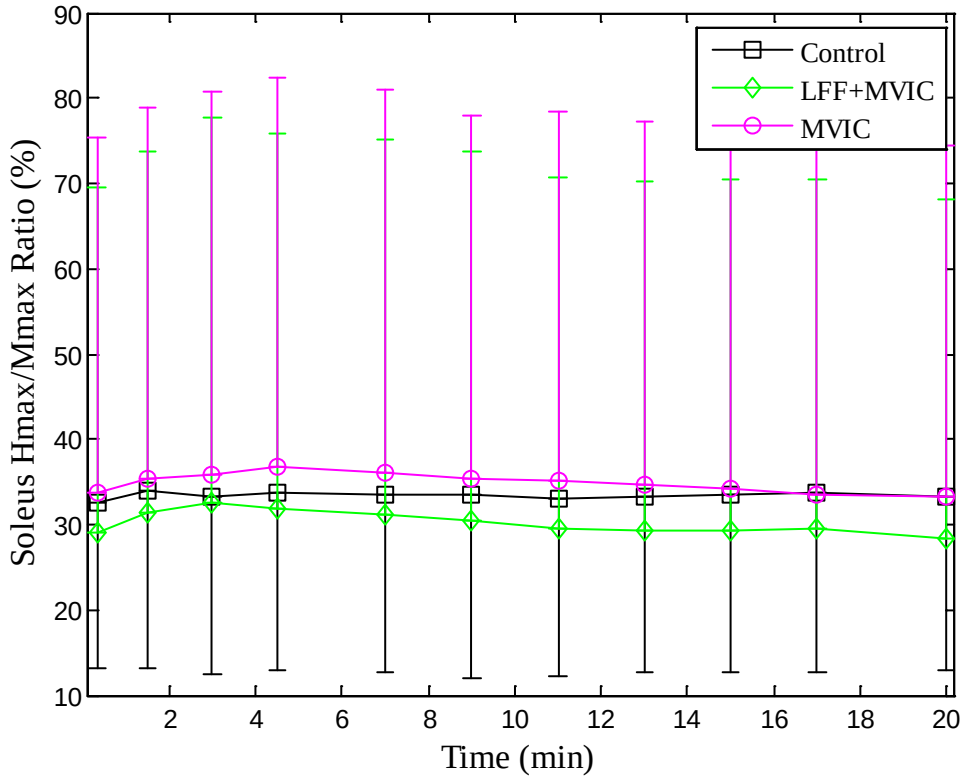

Figure 4.8: Soleus Hmax/Mmax ratio vs. time. No significant differences between Control and experimental conditions. See text for inter-condition time points compared.

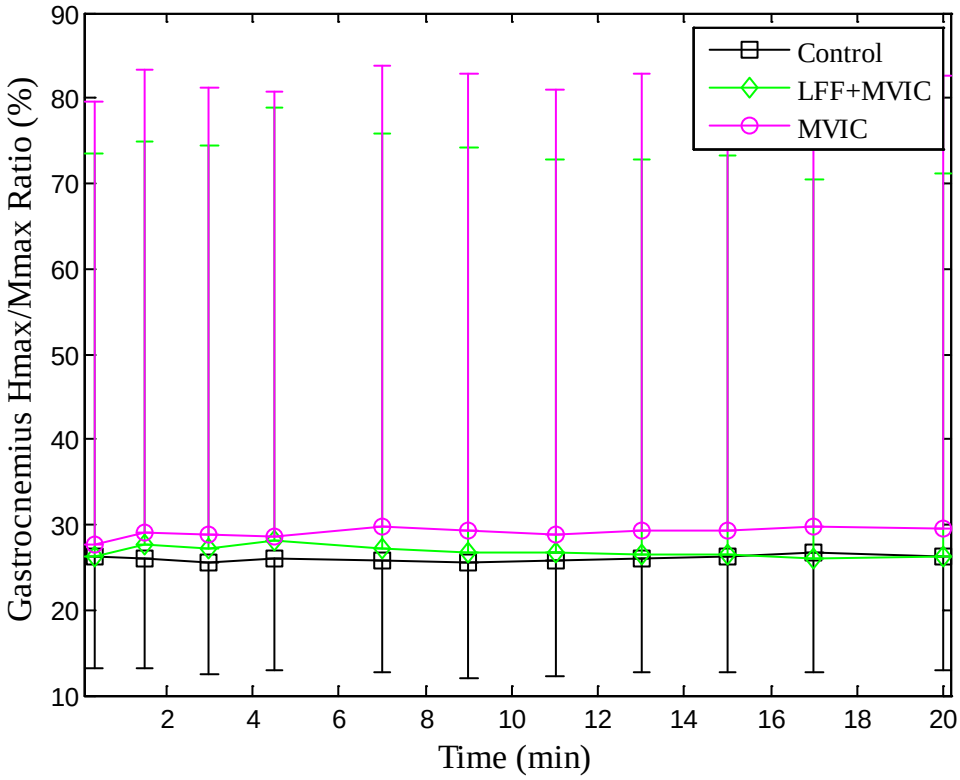

Figure 4.9: Gastrocnemius Hmax/Mmax ratio vs. time. No significant differences between Control and experimental conditions. See text for inter-condition time points compared.

Table 4.2: Percent change between Control vs. experimental conditions for EMG values in response to twitches at Hmax at beginning, Hmax torque peak, and end time points after MVICs.

	20 sec.	4.5 min.	20 min.
	Soleus		
LFF+MVIC	-11.04	-2.92	-14.72
MVIC	3.12	9.41	-0.07
	Gastrocnemius		
LFF+MVIC	0.55	7.52	-0.05
MVIC	5.45	14.32	12.33

Positive values represent an increase vs. Control.

3.3 Coefficients of determination

The coefficients of determination that were calculated for the two measures of overall potentiation, Hmax torque and RFD, at initial (20 seconds), Hmax peak (3 minutes for torque, 4.5 minutes for RFD), and end time points (20 minutes) post-MVIC are shown in Table 4.3. Mmax torque and RFD significantly contributed to Hmax torque and RFD at all time points analyzed (torque: $r^2 = 0.54$, 0.76, and 0.70 at 20 seconds, 3 minutes, and 20 minutes, $p \leq 0.05$; RFD: $r^2 = 0.46$, 0.59, 0.53 at 20 seconds, 4.5 minutes, and 20 minutes, $p \leq 0.05$). The soleus' EMG ratio significantly contributed to Hmax torque at 20 seconds and 20 minutes post-MVIC ($r^2 = 0.26$ and 0.34, $p \leq 0.05$). It also significantly contributed to Hmax RFD at 20 seconds, 4.5 minutes, and 20 minutes time points ($r^2 = 0.65$, 0.52, 0.41, $p \leq 0.05$). The gastrocnemius did not significantly contribute to the variation in Hmax torque or RFD at any time point.

Table 4.3: Coefficients of determination for EMG and Mmax torque and RFD contributions to Hmax torque and Hmax RFD at initial, Hmax peak, and end time points in MVIC condition.

	Hmax Torque		
	20 sec.	3 min.	20 min.
Gastrocnemius	5.80E-05	3.00E-04	0.02
Soleus	0.26*	0.22	0.34*
Mmax	0.54*	0.76*	0.70*
	Hmax RFD		
	20 sec.	4.5 min.	20 min.
Gastrocnemius	3.00E-03	0.02	0.07
Soleus	0.65*	0.52*	0.41*
Mmax	0.46*	0.59*	0.53*

Values reported as r^2. * denotes significance ($p \leq .05$). Comparisons with Mmax made using temporally closest time point.

Discussion

We investigated both peak torque and RFD as measures of overall PAP after a non-fatiguing MVIC. Alterations in twitch torque at Hmax intensity did not reach statistical significance in either of the experimental conditions. It was depressed by 3% in the LFF+MVIC condition, and enhanced by 7% in the MVIC condition, at 20 seconds post MVIC. Two previous studies reported conflicting results in this area. Gullich and Schmidtbleicher (1996) tested subjects for dynamic voluntary isometric force of the plantarflexors after MVICs. They reported that voluntary force was significantly depressed by approximately 12% for the first two minutes of recovery before being potentiated starting at 4 minutes. A more recent study measured torque at Hmax, similar to the methods used in the present investigation (Folland et al. 2008). They did not utilize a LFF condition, and reported similar results as our MVIC condition, although the percent change reached up to approximately 20% in their study. This difference may be due to the fact that we tested the plantarflexors versus their testing of the quadriceps. The dissimilar results of these three studies likely has to do with the conditioning activities utilized. Whereas we and Folland (2008) used a 10 second MVIC, Gullich and colleague (1996) used five 5 second MVICs. The 25 seconds of MVICs could have elicited peripheral fatigue that the 10 second MVICs did not. However, peak twitch torque after 10 and 30 second plantarflexion MVICs has been reported to be similar (Vandervoort et al. 1983), suggesting similar peripheral conditions. It is more likely that PAD was elicited as a result of the longer conditioning activity. This is substantiated by the authors reporting that H-wave amplitude was depressed by approximately 20% during the time

period where reduced voluntary plantarflexion force was observed (Gullich and Schmidtbleicher 1996).

Our study is the first to investigate RFD as a measure of overall potentiation. Similar to torque, RFD at Hmax stimulation intensities did not reach statistical significance between conditions at any of the three time points analyzed. Previous studies have reported equivocal results for potentiation of RFD after a conditioning activity. In mammalian models RFD is increased in response to a supramaximal twitch (Vandenboom et al. 1995) and tetanic stimulation (Abbate et al. 2000). In humans, voluntary knee extension velocity was not increased following a 10 second MVIC (Gossen and Sale 2000), as was used in the present study. An increase in velocity would be indicative of an enhanced ability to produce force quickly. Increased velocity has been shown in the mouse extensor digitorum longus, however (Grange et al. 1995). Baudry and Duchateau (2007) reported an increase in RFD of the thumb adductors during maximum effort contractions for the 5 minutes following a 6 second MVIC. In the current study the percent changes in RFD at Hmax compared to the control condition were approximately 20% greater at 20 seconds and 4.5 minutes post-MVIC, and slightly lower at 20 minutes, as shown in Figure 4.7. The relative effect of PAP is more pronounced at lower stimulation frequencies (Baudry et al. 2005; Baudry and Duchateau 2007). Therefore, the statistically insignificant results of the present study and Gossen and Sale (2000) could be due to the use of measures of maximal volitional effort in comparison to submaximal effort.

The most important findings of our study are the contributions of TP and RP to overall potentiation. Our hypothesis that both TP and RP would significantly contribute

to overall PAP was supported. Mmax torque significantly accounted for between 54 and 76% of the variance in Hmax torque, increasing from the first to last time point, with other factors such as RP accounting for the remainder. The Hmax/Mmax ratio of the soleus accounted for 22 to 34% of the variance in Hmax torque. RP, specifically via the soleus, accounted for more of the variance in RFD at Hmax than it did with torque. The r^2 of Mmax RFD to Hmax RFD was between 0.46 and 0.59. Based on these data our hypothesis that TP would have more of an influence on overall PAP soon after the MVIC, and RP would have more of an influence thereafter, was not supported. It has previously been shown that overall torque PAP is closely related to TP soon after an MVIC, and closely related to RP starting approximately three minutes after (Folland et al. 2008). Twitch responses have also been related to volitional performance in both the quadriceps (Mitchell and Sale 2011) and plantarflexors (Miyamoto et al. 2011) for up to 4 minutes following a conditioning activity, while H-reflex amplitude has been closely related to force at various time points post MVIC (Gullich and Schmidtbleicher 1996). This study is the first to statistically quantify TP and RP to overall PAP. Both did significantly contribute to overall PAP at each time point analyzed, however, their relative contributions were somewhat different than what was hypothesized based on previous research.

An interesting finding of our study is the high relative contribution of RP to RFD versus its contribution to torque. The coefficient of determination of the soleus to Hmax RFD declined progressively from 0.65 to 0.41 between the 20 second and 20 minute time points. These values are considerably higher than those for torque. TP was also responsible for more of the variance with torque than RFD. Together these results

suggest that neural factors play a more prominent role in RFD than in torque production, particularly in the near-term after a conditioning activity. There may be two contributing factors to this finding. First, conditioning activities result in phosphorylation of the myosin light chains (Moore and Stull 1984; Houston et al. 1985; Sweeney et al. 1993) which has been shown to increase calcium sensitivity (Metzger and Moss 1992) by moving the myosin head closer to the thin filament (Persechini et al. 1985; Sweeney and Stull 1986, 1990). Increased calcium sensitivity would lead to a greater EMG response to a stimulation at Hmax by increasing the number of actin and myosin attachments, increasing the Hmax/Mmax ratio. The second factor is likely related to the acute reflexive contribution to neural drive (Hodgson et al. 2005). A clearer understanding of the mechanisms responsible for these findings requires further research.

We expected the gastrocnemius to have a meaningful contribution due to its fiber type. However, its contribution to Hmax torque and RFD was not significant at any time point analyzed, in contrast to our hypothesis that it would contribute more to overall PAP than the soleus. The gastrocnemius is composed of mostly type II fibers (Johnson et al. 1973), which experience PAP to a greater degree than slow twitch fibers (Belanger and Mccomas 1985; Houston and Grange 1991; Hamada et al. 2000b). Like previous studies that utilized similar knee (Enoka et al. 1980; Gullich and Schmidtbleicher 1996) and ankle (Enoka et al. 1980; Gullich and Schmidtbleicher 1996; Trimble and Harp 1998) angles we successfully elicited M and H-waves in the gastrocnemius. The Hmax/Mmax ratios for the soleus and gastrocnemius were similar (Figures 4.8 and 4.9). However, as knee flexion increases from 180 degrees (i.e.: a straight-leg position) to 120 degrees the contribution of the gastrocnemius to isometric plantarflexion torque decreases (Cresswell

et al. 1995; Pinniger et al. 2000). It is possible that a greater contribution of the gastrocnemius to overall potentiation would have been observed if a more extended knee position was utilized, as was done in one other study (Trimble and Harp 1998). Measurements at a more extended knee position may also be readily transferred to dynamic movements such as jumping. Regardless of the relative contribution of the gastrocnemius and soleus within RP to overall potentiation, the contributions of TP and RP remains important.

Twitch torque and RFD at Mmax were both significantly higher in both experimental conditions vs. the control condition immediately after the MVIC. Twitch torque potentiation of approximately 38% to 50% in the triceps surae has been reported previously (Hamada et al. 2000a). This corresponds with the results of this study. In the quadriceps twitch torque potentiation after a conditioning activity has been shown to vary between 10.7% (Mitchell and Sale 2011) and 66.6% (Folland et al. 2008; Gossen and Sale 2000). Not surprisingly, the 10 second MVIC used in the present study showed significant potentiation of approximately 60% immediately after the conditioning activity. By 18 minutes post-MVIC both torque and RFD returned to baseline, being insignificantly elevated between 1.33% and 7.51%. One study reported twitch torque potentiation to last for up to 18 minutes following a conditioning activity (Folland et al. 2008), while others have taken measurements for only up to 10 minutes (Moore and Stull 1984; Houston and Grange 1990). The mechanism for the increased torque and RFD at Mmax are both likely myosin light chain phosphorylation, mentioned previously (Baudry and Duchateau 2007; Sale 2002). The number of cross bridge attachments at a given calcium concentration increases since the probability of myosin attaching to actin

increases as their separation distance decreases (MacIntosh 2003). More cross bridges would result in both more force and force being produced more quickly, enhancing both twitch torque and RFD.

It is well documented in the scientific literature that short-term depression of the H-reflex occurs in response to conditioning activities compromised of electrical stimulations (Crone and Nielsen 1989). Other studies have found PAD in the triceps surae after volitional muscle contractions (Trimble and Harp 1998; Enoka et al. 1980; Gullich and Schmidtbleicher 1996), followed by RP several minutes thereafter. Although in our study RP significantly contributed to overall PAP, there were no significant differences in either the gastrocnemius or soleus Hmax/Mmax ratios between conditions at any of the time points analyzed. This may partially explain why significance between conditions for measures of potentiation, mentioned earlier, were not found. Trimble and Harp (1998) reported PAD of the soleus and gastrocnemius for the first several minutes following dynamic plantarflexions and plantarextensions. However, they observed equivocal results regarding RP of the gastrocnemius and soleus, with some subjects showing RP in one muscle and not the other, starting at 3 minutes after the conditioning activity. Enoka et al. (1980) also reported a lack of RP in the soleus after isometric plantarflexions. In contrast, other authors have reported that the soleus H-reflex is highly related to torque depression and subsequent potentiation after tetanic nerve stimulation (Klakowicz et al. 2006) or volitional force (Gullich and Schmidtbleicher 1996) using lower extremity joint angles similar to those used in the present study. Because of the gastrocnemius' lack of contribution to overall PAP, shown in Table 4.3, any possible RP would be due to the soleus. As shown in Table 4.2, RP of the soleus was only marginally

and insignificantly enhanced in the MVIC condition and depressed in the LFF+MVIC condition, helping to explain why we did not observe significant PAP for torque or RFD at Hmax despite our results matching closely with the time course of the Hmax/Mmax ratio in the quadriceps after a 10 second MVIC (Folland et al. 2008). That is, overall PAP was slightly elevated above baseline and rose for several minutes before decreasing for the duration of testing.

The two experimental conditions were not statistically different from each other for any variable at any time point analyzed. The stimulation procedures utilized have successfully induced LFF in the triceps surae in previous studies (Shields and Chang 1997; Shields et al. 1997; Shields et al. 2006). That our subjects did not have their muscles paralyzed may have enabled muscular conditions to exist, such as spinal intervention, that did not allow LFF to occur (Chang and Shields 2011). The percent change for torque and RFD in twitches at Hmax were marginally, but consistently, less in the LFF+MVIC condition vs. the control compared to the MVIC condition. This, combined with the consistently lower Hmax/Mmax ratios for both the gastrocnemius and soleus in the LFF+MVIC condition compared to the MVIC condition, suggests that a small amount of central fatigue may have been elicited by the LFF protocol. At Mmax the torque and RFD were slightly higher in the LFF+MVIC condition. The LFF protocol may have resulted in phosphorylation that was additive to that experienced as a result of the MVIC (Fowles and Green 2003), though this is speculation as we did not test for phosphorylation of the myosin light chains.

Conclusions

This study was the first to statistically quantify the effects of TP and RP on overall PAP. Both TP and RP were significantly related to torque and RFD overall PAP for the duration of testing, with the soleus being almost exclusively responsible for RP over the gastrocnemius. Additionally, RP was more highly related to RFD potentiation than to torque potentiation, with the opposite being true for TP. Neural adaptations to power training include a greater ability to recruit motor units (Knight and Kamen 2001; Sale et al. 1983), preferential recruitment of type II muscle fibers (Moritani et al. 1991), and increased synchronization of motor units (del Olmo et al. 2006). Considering the relationship found between RFD and RP, athletes involved in sports where RFD is important, such as those that involve throwing, running, or jumping, could benefit from training methodologies designed to increase neural drive.

Conflict of Interest

The authors declare no conflict of interest.

Chapter 5: Summary, Conclusions, and Recommendations for Future Research

Summary

PAP has most often been measured as twitch potentiation (TP), defined as the muscle force or RFD produced in response to a relatively high intensity twitch that produces maximum muscle force, delivered by electrical stimulation (Gossen and Sale 2000; Vandenboom et al. 1995). However, this fails to account for the influence of the nervous system in muscle recruitment during volitional effort (Hamada et al. 2000b; Tillin and Bishop 2009; Klakowicz et al. 2006). TP is highest immediately after the conditioning activity (Vandervoort et al. 1983; Houston and Grange 1990; Requena et al. 2008) and quickly subsides (Houston and Grange 1990). The timing of RP better relates temporally to when PAP has been reported with volitional performance-oriented movements (Wilson et al. 2013). Thus, the overall purpose of this study was to explain the relationship between muscle and neural factors contributing to overall PAP (i.e.: torque and RFD at Hmax) (Folland et al. 2008).

To test this purpose a quasi-experimental repeated measures study that investigated measures of TP and RP to overall potentiation after a 10 second MVIC of the plantarflexors was conducted. The main hypotheses were: 1) TP would be apparent as a result of the MVIC for both torque and RFD, 2) the Hmax/Mmax ratio would be initially depressed before being potentiated, 3) the relationship between overall PAP torque, and TP and RP would be time dependent, 4) overall PAP RFD would follow the same temporal profile as overall PAP torque, and 5) the gastrocnemius would show more potentiation than the soleus. Fifteen resistance trained subjects (11 men, 4 women)

performed three testing sessions on separate days. The conditions were: Control, LFF+MVIC, and MVIC. Dependent variables were the measures of overall PAP, twitch torque and RFD at Mmax, and peak-to-peak EMG values for the gastrocnemius and soleus.

Results indicated that TP was achieved by the MVIC, however statistically significant RP was not. Both TP and RP were significantly related to torque and RFD through the duration of testing, with the soleus being almost exclusively responsible for RP over the gastrocnemius. Additionally, RP was more highly related to RFD than to torque, and TP less related to torque than to RFD.

Conclusions

This study is the first to statistically quantify TP and RP to overall PAP. The results indicate that both significantly contributed to overall PAP at each time point analyzed, with the soleus being almost exclusively responsible for RP compared to the gastrocnemius. Additionally, RP was more highly related to RFD potentiation than to torque potentiation, with the opposite being true for TP. Obtaining high RFD is more important than generating a high amount of peak force in many sports (Wilson et al. 1995). As a result many physical training programs are often designed with the goal of improving RFD through the use of plyometrics or Olympic weightlifting movements and their variants (Wallace and Janz 2009). The mechanisms of increased RFD as a result of targeted training could be a greater ability to recruit motor units (Knight and Kamen 2001; Sale et al. 1983), preferential recruitment of type II fibers (Moritani et al. 1991), or increased synchronization (del Olmo et al. 2006). This study has demonstrated that

potentiation of RFD is largely a consequence of neural factors, whereas torque potentiation is more a consequence of muscle factors. Therefore, this is further evidence that training programs should be designed according to the principle of specificity. Specifically, athletes involved in sports where RFD is important, such as those that involve throwing, running, or jumping, could benefit from training methodologies designed to increase neural drive.

Recommendations for Future Research

Although we obtained EMG responses from the gastrocnemius, its contribution to torque and RFD was minimal. To obtain a clearer understanding of the gastrocnemius' contribution to overall PAP future studies of the nature of the present one should be performed with a more extended knee position, and the foot placed in a slightly more plantarflexed position. This would place the gastrocnemius in a position more similar to what is experienced during the propulsive phase of *in-vivo* dynamic movements such as jumping. Future studies should also directly measure myosin light chain phosphorylation to gain a further understanding of the physiology involved with overall potentiation. Lastly, the relationship between the measures of isometric PAP, such as those utilized in this investigation, and dynamic PAP, requires further study. Even under the same physiological conditions muscles may behave different with dynamic movement (Gossen and Sale 2000).

Appendix A: Informed Consent form

Consent to Participate in a Research Study

Muscular and Neural Contributions to Postactivation Potentiation

WHY ARE YOU BEING INVITED TO TAKE PART IN THIS RESEARCH?

You are being invited to take part in a research study about postactivation potentiation (i.e.: a muscle being able to generate more force as a result of a recent contraction(s)) being conducted by Brian Wallace of the Department of Kinesiology and Health Promotion at the University of Kentucky. You are being invited to take part in this research study because you are an active individual of a relatively high training status. If you volunteer to take part in this study, you will be one of about 15-20 people to do so.

WHO IS DOING THE STUDY?

The person in charge of this study is Brian Wallace, MS, CSCS, of the Department of Kinesiology and Health Promotion at the University of Kentucky. He is being guided in this research by Robert Shapiro, Ph.D. Mr. Wallace is a Ph.D. candidate in Biomechanics, and Dr. Shapiro is a Professor. There may be other people on the research team assisting at different times during the study.

WHAT IS THE PURPOSE OF THIS STUDY?

The overall purpose of this study is to determine the magnitude and timing of muscular and neural factors to postactivation potentiation. It is hoped that this study will provide useful information to performance enhancement coaches, trainers, the lifting community, and sports athletes regarding the mechanisms responsible for postactivation potentiation.

ARE THERE REASONS WHY YOU SHOULD NOT TAKE PART IN THIS STUDY?

You should not take part in this study if you currently have a neuromuscular injury. You should not participate if you are not able to perform maximal contractions with the muscles of your lower extremity. You should not participate if you are not between the ages of 18 and 35. You also should not participate if you have not been engaged in a whole body resistance training program at least twelve times per week for at least the three previous months. If you are unable to verbally confirm that you are able to

maximally squat a mass equal to your bodyweight (females) or 1.5 times your bodyweight (males) you should not participate.

WHERE IS THE STUDY GOING TO TAKE PLACE AND HOW LONG WILL IT LAST?

The research procedures will be conducted in the research laboratory of Brock Symons, Ph.D. in the Graduate Center for Gerontology on the University of Kentucky campus. You will need to go to the laboratory three times during the study. Each testing session will last approximately 2 hours. The total amount of time you will be asked to volunteer for this study will be approximately 5-6 hours over a 2-4 week period.

WHAT WILL YOU BE ASKED TO DO?

When you arrive at the laboratory for the first testing day the investigation will be explained to you and you will have an opportunity to review and sign this consent form. If you agree to participate you will be required to wear typical workout clothing (specifically athletic shorts) for all three testing sessions. Each testing session will last approximately two hours. During all testing sessions you will be secured in an adjustable chair and have your right foot raised and secured to a foot plate with adjustable straps. You will have electrodes taped to your skin on your right leg that will allow certain muscles to be electrically stimulated, and their responses to those stimulations recorded. During the first testing session you will undergo electrical stimulations to the tibial nerve behind your knee that will contract your gastrocnemius and soleus (muscles on the back side of your lower leg). The intensity of these stimulations will vary and there will be rest periods (where no stimulations will be applied) of varying time periods (up to 20 minutes) during the testing session. You will be asked to put your maximum effort into plantarflexing (i.e.: pushing down with your toes) for 3-5 trials of 10 seconds. You will also undergo a stimulation protocol designed to induce fatigue into your muscles that will last approximately 3 minutes. This protocol has been used in other studies. Days 2 and 3 will be similar to Day 1 in terms of what stimulation procedures to expect, except you will only be maximally plantarflexing for 1 trial.

WHAT ARE THE POSSIBLE RISKS AND DISCOMFORTS?

To the best of our knowledge, the procedures you will be doing have no more risk of harm than you would experience while engaging in regular exercise or sports. The muscle stimulations could cause some temporary discomfort during the testing, or muscle soreness that will appear after the testing. There may be some discomfort and skin irritation due to the skin preparation (including shaving if necessary), use of conductive gel on the skin, and use of electrodes.

WILL YOU BENEFIT FROM TAKING PART IN THIS STUDY?

There is no guarantee that you will get any personal benefit from taking part in this study. Your willingness to take part, however, may help researchers, practitioners, and athletes better understand this topic.

DO YOU HAVE TO TAKE PART IN THE STUDY?

If you decide to take part in the study, it should be because you really want to volunteer. You will not lose any benefits or rights you would normally have if you choose not to volunteer. You can stop at any time during the study and still keep the benefits and rights you had before volunteering. If you are a student, if you decide not to take part in this study, your choice will have no effect on you academic status or grade in the class.

IF YOU DON'T WANT TO TAKE PART IN THE STUDY, ARE THERE OTHER CHOICES?

If you do not want to be in the study, there are no other choices except not to take part in the study.

WHAT WILL IT COST YOU TO PARTICIPATE?

There are no costs associated with taking part in the study.

WILL YOU RECEIVE ANY REWARDS FOR TAKING PART IN THIS STUDY?

You will not receive any rewards or payment for taking part in the study.

WHO WILL SEE THE INFORMATION THAT YOU GIVE?

Your information will be combined with information from other people taking part in the study. When we write about the study to share it with other researchers, we will write about the combined information we have gathered. You will not be identified in these written materials. We may publish the results of this study; however, we will keep your name and other identifying information private.

We will make every effort to prevent anyone who is not on the research team from knowing that you gave us information, or what that information is. For example, your name will be kept separate from the information you give, and these two things will be stored under lock and key. Any computer files of your data will be encoded as to not identify that the information is yours. However, there are some circumstances in which we may have to show your information to other people. For example, the law may require us to show your information to a court. Officials from the University of Kentucky may look at or copy pertinent portion of records that identify you.

CAN YOUR TAKING PART IN THE STUDY END EARLY?

If you decide to take part in the study you still have the right to decide at any time that you no longer want to continue. You will not be treated differently if you decide to stop taking part in the study. The individuals conducting the study may need to withdraw you from the study. This may occur if you are not able to follow the directions they give you, or if they find that your being in the study is more risk than benefit to you.

ARE YOU PARTICIPATING OR CAN YOU PARTICIPATE IN ANOTHER RESEARCH STUDY AT THE SAME TIME AS PARTICIPATING IN THIS ONE?

You may take part in this study if you are currently involved in another research study. It is important to let the investigator know if you are in another research study. You should also discuss with the investigator before you agree to participate in another research study while you are enrolled in this study.

WHAT HAPPENS IF YOU GET HURT OR SICK DURING THE STUDY?

If you believe you are hurt or if you get sick because of something that is due to the study, you should call Brian Wallace at 859-940-3691 immediately. Brian Wallace will determine what type of treatment, if any, that is best for you at that time.

It is important for you to understand that the University of Kentucky does not have funds set aside to pay for the cost of any care or treatment that might be necessary because you get hurt or sick while taking part in this study. Also, the University of Kentucky will not pay for any wages you may lose if you are harmed by this study.

Medical costs that result from research related harm can not be included as regular medical costs. Therefore, the medical costs related to your care and treatment because of research related harm will be your responsibility.

You do not give up your legal rights by signing this form.

WHAT IF YOU HAVE QUESTIONS, SUGGESTIONS, CONCERNS, OR COMPLAINTS?

Before you decide whether to accept this invitation to take part in the study, please ask any questions that might come to mind now. Later, if you have questions, suggestions, concerns, or complaints about the study, you can contact the principal investigator, Brian Wallace at brian.wallace@uky.edu or 859-940-3691. If you have any questions about your rights as a volunteer in this research, contact the staff in the Office of Research Integrity at the University of Kentucky at 859-257-9428 or toll free at 1-866-400-9428. We will give you a signed copy of this consent form to take with you.

WHAT IF NEW INFORMATION IS LEARNED DURING THE STUDY THAT MIGHT AFFECT YOUR DECISION TO PARTICIPATE?

If the researcher learns of new information in regards to this study, and it might change your willingness to stay in this study, the information will be provided to you. You may be asked to sign a new informed consent form if the information is provided to you after you have joined the study.

WHAT ELSE DO YOU NEED TO KNOW?

The University of Kentucky has supplied the equipment in the laboratory that will be used for the study.

_____ _____
Signature of person agreeing to take part in the study Date

_____
Printed name of person agreeing to take part in the study

_____ _____
Name of [authorized] person obtaining informed consent Date

Appendix B: Training and Health and Par-Q questionnaires

Training and Health Questionnaire

Subject name: _____Date: _____

Has your physician told you that you should not currently take part in strenuous exercise?

YES NO

Did you answer "yes" to any of the questions on the Par-Q questionnaire?

YES NO

Do you currently have any lower extremity neuromuscular injury?

YES NO

If YES, please describe: _____

_____

Have you ever had a major lower extremity injury OR neurological disorder?

YES NO

If YES, please describe: _____

_____

Have you been engaged continuously in a whole body resistance training program for at least the past twelve consecutive months?

YES NO

Can you maximally back squat a weight equal to your bodyweight (females) or 1.5 times your bodyweight (males)?

YES NO

If you were asked to kick a ball as far as possible, which leg would you use?

RIGHT LEFT

Please provide any other information that the researchers should know regarding your ability to participate in this research study: _____

_____

_____

_____

_____

Please sign and date below stating that you agree to the following:

I affirm that the information I provided above is truthful. I affirm that I am healthy enough to participate in this research study, and that I have no medical or other conditions that would make my participation unsafe. I also agree to notify the primary investigator, Brian Wallace, if any injuries or medical conditions arise during the time course of my participation in this study that may compromise my safe participation.

Subject signature: _____

Subject name (printed): _____

Date: _____

Physical Activity Readiness Questionnaire - PAR-Q (revised 2002)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO		
☐	☐	1.	**Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**
☐	☐	2.	**Do you feel pain in your chest when you do physical activity?**
☐	☐	3.	**In the past month, have you had chest pain when you were not doing physical activity?**
☐	☐	4.	**Do you lose your balance because of dizziness or do you ever lose consciousness?**
☐	☐	5.	**Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?**
☐	☐	6.	**Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**
☐	☐	7.	**Do you know of any other reason why you should not do physical activity?**

If you answered

YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active – begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal – this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

DELAY BECOMING MUCH MORE ACTIVE:

- If you are not feeling well because of a temporary illness such as a cold or a fever – wait until you feel better; or
- If you are or may be pregnant – talk to your doctor before you start becoming more active.

PLEASE NOTE: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

Informed Use of the PAR-Q: The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME _____

SIGNATURE _____ DATE _____

SIGNATURE OF PARENT _____ WITNESS _____
or GUARDIAN (for participants under the age of majority)

Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.

CSEP SCPE

Appendix C: Sample H and M wave EMG responses

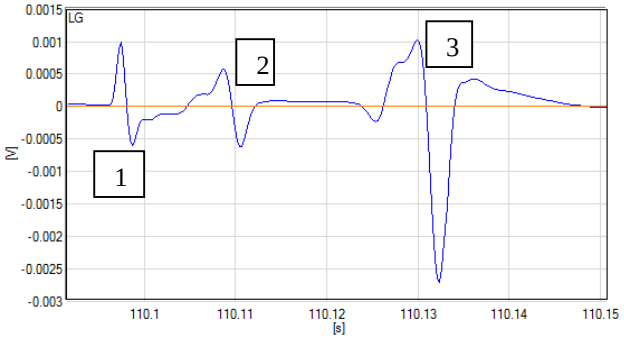

Figure C.1: Action potentials associated with the (1) electrical stimulation, (2) M-wave, and (3) H-wave in the lateral gastrocnemius in response to a submaximal twitch.

Appendix D: H-wave reliability

Table D.1: Intraclass correlation coefficient for H-wave amplitude (Hmax/Mmax) from H and M wave stimulus response curves between the three testing days for the gastrocnemius.

Intraclass Correlation Coefficient

	Intraclass Correlation	95% Confidence Interval		F Test with True Value 0			
		Lower Bound	Upper Bound	Value	df1	df2	Sig
Average Measures	.946	.873	.980	17.893	14	28	.000

Table D.2: Intraclass correlation coefficient for H-wave amplitude (Hmax/Mmax) from H and M wave stimulus response curves between the three testing days for the soleus.

Intraclass Correlation Coefficient

	Intraclass Correlation	95% Confidence Interval		F Test with True Value 0			
		Lower Bound	Upper Bound	Value	df1	df2	Sig
Average Measures	.914	.794	.969	10.955	14	28	.000

Appendix E: Matlab code for pulse generations

```
%% Creates a pulse train using a NI board named "Dev2".

%  This is for creating a H-M wave curve.  Start at 2 mA and go up to
%  50 mA on stimulator in increments of 2 mA.

%  Made with Matlab 2012b.  Brian Wallace.

%% Create a session object and save as variable "s". 'ni' is the hardware
%  vendor

s = daq.createSession('ni');

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Add a counter output channel with device named "Dev2", on channel 0,
%  and output measurement type called "pulse generation".  Add analog input
%  on channel 0 used to record and graph the counter output.

s.addAnalogInputChannel('Dev2', 0, 'Voltage');
s.addCounterOutputChannel('Dev2', 0, 'PulseGeneration');
s;

%% Determine the Terminal of the Counter Output Channel
% To connect the output signal to the correct terminal, examine the
% |Terminal| property of the counter channel. The terminal is determined by
% the hardware.

s.Channels(2).Terminal

for i=1:30;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 10 seconds, with a
% 1% duty cycle.

% Start at an intensity on the stimulator of 6 mA and go up to 50 mA
% in 2 mA increments.

% The "for" loop generates the train 1:n times ('n' being how many times).

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 10; % delay in seconds until generation starts
```

```
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 10.01;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end
s.release()



%% Creates a pulse train using a NI board named "Dev2".

%  This is for creating a H-M wave curve for the 5 intensities closest to
%  Hmas as determiend in the H_M_Curve_30 code.

%  Made with Matlab 2012b.  Brian Wallace.

%% Create a session object and save as variable "s". 'ni' is the hardware
%  vendor

s = daq.createSession('ni');

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Add a counter output channel with device named "Dev2", on channel 0,
%  and output measurement type called "pulse generation".  Add analog input
%  on channel 0 used to record and graph the counter output.

s.addAnalogInputChannel('Dev2', 0, 'Voltage');
s.addCounterOutputChannel('Dev2', 0, 'PulseGeneration');
```

```
s;

%% Determine the Terminal of the Counter Output Channel
% To connect the output signal to the correct terminal, examine the
% |Terminal| property of the counter channel. The terminal is determined by
% the hardware.

s.Channels(2).Terminal

for i=1:5;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 10 seconds, with a
% 1% duty cycle.

% Start at an intensity on the stimulator of 6 mA and go up to 50 mA.

% The "for" loop generates the train 1:n times ('n' being how many times).

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 10; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 10.01;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end
s.release()
```

```
%% Creates a pulse train using a NI board named "Dev2".
%  Made with Matlab 2012b.  Brian Wallace.


%% Create a session object and save as variable "s". 'ni' is the hardware
%  vendor

s = daq.createSession('ni');

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Add a counter output channel with device named "Dev2", on channel 0,
%  and output measurement type called "pulse generation".  Add analog input
%  on channel 0 used to record and graph the counter output.

s.addAnalogInputChannel('Dev2', 0, 'Voltage');
s.addCounterOutputChannel('Dev2', 0, 'PulseGeneration');
s;

%% Determine the Terminal of the Counter Output Channel
% To connect the output signal to the correct terminal, examine the
% |Terminal| property of the counter channel. The terminal is determined by
% the hardware.

s.Channels(2).Terminal

for i=1:5;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on terminal PFI0. Trigger after 1.2 seconds, with a
% 30% duty cycle.

ch = s.Channels(2);
ch.Frequency = 20;
ch.InitialDelay = 1.2;
ch.DutyCycle = 0.3;

% Set the length of generation/acquisition
s.DurationInSeconds = 1.5;

%% Rate (frequency) of analog input channel
s.Rate = 10000;

%%
```

```
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end
s.release()



%% Creates a pulse train using a NI board named "Dev2".
%  Made with Matlab 2012b.  Brian Wallace.


%% Create a session object and save as variable "s". 'ni' is the hardware
%  vendor

s = daq.createSession('ni');

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Add a counter output channel with device named "Dev2", on channel 0,
%  and output measurement type called "pulse generation".  Add analog input
%  on channel 0 used to record and graph the counter output.

s.addAnalogInputChannel('Dev2', 0, 'Voltage');
s.addCounterOutputChannel('Dev2', 0, 'PulseGeneration');
s;

%% Determine the Terminal of the Counter Output Channel
% To connect the output signal to the correct terminal, examine the
% |Terminal| property of the counter channel. The terminal is determined by
% the hardware.

s.Channels(2).Terminal

for i=1:120;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on terminal PFI0. Trigger after 1.2 seconds, with a
```

```
% 30% duty cycle.

ch = s.Channels(2);
ch.Frequency = 20;
ch.InitialDelay = 1.2;
ch.DutyCycle = 0.3;

% Set the length of generation/acquisition
s.DurationInSeconds = 1.5;

%% Rate (frequency) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end
s.release()



%% Creates a pulse train using a NI board named "Dev2".

%  This is for the Mmax and Hmax protocol following rest of the 10 second
%  MVIC.  This starts 10 seonds after and goes for 20 minutes.

%  The times for Mmax are: 10s, 30s, 1m, 2.5m, 4m, 6m, 8m, 10m, 12m, 14m,
%  16m, and 18m.
%  The times for Hmax are: 20s, 1.5m, 3m, 4.5m, 7m, 9m, 11m, 13m, 15m, 17m,
%  and 20m.
%  The combined times are: 10s, 20s, 30s, 1m, 1.5m, 2.5m, 3m, 4m, 4.5m, 6m,
%  7m, 8m, 9m, 10m, 11m, 12m, 13m, 14m, 15m, 16m, 17m, 18m, and 20m.

%  Made with Matlab 2012b.  Brian Wallace.

%% Create a session object and save as variable "s". 'ni' is the hardware
%  vendor
```

```
s = daq.createSession('ni');

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Add a counter output channel with device named "Dev2", on channel 0,
%  and output measurement type called "pulse generation".  Add analog input
%  on channel 0 used to record and graph the counter output.

s.addAnalogInputChannel('Dev2', 0, 'Voltage');
s.addCounterOutputChannel('Dev2', 0, 'PulseGeneration');
s;

%% Determine the Terminal of the Counter Output Channel
% To connect the output signal to the correct terminal, examine the
% |Terminal| property of the counter channel. The terminal is determined by
% the hardware.

s.Channels(2).Terminal

for i=1:3;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 10 seconds, with a
% 1% duty cycle.

% The "for" loop generates the train 1:n times ('n' being how many times).

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 10; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 10.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.
```

```
[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

for i=1:2;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 30 seconds, with a
% 1% duty cycle.

% The "for" loop generates the train 1:n times ('n' being how many times).

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 30; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 30.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()
```

```
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 60 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 60; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 60.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 30 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 30; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 30.0;
```

```
%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 60 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 60; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 60.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
```

```
%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 30 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 30; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 30.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 90 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 90; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.
```

```
% Set the total length of generation/acquisition
s.DurationInSeconds = 90.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

for i=1:12;
%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 60 seconds, with a
% 1% duty cycle.

% This handles the stmiulation every 1 minute from the 7 minute to the 18
% minute mark.

% The "for" loop generates the train 1:n times ('n' being how many times).

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 60; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 60.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
```

```
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);
end

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()

%% Clocked Counter Output
% Use counter output channel 0 to generate a fixed pulse width modulated
% signal on screw terminal PFI12. Trigger after 120 seconds, with a
% 1% duty cycle.

ch = s.Channels(2);
ch.Frequency = 1;  % frequency = 1 Hz
ch.InitialDelay = 120; % delay in seconds until generation starts
ch.DutyCycle = 0.01; % pulse width as a percent (1%) of frequency; in this
    % case 1% of 1 second, or 0.01 seconds.

% Set the total length of generation/acquisition
s.DurationInSeconds = 120.0;

%% Rate (frequency in Hz) of analog input channel
s.Rate = 10000;

%%
%  Start operations of the session object, "s", and blocks MATLAB command
%  line and other code until the sessionoperation is complete.

% StartForeground returns data for input channels only. The data variable
% will contain one column of data.

[data, time] = s.startForeground();

% Plot and display the acquisition of each train
plot(time, data);

%% Release the hardware.  Or, release the hounds, as Mr. Burns says.
s.release()
```

Appendix F: Matlab code for data analysis

```
%% Calculates peaks for series of twitches from text files of data exported
%  as text file from EMGWorks.

% Brian Wallace
% 5/6/2013

% Created with MatLab R2012b.

%%
close all;
clear all;

dire=fullfile('C:','RFD','keyfile.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);

%%
for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('C:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data

% Specify the files to be read in are tab '\t' delimited and 3 header lines
delimiterIn = '\t';
headerlinesIn = 3;

% Import the text files containing the data
wholefile = importdata(openname,delimiterIn,headerlinesIn);

% Define "alldata" from the "wholefile" structure
alldata=wholefile.data;

% Determine the length of the data column
length_data=length(alldata);

% Define 'torq_data_withTrend' as the whole 3rd column of "alldata" and convert to Nm
```

```
torq_data_withTrend=(alldata(:,3)*1.35582);

% Define trigger data (2nd column)
data_trigger=(alldata(:,2));

%% Detrend torque data (take out baseline) using a 6th order polynomial

[p,s,mu]=polyfit((1:numel(torq_data_withTrend))',torq_data_withTrend,6);
f_y=polyval(p,(1:numel(torq_data_withTrend))',[],mu);

% Define variable 'torq_data'
torq_data=torq_data_withTrend-f_y;

%% Find peaks of torque and trigger data

% Find torque peaks with a minimum amplitude of 3Nm
[torq_pks_vals, torq_pks_locs]= findpeaks(torq_data,'MINPEAKHEIGHT',3,
'MINPEAKDISTANCE',35000);

% Time of torque peaks in seconds from start of file (frame/FS)
torq_sec_pks= torq_pks_locs/4000;

% Find trigger peaks
[trig_pks_vals, trig_pks_locs]= findpeaks(data_trigger,'MINPEAKHEIGHT',3,
'MINPEAKDISTANCE',35000);

% Time of trigger peaks in seconds from start of file (frame/FS)
trig_sec_pks= trig_pks_locs/4000;

%%
% Change directory
cd('C:\RFD\Processed_Data');

% Write data to 'openname'
fprintf('%s\n', ['writing ' openname]);

%% Place data in spreadsheet

% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

% Write to excel file in separate sheets, transposing data
sheet=1;
xlswrite('Peaksdata.xlsx', torq_pks_vals', sheet, dataPlacement);

sheet=2;
```

```
xlswrite('Peaksdata.xlsx', torq_sec_pks', sheet, dataPlacement);

sheet=3;
xlswrite('Peaksdata.xlsx', trig_sec_pks', sheet, dataPlacement);

end



%% Calculates lower threshold time (LT) for series of twitches from
%  data exported as text file from EMGWorks.

% 'data*' refers to torque, 'data_trigger' refers to trigger data

% Brian Wallace
% 5/6/2013

% Created with MatLab R2012b.

%%
close all;
clear all;

dire=fullfile('C:','RFD','keyfile_test.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);

%%
for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('C:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data

% Specify the files to be read in are tab '\t' delimited and 3 header lines
delimiterIn = '\t';
headerlinesIn = 3;

% Import the text files containing the data
```

```
wholefile = importdata(openname,delimiterIn,headerlinesIn);

% Define "alldata" from the "wholefile" structure
alldata=wholefile.data;

% Determine the length of the data column
length_data=length(alldata);

% Define 'data_withTrend' as the whole third column of "alldata" and convert to Nm
data_withTrend=(alldata(:,3)*1.35582);

% Define trigger data (2nd column)
data_trigger=(alldata(:,2));

%% Detrend the data (take out baseline) using a 6th order polynomial

[p,s,mu]=polyfit((1:numel(data_withTrend))',data_withTrend,6);
f_y=polyval(p,(1:numel(data_withTrend))',[],mu);

% Define variable 'data'
data=data_withTrend-f_y;

%% Find lower times for torque, and times of trigger

% Risetime function, sample frequency (FS) set to 4000,
% tolerance level set to 9.99, reference levels LL=20 UL=55
% LT = time signal crosses lower threshold
FS=4000;
[R,LT,UT]=risetime(data,FS,'tolerance',9.99,'PctRefLevels',[20 55]);

% Find trigger peaks
[trig_pks_vals, trig_pks_locs]= findpeaks(data_trigger,'MINPEAKHEIGHT',3,
'MINPEAKDISTANCE',35000);

% Time of trigger peaks in seconds from start of file (frame/FS)
trig_sec_pks= trig_pks_locs/4000;

%%
% Change directory
cd('C:\RFD\Processed_Data');

% Write data to 'openname'
fprintf('%s\n', ['writing ' openname]);

%% Place data in spreadsheet
```

```
% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

% Write to excel file, transposing data
sheet=1;
xlswrite('LowerTimedata.xlsx', LT', sheet, dataPlacement);

sheet=2;
xlswrite('LowerTimedata.xlsx', trig_sec_pks', sheet, dataPlacement);

end



%% Calculates RFD for series of twitches from text files of data exported
%  as text file from EMGWorks.

% Brian Wallace
% 5/6/2013

% Created with MatLab R2012b.

%%
close all;
clear all;

dire=fullfile('C:','RFD','keyfile.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);

%% Calculate RFD for stimulations

for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('C:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data

% Specify the files to be read in are tab '\t' delimited and 3 header lines
delimiterIn = '\t';
```

```
headerlinesIn = 3;

% Import the text files containing the data
wholefile = importdata(openname,delimiterIn,headerlinesIn);

% Define "alldata" from the "wholefile" structure
alldata=wholefile.data;

% Determine the length of the data column
length_data=length(alldata);

% Define 'data_withTrend' as the whole third column of "alldata" and convert to Nm
data_withTrend=(alldata(:,3)*1.35582);

%% Detrend the data (take out baseline) using a 6th order polynomial

[p,s,mu]=polyfit((1:numel(data_withTrend))',data_withTrend,6);
f_y=polyval(p,(1:numel(data_withTrend))',[],mu);

% Define variable 'data'
data=data_withTrend-f_y;

%% Find peaks and RFD

% Find the peaks with a minimum amplitude of 7Nm
[pks_value,locs_pks]= findpeaks(data,'MINPEAKHEIGHT',7,
'MINPEAKDISTANCE',35000);

% Time of peaks in seconds from start of file (frame/FS)
sec_of_pks=locs_pks/4000;

% Risetime function, sample frequency (FS) set to 4000,
% tolerance level set to 9.99, reference levels LL=20 UL=55
FS=4000;
[R,LT,UT]=risetime(data,FS,'tolerance',19.99,'PctRefLevels',[20 55]);

% Calculate time from when force exceeds 10% threshold, to peak
delta_time=sec_of_pks-LT;

% Calculates average RFD from 10% threshold to peak, in Nm/s
RFD=pks_value./delta_time;

%%
%change directory
cd('C:\RFD\Processed_Data');
```

```
%write data to 'openname'
fprintf('%s\n', ['writing' openname]);

%% Place data in spreadsheet

% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

%write to excel file, transposing RFD data
sheet=1;
xlswrite('RFDdata.xlsx', RFD', sheet, dataPlacement);

end



%% Reads in Excel files and creates files for each variable for each
% condition.

% Brian Wallace
% 5/10/2013

% Created with MatLab R2012b.

%%
close all;
clear all;

dire=fullfile('E:','EditedFiles','keyfile_d1.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);

%% Calculate RFD for stimulations

for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('E:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data
```

```
% Import the ranges for different variables

sheet=1;

xlrange='H3:H13';
Htime=xlsread(openname,sheet,xlrange);

xlrange='I3:I13';
GasHmaxDivMmax = xlsread(openname,sheet,xlrange);

xlrange='J3:J13';
SolHmaxDivMmax=xlsread(openname,sheet,xlrange);

xlrange='K3:K13';
GasHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

xlrange='L3:L13';
SolHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

%%
%change directory
cd('E:\EditedFiles\Processed_Data\day1');

%write data to 'openname'
fprintf('%s\n',['writing' openname]);

%% Place data in spreadsheet

% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

%write to excel file, transposing RFD data

sheet=1;
xlrange='B1';

xlswrite('GasHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax.xlsx',GasHmaxDivMmax',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('SolHmaxDivMmax.xlsx',SolHmaxDivMmax',sheet,dataPlacement);

xlswrite('GasHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax_avg.xlsx',GasHmaxDivMmax_avg',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
```

```
xlswrite('SolHmaxDivMmax_avg.xlsx',SolHmaxDivMmax_avg',sheet,dataPlacement);

end



%%
close all;
clear all;

dire=fullfile('E:','EditedFiles','keyfile_LFF.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);

%% Calculate RFD for stimulations

for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('E:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data

% Import the ranges for different variables

sheet=1;

xlrange='H3:H13';
Htime=xlsread(openname,sheet,xlrange);

xlrange='I3:I13';
GasHmaxDivMmax = xlsread(openname,sheet,xlrange);

xlrange='J3:J13';
SolHmaxDivMmax=xlsread(openname,sheet,xlrange);

xlrange='K3:K13';
GasHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

xlrange='L3:L13';
```

```
SolHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

%%
%change directory
cd('E:\EditedFiles\Processed_Data\LFF');

%write data to 'openname'
fprintf('%s\n',['writing' openname]);

%% Place data in spreadsheet

% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

%write to excel file, transposing RFD data

sheet=1;
xlrange='B1';

xlswrite('GasHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax.xlsx',GasHmaxDivMmax',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('SolHmaxDivMmax.xlsx',SolHmaxDivMmax',sheet,dataPlacement);

xlswrite('GasHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax_avg.xlsx',GasHmaxDivMmax_avg',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
xlswrite('SolHmaxDivMmax_avg.xlsx',SolHmaxDivMmax_avg',sheet,dataPlacement);

end



%%
close all;
clear all;

dire=fullfile('E:','EditedFiles','keyfile_nonLFF.txt') %keyfile.txt generated with dir >
%keyfile.txt reads off the drive

[filedir, subdir, subdir2, subdir3]=textread(dire,'%s %s %s %s');

%Determines the number of files in the folder that will be read in
numfiles = length(subdir3);
```

```
%% Calculate RFD for stimulations

for i=1:numfiles;

% Combines file path to make file name "openname"
% char converts a string cell to a character array
openname=fullfile('E:\',char(subdir(i)), char(subdir2(i)), char(subdir3(i)));

%% Import data

% Import the ranges for different variables

sheet=1;

xlrange='H3:H13';
Htime=xlsread(openname,sheet,xlrange);

xlrange='I3:I13';
GasHmaxDivMmax = xlsread(openname,sheet,xlrange);

xlrange='J3:J13';
SolHmaxDivMmax=xlsread(openname,sheet,xlrange);

xlrange='K3:K13';
GasHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

xlrange='L3:L13';
SolHmaxDivMmax_avg=xlsread(openname,sheet,xlrange);

%%
%change directory
cd('E:\EditedFiles\Processed_Data\nonLFF');

%write data to 'openname'
fprintf('%s\n',['writing' openname]);

%% Place data in spreadsheet

% i means to place the data in the "i"th row (one currently in use)
dataPlacement=['B' num2str(i+1)];

%write to excel file, transposing RFD data

sheet=1;
xlrange='B1';
```

```
xlswrite('GasHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax.xlsx',GasHmaxDivMmax',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax.xlsx',Htime',sheet,xlrange);
xlswrite('SolHmaxDivMmax.xlsx',SolHmaxDivMmax',sheet,dataPlacement);

xlswrite('GasHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
xlswrite('GasHmaxDivMmax_avg.xlsx',GasHmaxDivMmax_avg',sheet,dataPlacement);

xlswrite('SolHmaxDivMmax_avg.xlsx',Htime',sheet,xlrange);
xlswrite('SolHmaxDivMmax_avg.xlsx',SolHmaxDivMmax_avg',sheet,dataPlacement);

end



%% This script creates figures for Mmax Variables (MRFD and Mtorque).

% Created with Matlab R2013b.

% Brian Wallace

% Created June 14, 2014

%% Create Figures for Mmax variables

% Change directory
cd('C:/Data');

% Identify filename
filename='M_Matlab.xlsx';

% Read in MRFD and Mtorque data

% Create time variable
time=xlsread(filename,'Mtorque_d1','B2:B13');
time=time';

d1_MRFD_data=xlsread(filename,'MRFD_d1','a2:L16');
LFF_MRFD_data=xlsread(filename,'MRFD_LFF','a2:L16');
nonLFF_MRFD_data=xlsread(filename,'MRFD_nonLFF','a2:L16');

d1_Mtorque_data=xlsread(filename, 'Mtorque_d1','c2:n16');
```

```
LFF_Mtorque_data=xlsread(filename, 'Mtorque_LFF','a2:L16');
nonLFF_Mtorque_data=xlsread(filename, 'Mtorque_nonLFF','a2:L16');

% Get averages for data
avg_MRFD_d1=nanmean(d1_MRFD_data);
avg_MRFD_LFF=nanmean(LFF_MRFD_data);
avg_MRFD_nonLFF=nanmean(nonLFF_MRFD_data);

avg_Mtorque_d1=nanmean(d1_Mtorque_data);
avg_Mtorque_LFF=nanmean(LFF_Mtorque_data);
avg_Mtorque_nonLFF=nanmean(nonLFF_Mtorque_data);

% Get standard deviations for data
SD_MRFD_d1=nanstd(d1_MRFD_data);
SD_MRFD_LFF=nanstd(LFF_MRFD_data);
SD_MRFD_nonLFF=nanstd(nonLFF_MRFD_data);

SD_Mtorque_d1=nanstd(d1_Mtorque_data);
SD_Mtorque_LFF=nanstd(LFF_Mtorque_data);
SD_Mtorque_nonLFF=nanstd(nonLFF_Mtorque_data);

%% Create plots

% Create lower and upper error bar boundaries for MRFD
L_d1_MRFD=avg_MRFD_d1-SD_MRFD_d1;
U_LFF_MRFD=avg_MRFD_LFF+SD_MRFD_LFF;
U_nonLFF_MRFD=avg_MRFD_nonLFF+SD_MRFD_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0,0];
U=[0,0,0,0,0,0,0,0,0,0,0,0];

% Create MRFD Figure
figure (1)
clf

errorbar(time,avg_MRFD_d1,L_d1_MRFD,U,'-sk')
hold on
errorbar(time,avg_MRFD_LFF,L,U_LFF_MRFD,'-dg')
hold on
errorbar(time,avg_MRFD_nonLFF,L,U_nonLFF_MRFD,'-om')

% Label the axis
xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Twitch RFD at Mmax (Nm/s)','fontsize',12,'FontName','times new roman')
hleg1=legend('Control','LFF+MVIC','MVIC');
set(hleg1,'fontname','times new roman');
axis tight
```

```
axis 'auto y'

% Create lower and upper error bar boundaries for Mtorque
L_d1_Mtorque=avg_Mtorque_d1-SD_Mtorque_d1;
U_LFF_Mtorque=avg_Mtorque_LFF+SD_Mtorque_LFF;
U_nonLFF_Mtorque=avg_Mtorque_nonLFF+SD_Mtorque_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0,0];
U=[0,0,0,0,0,0,0,0,0,0,0,0];

% Create Mtorque Figure
figure (2)
clf

errorbar(time,avg_Mtorque_d1,L_d1_Mtorque,U,'-sk')
hold on
errorbar(time,avg_Mtorque_LFF,L,U_LFF_Mtorque,'-dg')
hold on
errorbar(time,avg_Mtorque_nonLFF,L,U_nonLFF_Mtorque,'-om')

xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Twitch Torque at Mmax (Nm)','fontsize',12,'FontName','times new roman')
hleg2=legend('Control','LFF+MVIC','MVIC');
set(hleg2,'fontname','times new roman');
axis tight
axis 'auto y'



%% This script creates figures for Hmax Variables (HRFD and Htorque).

% Created with Matlab R2013b.

% Brian Wallace

% Created June 15, 2014

%% Create Figures for Hmax variables

% Change directory
cd('C:/Data');

% Identify filename
filename='H_Matlab.xlsx';

% Read in MRFD and Mtorque data

% Create time variable
```

```
time=xlsread(filename,'HRFD_d1','C2:C12');
time=time';

d1_HRFD_data=xlsread(filename,'HRFD_d1','D2:N16');
LFF_HRFD_data=xlsread(filename,'HRFD_LFF','C2:M16');
nonLFF_HRFD_data=xlsread(filename,'HRFD_nonLFF','C2:M16');

d1_Htorque_data=xlsread(filename, 'Htorque_d1','C2:M16');
LFF_Htorque_data=xlsread(filename, 'Htorque_LFF','C2:M16');
nonLFF_Htorque_data=xlsread(filename, 'Htorque_nonLFF','C2:M16');

% Get averages for data
avg_HRFD_d1=nanmean(d1_HRFD_data);
avg_HRFD_LFF=nanmean(LFF_HRFD_data);
avg_HRFD_nonLFF=nanmean(nonLFF_HRFD_data);

avg_Htorque_d1=nanmean(d1_Htorque_data);
avg_Htorque_LFF=nanmean(LFF_Htorque_data);
avg_Htorque_nonLFF=nanmean(nonLFF_Htorque_data);

% Get standard deviations for data
SD_HRFD_d1=nanstd(d1_HRFD_data);
SD_HRFD_LFF=nanstd(LFF_HRFD_data);
SD_HRFD_nonLFF=nanstd(nonLFF_HRFD_data);

SD_Htorque_d1=nanstd(d1_Htorque_data);
SD_Htorque_LFF=nanstd(LFF_Htorque_data);
SD_Htorque_nonLFF=nanstd(nonLFF_Htorque_data);

%% Create plots

% Create lower and upper error bar boundaries for HRFD
L_d1_HRFD=avg_HRFD_d1-SD_HRFD_d1;
U_LFF_HRFD=avg_HRFD_LFF+SD_HRFD_LFF;
U_nonLFF_HRFD=avg_HRFD_nonLFF+SD_HRFD_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0];
U=[0,0,0,0,0,0,0,0,0,0,0];

% Create HRFD Figure
figure (1)
clf

errorbar(time,avg_HRFD_d1,L_d1_HRFD,U,'-sk')
hold on
errorbar(time,avg_HRFD_LFF,L,U_LFF_HRFD,'-dg')
hold on
```

```
errorbar(time,avg_HRFD_nonLFF,L,U_nonLFF_HRFD,'-om')

% Label the axis
xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Twitch RFD at Hmax (Nm/s)','fontsize',12,'FontName','times new roman')
hleg1=legend('Control','LFF+MVIC','MVIC');
set(hleg1,'fontname','times new roman');
axis tight
axis 'auto y'

% Create lower and upper error bar boundaries for Htorque
L_d1_Htorque=avg_Htorque_d1-SD_Htorque_d1;
U_LFF_Htorque=avg_Htorque_LFF+SD_Htorque_LFF;
U_nonLFF_Htorque=avg_Htorque_nonLFF+SD_Htorque_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0];
U=[0,0,0,0,0,0,0,0,0,0,0];

% Create Htorque Figure
figure (2)
clf

errorbar(time,avg_Htorque_d1,L_d1_Htorque,U,'-sk')
hold on
errorbar(time,avg_Htorque_LFF,L,U_LFF_Htorque,'-dg')
hold on
errorbar(time,avg_Htorque_nonLFF,L,U_nonLFF_Htorque,'-om')

xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Twitch Torque at Hmax (Nm)','fontsize',12,'FontName','times new roman')
hleg2=legend('Control','LFF+MVIC','MVIC');
set(hleg2,'fontname','times new roman');
axis tight
axis 'auto y'



%% This script creates figures for EMG (Gas and Sol) Variables.

% Created with Matlab R2013b.

% Brian Wallace

% Created June 15, 2014

%% Create Figures for Gas and Sol variables
```

```
% Change directory
cd('C:/Data');

% Identify filename
filename='GasSol_Matlab.xlsx';

% Read in Gas and Sol data

% Create time variable
time=xlsread(filename,'Gas_d1','C2:C12');
time=time';

d1_Gas_data=xlsread(filename,'Gas_d1','D2:N16');
LFF_Gas_data=xlsread(filename,'Gas_LFF','C2:M16');
nonLFF_Gas_data=xlsread(filename,'Gas_nonLFF','C2:M16');

d1_Sol_data=xlsread(filename, 'Sol_d1','C2:M16');
LFF_Sol_data=xlsread(filename, 'Sol_LFF','C2:M16');
nonLFF_Sol_data=xlsread(filename, 'Sol_nonLFF','C2:M16');

% Get averages for data
avg_Gas_d1=nanmean(d1_Gas_data);
avg_Gas_LFF=nanmean(LFF_Gas_data);
avg_Gas_nonLFF=nanmean(nonLFF_Gas_data);

avg_Sol_d1=nanmean(d1_Sol_data);
avg_Sol_LFF=nanmean(LFF_Sol_data);
avg_Sol_nonLFF=nanmean(nonLFF_Sol_data);

% Get standard deviations for data
SD_Gas_d1=nanstd(d1_Gas_data);
SD_Gas_LFF=nanstd(LFF_Gas_data);
SD_Gas_nonLFF=nanstd(nonLFF_Gas_data);

SD_Sol_d1=nanstd(d1_Gas_data);
SD_Sol_LFF=nanstd(LFF_Sol_data);
SD_Sol_nonLFF=nanstd(nonLFF_Sol_data);

%% Create plots

% Create lower and upper error bar boundaries for Sol
L_d1_Gas=avg_Gas_d1-SD_Gas_d1;
U_LFF_Gas=avg_Gas_LFF+SD_Gas_LFF;
U_nonLFF_Gas=avg_Gas_nonLFF+SD_Gas_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0];
```

```
U=[0,0,0,0,0,0,0,0,0,0,0];

% Create Gas Figure
figure (1)
clf

errorbar(time,avg_Gas_d1,L_d1_Gas,U,'-sk')
hold on
errorbar(time,avg_Gas_LFF,L,U_LFF_Gas,'-dg')
hold on
errorbar(time,avg_Gas_nonLFF,L,U_nonLFF_Gas,'-om')

% Label the axis
xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Gastrocnemius Hmax/Mmax Ratio (%)','fontsize',12,'FontName','times new
roman')
hleg1=legend('Control','LFF+MVIC','MVIC');
set(hleg1,'fontname','times new roman');
axis tight
axis 'auto y'

% Create lower and upper error bar boundaries for Sol
L_d1_Sol=avg_Sol_d1-SD_Sol_d1;
U_LFF_Sol=avg_Sol_LFF+SD_Sol_LFF;
U_nonLFF_Sol=avg_Sol_nonLFF+SD_Sol_nonLFF;
L=[0,0,0,0,0,0,0,0,0,0,0];
U=[0,0,0,0,0,0,0,0,0,0,0];

% Create Sol Figure
figure (2)
clf

errorbar(time,avg_Sol_d1,L_d1_Sol,U,'-sk')
hold on
errorbar(time,avg_Sol_LFF,L,U_LFF_Sol,'-dg')
hold on
errorbar(time,avg_Sol_nonLFF,L,U_nonLFF_Sol,'-om')

xlabel('Time (min)','fontsize',12,'FontName','times new roman')
ylabel('Soleus Hmax/Mmax Ratio (%)','fontsize',12,'FontName','times new roman')
hleg2=legend('Control','LFF+MVIC','MVIC');
set(hleg2,'fontname','times new roman');
axis tight
axis 'auto y'
```

References

Aagaard P, Simonsen EB, Andersen JL, Magnusson P, Dyhre-Poulsen P (2002) Neural adaptation to resistance training: changes in evoked V-wave and H-reflex responses. Journal of Applied Physiology 92 (6):2309-2318

Abbate F, Sargeant AJ, Verdijk PW, de Haan A (2000) Effects of high-frequency initial pulses and posttetanic potentiation on power output of skeletal muscle. J Appl Physiol 88 (1):35-40

Allen DG, Lamb GD, Westerblad H (2008) Skeletal muscle fatigue: Cellular mechanisms. Physiol Rev 88 (1):287-332

Alway SE, Hughson RL, Green HJ, Patla AE, Frank JS (1987) Twitch potentiation after fatiguing exercise in man. European Journal of Applied Physiology and Occupational Physiology 56 (4):461-466

Batista MA, Roschel H, Barroso R, Ugrinowitsch C, Tricoli V (2011) Influence of strength training background on postactivation potentiation response. Journal of Strength & Conditioning Research 25 (9):2496-2502

Baudry S, Duchateau J (2007) Postactivation potentiation in a human muscle: Effect on the rate of torque development of tetanic and voluntary isometric contractions. Journal of Applied Physiology 102 (4):1394-1401

Baudry S, Klass M, Duchateau J (2005) Postactivation potentiation influences differently the nonlinear summation of contractions in young and elderly adults. Journal of Applied Physiology 98 (4):1243-1250

Baudry S, Klass M, Duchateau J (2008) Postactivation potentiation of short tetanic contractions is differently influenced by stimulation frequency in young and elderly adults. European Journal of Applied Physiology 103 (4):449-459

Behm DG, Button DC, Barbour G, Butt JC, Young WB (2004) Conflicting effects of fatigue and potentiation on voluntary force. Journal of Strength & Conditioning Research 18 (2):365-372

Belanger AY, Mccomas AJ (1985) A comparison of contractile properties in human arm and leg muscles. European Journal of Applied Physiology and Occupational Physiology 54 (3):326-330

Belanger AY, McComas AJ, Elder GB (1983) Physiological properties of two antagonist human muscle groups. Eur J Appl Physiol Occup Physiol 51 (3):381-393

Bergquist AJ, Clair JM, Collins DF (2011) Motor unit recruitment when neuromuscular electrical stimulation is applied over a nerve trunk compared with a muscle belly: triceps surae. J Appl Physiol 110 (3):627-637

Berning JM, Adams KJ, DeBeliso M, Sevene-Adams PG, Harris C, Stamford BA (2010) Effect of Functional Isometric Squats on Vertical Jump in Trained and Untrained Men. J Strength Cond Res 24 (9):2285-2289

Binder-Macleod SA, Dean JC, Ding J (2002) Electrical stimulation factors in potentiation of human quadriceps femoris. Muscle & Nerve 25 (2):271-279

Blumenthal DK, Stull JT (1980) Activation of skeletal-muscle myosin light chain kinase by calcium(2+) and calmodulin. Biochemistry-Us 19 (24):5608-5614

Bowman BF, Peterson JA, Stull JT (1992) Proposed mechanism of activation of rabbit skeletal-muscle myosin light chain kinase by Ca2+/calmodulin. Faseb J 6 (1):A436-A436

Brinkworth RSA, Tuncer M, Tucker KJ, Jaberzadeh S, Turker KS (2007) Standardization of H-reflex analyses. J Neurosci Meth 162 (1-2):1-7

Bruton JD, Lannergren J, Westerblad H (1998) Mechanisms underlying the slow recovery of force after fatigue: importance of intracellular calcium. Acta Physiol Scand 162 (3):285-293

Calder KM, Hall LA, Lester SM, Inglis JG, Gabriel DA (2005) Reliability of the biceps brachii M-wave. Journal of neuroengineering and rehabilitation 2:33

Camen G, Gabriel D (2010) Essentials of Electromyography. Human Kinetics, Champaign, IL

Chang YJ, Shields RK (2011) Doublet electrical stimulation enhances torque production in people with spinal cord injury. Neurorehabil Neural Repair 25 (5):423-432

Chen YS, Zhou S, Cartwright C, Crowley Z, Baglin R, Wang F (2010) Test-retest reliability of the soleus H-reflex is affected by joint positions and muscle force levels. J Electromyogr Kinesiol 20 (5):980-987

Chiu LZF, Fry AC, Weiss LW, Schilling BK, Brown LE, Smith SL (2003) Postactivation potentiation response in athletic and recreationally trained individuals. Journal of Strength and Conditioning Research 17 (4):671-677

Christie A, Lester S, LaPierre D, Gabriel DA (2004) Reliability of a new measure of H-reflex excitability. Clin Neurophysiol 115 (1):116-123

Cornu C, Silveira MIA, Goubel F (1997) Influence of plyometric training on the mechanical impedance of the human ankle joint. European Journal of Applied Physiology and Occupational Physiology 76 (3):282-288

Corrie WS, Hardin WB, Jr. (1964) Post-tetanic potentiation of H-reflex in horman man: Quantitative study. Archives Of Neurology 11:317-323

Cresswell AG, Loscher WN, Thorstensson A (1995) Influence of gastrocnemius muscle length on triceps surae torque development and electromyographic activity in man. Exp Brain Res 105 (2):283-290

Crewther BT, Kilduff LP, Cook CJ, Middleton MK, Bunce PJ, Yang G-Z (2011) The acute potentiating effects of back squats on athlete performance. Journal Of Strength And Conditioning Research 25 (12):3319-3325

Crone C, Nielsen J (1989) Methodological implications of the post activation depression of the soleus H-reflex in man. Exp Brain Res 78 (1):28-32

del Olmo MF, Reimunde P, Viana O, Acero RM, Cudeiro J (2006) Chronic neural adaptation induced by long-term resistance training in humans. Eur J Appl Physiol 96 (6):722-728

Docherty D, Hodgson MJ (2007) The application of postactivation potentiation to elite sport. Intj Sport Physiol 2 (4):439-444

Docherty D, Robbins D, Hodgson M (2004) Complex training revisited: A review of its current status as a viable training approach. Strength & Conditioning Journal 26 (6):52-57

Enoka RM, Hutton RS, Eldred E (1980) Changes in excitability of tendon tap and Hoffmann reflexes following voluntary contractions. Electroencephalography And Clinical Neurophysiology 48 (6):664-672

Esformes JI, Cameron N, Bampouras TM (2010) Postactivation potentiation following different modes of exercise. Journal of Strength & Conditioning Research (Lippincott Williams & Wilkins) 24 (7):1911-1916

Folland JP, Wakamatsu T, Fimland MS (2008) The influence of maximal isometric activity on twitch and H-reflex potentiation, and quadriceps femoris performance. European Journal of Applied Physiology 104 (4):739-748

Ford KR, Myer GD, Hewett TE (2003) Valgus knee motion during landing in high school female and male basketball players. Medicine and Science in Sports and Exercise 35 (10):1745-1750

Fowles JR, Green HJ (2003) Coexistence of potentiation and low-frequency fatigue during voluntary exercise in human skeletal muscle. Canadian Journal of Physiology & Pharmacology 81 (12):1092-1100

French DN, Kraemer WJ, Cooke CB (2003) Changes in dynamic exercise performance following a sequence of preconditioning isometric muscle actions. J Strength Cond Res 17 (4):678-685

Gollhofer A, Schöpp A, Rapp W, Stroinik V (1998) Changes in reflex excitability following isometric contraction in humans. European Journal Of Applied Physiology And Occupational Physiology 77 (1-2):89-97

Gossen ER, Sale DG (2000) Effect of postactivation potentiation on dynamic knee extension performance. European Journal Of Applied Physiology 83 (6):524-530

Gourgoulis V, Aggeloussis N, Kasimatis P, Mavromatis G, Garas A (2003) Effect of a submaximal half-squats warm-up program on vertical jumping ability. J Strength Cond Res 17 (2):342-344

Grange RW, Cory CR, Vandenboom R, Houston ME (1995) Myosin phosphorylation augments force-displacement and force-velocity relationships of mouse fast muscle. Am J Physiol 269 (3 Pt 1):C713-724

Grange RW, Houston ME (1991) Simultaneous potentiation and fatigue in quadriceps after a 60-second maximal voluntary isometric contraction. J Appl Physiol 70 (2):726-731

Grange RW, Vandenboom R, Houston ME (1993) Physiological significance of myosin phosphorylation in skeletal muscle. Canadian Journal of Applied Physiology 18 (3):229-242

Grospretre S, Martin A (2012) H reflex and spinal excitability: methodological considerations. Journal of neurophysiology 107 (6):1649-1654

Gullich A, Schmidtbleicher D (1996) MVC-induced short-term potentiation of explosive force. New Studies in Athletics 11 (4):67-81

Haff GG, Stone M, OBryant HS, Harman E, Dinan C, Johnson R, Han KH (1997) Force-time dependent characteristics of dynamic and isometric muscle actions. J Strength Cond Res 11 (4):269-272

Hamada T, Sale DG, Macdougall JD (2000a) Postactivation potentiation in endurance-trained male athletes. Med Sci Sports Exerc 32 (2):403-411

Hamada T, Sale DG, MacDougall JD, Tarnopolsky MA (2000b) Postactivation potentiation, fiber type, and twitch contraction time in human knee extensor muscles. Journal Of Applied Physiology 88 (6):2131-2137

Hamada T, Sale DG, MacDougall JD, Tarnopolsky MA (2003) Interaction of fibre type, potentiation and fatigue in human knee extensor muscles. Acta Physiol Scand 178 (2):165-173

Hicks A, Fenton J, Garner S, McComas AJ (1989) M-wave potentiation during and after muscle activity. J Appl Physiol 66 (6):2606-2610

Hodgson M, Docherty D, Robbins D (2005) Post-Activation Potentiation. Sports Medicine 35 (7):585-595

Hopkins JT, Wagie NC (2003) Intrasession and intersession reliability of the quadriceps Hoffmann reflex. Electromyography and Clinical Neurophysiology 43 (2):85-89

Houston ME, Grange RW (1990) Myosin phosphorylation, twitch potentiation, and fatigue in human skeletal muscle. Can J Physiol Pharmacol 68 (7):908-913

Houston ME, Grange RW (1991) Torque potentiation and myosin light-chain phosphorylation in human muscle following a fatiguing contraction. Can J Physiol Pharmacol 69 (2):269-273

Houston ME, Green HJ, Stull JT (1985) Myosin light chain phosphorylation and isometric twitch potentiation in intact human muscle. Pflügers Archiv: European Journal of Physiology 403 (4):348-352

Houston ME, Lingley MD, Stuart DS, Grange RW (1987) Myosin light chain phosphorylation in intact human muscle. FEBS Letters 219 (2):469-471

Hubscher M, Zech A, Pfeifer K, Hansel F, Vogt L, Banzer W (2010) Neuromuscular training for sports injury prevention: A systematic review. Medicine and Science in Sports and Exercise 42 (3):413-421

Huxley HE (1969) Mechanism of muscular contraction. Science 164 (3886):1356-

Jensen RL, Ebben WP (2003) Kinetic analysis of complex training rest interval effect on vertical jump performance. J Strength Cond Res 17 (2):345-349

Jo E, Judelson DA, Brown LE, Coburn JW, Dabbs NC (2010) Influence of recovery duration after a potentiating stimulus on muscular power in recreationally trained individuals. Journal of Strength & Conditioning Research 24 (2):343-347

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

Judge LW, Bellar D, Judge M (2010) Efficacy of potentiation of performance through overweight implement throws on male and female high-school weight throwers. Journal of Strength & Conditioning Research 24 (7):1804-1809

Kilduff LP, Owen N, Bevan H, Bennett M, Kingsley MIC, Cunningham D (2008) Influence of recovery time on post-activation potentiation in professional rugby players. J Sport Sci 26 (8):795-802

Kitago T, Mazzocchio R, Liuzzi G, Cohen LG (2004) Modulation of H-reflex excitability by tetanic stimulation. Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology 115 (4):858-861

Klakowicz PM, Baldwin ERL, Collins DF (2006) Contribution of M-waves and H-reflexes to contractions evoked by tetanic nerve stimulation in humans. Journal of Neurophysiology 96 (3):1293-1302

Klug GA, Botterman BR, Stull JT (1982) The effect of low-frequency stimulation on myosin light chain phosphorylation in skeletal-muscle. J Biol Chem 257 (9):4688-4690

Knight CA, Kamen G (2001) Adaptations in muscular activation of the knee extensor muscles with strength training in young and older adults. J Electromyogr Kines 11 (6):405-412

Levine RJ, Kensler RW, Yang Z, Stull JT, Sweeney HL (1996) Myosin light chain phosphorylation affects the structure of rabbit skeletal muscle thick filaments. Biophysical Journal 71 (2):898-907

Macdonell RA, Talalla A, Swash M, Grundy D (1989) Intrathecal baclofen and the H-reflex. J Neurol Neurosurg Psychiatry 52 (9):1110-1112

MacIntosh BR (2003) Role of calcium sensitivity modulation in skeletal muscle performance. News Physiol Sci 18:222-225

MacIntosh BR, Grange RW, Cory CR, Houston ME (1993) Myosin light chain phosphorylation during staircase in fatigued skeletal muscle. Pflügers Archiv: European Journal Of Physiology 425 (1-2):9-15

MacIntosh BR, Taub EC, Dormer GN, Tomaras EK (2008) Potentiation of isometric and isotonic contractions during high-frequency stimulation. Pflugers Archiv European Journal of Physiology 456 (2):449-458

MacIntosh BR, Willis JC (2000) Force-frequency relationship and potentiation in mammalian skeletal muscle. Journal of Applied Physiology 88 (6):2088-2096

Magnus BC, Takahashi M, Mercer JA, Holcomb WR, McWhorter JW (2006) Investigation of vertical jump performance after completing heavy squat exercises. Journal of Strength & Conditioning Research 20 (3):597-600

Manning DR, Stull JT (1979) Myosin light chain phosphorylation and phosphorylase-a activity in rat extensor digitorum longus muscle. Biochem Bioph Res Co 90 (1):164-170

Manning DR, Stull JT (1982) Myosin light chain phosphorylation-dephosphorylation in mammalian skeletal muscle. Am J Physiol 242 (3):C234-C241

Matsunaga T, Shimada Y, Sato K (1999) Muscle fatigue from intermittent stimulation with low and high frequency electrical pulses. Arch Phys Med Rehab 80 (1):48-53

McBride JM, Nimphius S, Erickson TM (2005) The acute effects of heavy-load squats and loaded countermovement jumps on sprint performance. Journal of Strength & Conditioning Research 19 (4):893-897

Metzger JM, Moss RL (1992) Myosin light chain 2 modulates calcium-sensitive cross-bridge transitions in vertebrate skeletal muscle. Biophys J 63 (2):460-468

Mitchell CJ, Sale DG (2011) Enhancement of jump performance after a 5-RM squat is associated with postactivation potentiation. European Journal of Applied Physiology 111 (8):1957-1963

Miyamoto N, Kanehisa H, Fukunaga T, Kawakami Y (2011) Effect of postactivation potentiation on the maximal voluntary isokinetic concentric torque in humans. Journal of Strength & Conditioning Research 25 (1):186-192

Moore RL, Houston ME, Iwamoto GA, Stull JT (1985) Phosphorylation of Rabbit Skeletal-Muscle Myosin Insitu. J Cell Physiol 125 (2):301-305

Moore RL, Stull JT (1984) Myosin light chain phosphorylation in fast and slow skeletal muscles in situ. The American Journal of Physiology 247 (5 Pt 1):C462-C471

Morana C, Perrey S (2009) Time course of postactivation potentiation during intermittent submaximal fatiguing contractions in endurance- and power-trained athletes. Journal of Strength & Conditioning Research 23 (5):1456-1464

Moritani T, Oddsson L, Thorstensson A (1991) Phase-dependent preferential activation of the soleus and gastrocnemius muscles during hopping in humans. J Electromyogr Kinesiol 1 (1):34-40

Moritani T, Shibata M (1994) Premovement electromyographic silent period and alpha-motoneuron excitability. J Electromyogr Kinesiol 4 (1):27-33

O'Leary DD, Hope K, Sale DG (1997) Posttetanic potentiation of human dorsiflexors. Journal of Applied Physiology 83 (6):2131-2138

O'Leary DD, Hope K, Sale DG (1998) Influence of gender on post-tetanic potentiation in human dorsiflexors. Can J Physiol Pharm 76 (7-8):772-779

Paasuke M, Ereline J, Gapeyeva H (2000) Twitch contraction properties of plantar flexor muscles in pre- and post-pubertal boys and men. European Journal of Applied Physiology 82 (5-6):459-464

Paasuke M, Ereline J, Gapeyeva H, Maamagi H (2002a) Comparison of twitch-contractile properties of plantar-flexor muscles in young and 52-to 63-year-old men. J Aging Phys Activ 10 (2):160-168

Paasuke M, Ereline J, Gapeyeva H, Sander P, Sirkel S (2002b) Twitch potentiation capacity of plantarflexor muscles in women with increasing age. Biol Sport 19 (3):213-223

Paasuke M, Ereline J, Gapeyeva H, Torop T (2002c) Twitch contractile properties of plantarflexor muscles in female power-trained athletes. Med Sport 55 (4):279-286

Paasuke M, Saapar L, Ereline J, Gapeyeva H, Requena B, Oopik V (2007) Postactivation potentiation of knee extensor muscles in power- and endurance-trained, and untrained women. European Journal of Applied Physiology 101 (5):577-585

Palmieri RM, Ingersoll CD, Hoffman MA (2004) The Hoffmann reflex: Methodologic considerations and applications for use in sports medicine and athletic training research. Journal of Athletic Training 39 (3):268-277

Palmieri TM, Hoffman MA, Ingersoll CD (2002) Intersession reliability for H-reflex measurements arising from the soleus, peroneal, and tibialis anterior musculature. Int J Neurosci 112 (7):841-850

Papaiordanidou M, Guiraud D, Varray A (2010) Kinetics of neuromuscular changes during low-frequency electrical stimulation. Muscle & Nerve 41 (1):54-62

Persechini A, Stull JT, Cooke R (1985) The effect of myosin phosphorylation on the contractile properties of skinned rabbit skeletal muscle fibers. The Journal of Biological Chemistry 260 (13):7951-7954

Petrella RJ, Cunningham DA, Vandervoort AA, Paterson DH (1989) Comparison of twitch potentiation in the gastrocnemius of young and elderly men. European Journal of Applied Physiology and Occupational Physiology 58 (4):395-399

Pinniger GJ, Steele JR, Thorstensson A, Cresswell AG (2000) Tension regulation during lengthening and shortening actions of the human soleus muscle. Eur J Appl Physiol 81 (5):375-383

Place N, Maffiuletti NA, Ballay Y, Lepers R (2005) Twitch potentiation is greater after a fatiguing submaximal isometric contraction performed at short vs. long quadriceps muscle length. Journal of Applied Physiology 98 (2):429-436

Rassier DE (2000) The effects of length on fatigue and twitch potentiation in human skeletal muscle. Clin Physiol 20 (6):474-482

Rassier DE, Macintosh BR (2000) Coexistence of potentiation and fatigue in skeletal muscle. Brazilian Journal of Medical and Biological Research 33 (5):499-508

Rassier DE, MacIntosh BR (2002) Sarcomere length-dependence of activity-dependent twitch potentiation in mouse skeletal muscle. BMC Physiology 2:19-19

Rassier DE, Tubman LA (1997) Length-dependent potentiation and myosin light chain phosphorylation in rat gastricnemius muscle. American Journal of Physiology: Cell Physiology 42 (1):C198

Requena B, de Villarreal ESS, Gapeyeva H, Ereline J, Garcia I, Paasuke M (2011) Relationship between postactivation potentiation of knee extensor muscles, sprinting and vertical jumping performance in professional soccer players. J Strength Cond Res 25 (2):367-373

Requena B, Ereline J, Gapeyeva H, Paasuke M (2005a) Posttetanic potentiation in knee extensors after high-frequency submaximal percutaneous electrical stimulation. J Sport Rehabil 14 (3):248-257

Requena B, Gapeyeva H, Garcia I, Ereline J, Paasuke M (2008) Twitch potentiation after voluntary versus electrically induced isometric contractions in human knee extensor muscles. European Journal of Applied Physiology 104 (3):463-472

Requena B, Zabala M, Ribas J, Ereline J, Paasuke M, Gonzalez-Badillo JJ (2005b) Effect of post-tetanic potentiation of pectoralis and triceps brachii muscles on bench press performance. Journal of Strength & Conditioning Research 19 (3):622-627

Rixon KP, Lamont HS, Bemben MG (2007) Influence of type of muscle contraction, gender, and lifting experience on postactivation potentiation performance. Journal of Strength & Conditioning Research (Allen Press Publishing Services Inc) 21 (2):500-505

Robbins DW (2005) Postactivation potentiation and its practical applicability: A brief reivew. Journal of Strength & Conditioning Research 19 (2):453-458

Sale D (2004) Postactivation potentiation: role in performance. British Journal of Sports Medicine 38 (4):386-387

Sale DG (2002) Postactivation potentiation: role in human performance. Exercise & Sport Sciences Reviews 30 (3):138-143

Sale DG, Macdougall JD, Upton ARM, Mccomas AJ (1983) Effect of strength training upon motoneuron excitability in man. Medicine and Science in Sports and Exercise 15 (1):57-62

Shields RK, Chang YJ (1997) The effects of fatigue on the torque-frequency curve of the human paralysed soleus muscle. J Electromyogr Kines 7 (1):3-13

Shields RK, Dudley-Javoroski S, Littmann AE (2006) Postfatigue potentiation of the paralyzed soleus muscle: evidence for adaptation with long-term electrical stimulation training. J Appl Physiol 101 (2):556-565

Shields RK, Law LF, Reiling B, Sass K, Wilwert J (1997) Effects of electrically induced fatigue on the twitch and tetanus of paralyzed soleus muscle in humans. Journal of Applied Physiology 82 (5):1499-1507

Smith JC, Fry AC (2007) Effects of a ten-second maximum voluntary contraction on regulatory myosin light-chain phosphorylation and dynamic performance measures. Journal of Strength and Conditioning Research 21 (1):73-76

Staron RS, Karapondo DL, Kraemer WJ, Fry AC, Gordon SE, Falkel JE, Hagerman FC, Hikida RS (1994) Skeletal-muscle adaptations during early phase of heavy-resistance training in men and women. Journal of Applied Physiology 76 (3):1247-1255

Staron RS, Leonardi MJ, Karapondo DL, Malicky ES, Falkel JE, Hagerman FC, Hikida RS (1991) Strength and skeletal-muscle adaptations in heavy-resistance-trained women after detraining and retraining. Journal of Applied Physiology 70 (2):631-640

Stein RB, Thompson AK (2006) Muscle reflexes in motion: how, what, and why? Exercise and sport sciences reviews 34 (4):145-153

Stuart DS, Lingley MD, Grange RW, Houston ME (1988) Myosin light chain phosphorylation and contractile performance of human skeletal muscle. Can J Physiol Pharm 66 (1):49-54

Sweeney HL, Bowman BF, Stull JT (1993) Myosin light chain phosphorylation in vertebrate striated muscle: regulation and function. The American Journal Of Physiology 264 (5 Pt 1):C1085-C1095

Sweeney HL, Stull JT (1986) Phosphorylation of myosin in permeabilized mammalian cardiac and skeletal muscle cells. Am J Physiol 250 (4):C657-C660

Sweeney HL, Stull JT (1990) Alteration of cross-bridge kinetics by myosin light chain phosphorylation in rabbit skeletal muscle: Implications for regulation of actin

myosin interaction. Proceedings of the National Academy of Sciences of the United States of America 87 (1):414-418

Tidow G (1990) Aspects of strength training in athletics. New Studies in Athletics 1:93-110

Till KA, Cooke C (2009) The effects of postactivation potentiation on sprint and jump performance of male academy soccer players. Journal of Strength & Conditioning Research (Lippincott Williams & Wilkins) 23 (7):1960-1967

Tillin NA, Bishop D (2009) Factors modulating post-activation potentiation and its effect on performance of subsequent explosive activities. Sports Medicine 39 (2):147-166

Trimble MH, Harp SS (1998) Postexercise potentiation of the H-reflex in humans. Medicine and Science in Sports and Exercise 30 (6):933-941

Tsimahidis K, Galazoulas C, Skoufas D, Papaiakovou G, Bassa E, Patikas D, Kotzamanidis C (2010) The effect of sprinting after each set of heavy resistance training on the running speed and jumping performance of young basketball players. Journal of Strength & Conditioning Research 24 (8):2102-2108

Tubman LA, MacIntosh BR, Maki WA (1996) Myosin light chain phosphorylation and posttetanic potentiation in fatigued skeletal muscle. European Journal of Physiology 431 (6):882-887

Tubman LA, Rassier DE, MacIntosh BR (1997) Attenuation of myosin light chain phosphorylation and posttetanic potentiation in atrophied skeletal muscle. European Journal Of Physiology 434 (6):848-851

Vandenboom R, Grange RW, Houston ME (1993) Threshold for force potentiation associated with skeletal myosin phosphorylation. The American Journal of Physiology 265 (6 Pt 1):C1456-C1462

Vandenboom R, Grange RW, Houston ME (1995) Myosin phosphorylation enhances rate of force development in fast-twitch skeletal muscle. Am J Physiol-Cell Ph 268 (3):C596-C603

Vandenboom R, Houston ME (1996) Phosphorylation of myosin and twitch potentiation in fatigued skeletal muscle. Can J Physiol Pharmacol 74 (12):1315-1321

Vandervoort AA, Quinlan J, McComas AJ (1983) Twitch potentiation after voluntary contraction. Experimental Neurology 81 (1):141-152

Wallace B, Janz J (2009) Implications of Motor Unit Activity on Ballistic Movement. Int J Sports Sci Coa 4 (2):285-292

Wallace BJ, Kernozek TW, Mikat RP, Wright GA, Simons SZ, Wallace KL (2008) A comparison between back squat exercise and vertical jump kinematics: Implications for determining anterior cruciate ligament injury risk. J Strength Cond Res 22 (4):1249-1258

Weber KR, Brown LE, Coburn JW, Zinder SM (2008) Acute effects of heavy-load squats on consecutive squat jump performance. Journal of Strength & Conditioning Research (Lippincott Williams & Wilkins) 22 (3):726-730

Westerblad H, Duty S, Allen DG (1993) Intracellular calcium-concentration during low-frequency fatigue in isolated single fibers of mouse skeletal muscle. Journal of Applied Physiology 75 (1):382-388

Wilson GJ, Lyttle AD, Ostrowski KJ, Murphy AJ (1995) Assessing Dynamic Performance: A Comparison of Rate of Force Development Tests. The Journal of Strength & Conditioning Research 9 (3):176-181

Wilson JM, Duncan NM, Marin PJ, Brown LE, Loenneke JP, Wilson SMC, Jo E, Lowery RP, Ugrinowitsch C (2013) Meta-Analysis of Postactivation Potentiation and Power: Effects of Conditioning Activity, Volume, Gender, Rest Periods, and Training Status. J Strength Cond Res 27 (3):854-859

Winter D (1990) Biomechancis and Motor Control of Human Movement. 2 edn. Wiley-Interscience.

Young WB, Jenner A, Griffiths K (1998) Acute enhancement of power performance from heavy load squats. Journal of Strength & Conditioning Research 12 (2):82-84

Zehr EP (2002) Considerations for use of the Hoffmann reflex in exercise studies. European Journal of Applied Physiology 86 (6):455-468

Zhi G, Ryder JW, Huang J, Ding PG, Chen Y, Zhao YM, Kamm KE, Stull JT (2005) Myosin light chain kinase and myosin phosphorylation effect frequency-dependent potentiation of skeletal muscle contraction. Proceedings of the National Academy of Sciences of the United States of America 102 (48):17519-17524

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Educational Institutions

Master of Science	Human Performance, January 2007 University of Wisconsin–La Crosse, La Crosse, WI
Bachelor of Science	Exercise and Sport Science, December 2004 University of Wisconsin–La Crosse, La Crosse, WI

Professional Positions

Lecturer, Kinesiology and Health Promotion, University of Kentucky, Lexington, KY. 2013-present.

Biodynamics Lab Manager, Kinesiology and Health Promotion, University of Kentucky, Lexington, KY. 2012-2013.

Teaching Assistant, Kinesiology and Health Promotion, University of Kentucky, Lexington, KY. 2007-2013.

Kinesiologist, Shriners Hospitals for Children, Lexington, KY. 2009-2010.

Adjunct Faculty, Kinesiology and Health Studies, Georgetown College, Georgetown, KY. 2008.

Graduate Assistant, Department of Exercise and Sport Science, University of Wisconsin–La Crosse, La Crosse, WI. 2005-2006.

Strength Centers Staff, Department of Exercise and Sport Science, University of Wisconsin–La Crosse, La Crosse, WI. 2003-2005.

Intern, Community and Preventative Care Services, Gundersen Lutheran Hospital, La Crosse, WI. 2004.

Scholastic and Professional Honors

Arvle and Ellen Turner Thacker Research Fund. (2012). College of Education, University of Kentucky. Muscular and neural contributions to postactivation potentiation.

American College of Sports Medicine (ACSM) Biomechanics Interest Group (BIG) Student Travel Award. (2008). Quantification of kinetic parameters of various bilateral plyometric exercises.

Conference Student Support Funding. (2008). University of Kentucky. Quantification of kinetic parameters of various bilateral plyometric exercises.

University of Wisconsin - La Crosse Graduate Student Research, Service, and Educational Leadership Grant. (2005). University of Wisconsin-La Crosse. A Comparison between back squat exercise and vertical jump kinematics: Implications for diagnosing ACL injury risk.

Professional Publications - Manuscripts

Wallace, B., Kernozek, T., White, J., Kline, D, Wright, G., Peng, H., & Huang, C. (2010). Quantification of vertical ground reaction forces of popular bilateral plyometric exercises. *Journal of Strength and Conditioning Research.* 24(1). 207-212.

Wallace, B.J. & Abel, M.G. (2010). Effects of chocolate milk on perceived exertion and muscular strength following resistance training: A Pilot study. *International Journal of Fitness*. 6(1). 25-32.

Wallace, B. & Janz, J. (2009). Implications of motor unit activity on ballistic movement. *International Journal of Sports Science & Coaching*. 4(2). 285-292.

Wallace, B., Kernozek, T., Mikat, R., Wright, G., Simons, S., & Wallace, K. (2008). A comparison between back squat exercise and vertical jump kinematics: Implications for determining ACL injury risk. *Journal of Strength and Conditioning Research.* 22(4). 1249-1258.

Wallace, K.L. & Wallace, B.J. (2008). Raising the bar: Pushing away the misconceptions and pulling out new ideas regarding youth strength training. *Physical and Health Education Journal.* 75(1). 16-22.

Wallace, B. & Metcalfe, E. (2008). Point/Counterpoint: Dual role athletic trainer/strength coach. *Strength and Conditioning Journal*. 30(1). 26-28.

Wallace, B., Kernozek, T., & Bothwell, E. (2007). Lower extremity kinematics and kinetics of division three collegiate baseball and softball players while performing a modified pro-agility task. *Journal of Sports Medicine and Physical Fitness*. 47(4). 377-384.

Wallace, B.J., Winchester, J.B., & McGuigan, M.R. (2006). Effects of elastic bands on force and power characteristics during the back squat exercise. *Journal of Strength and Conditioning Research*. 20(2). 268-272.

Wallace, B.J. & Kline, D.E. (2005). Prevent defense. *Athletic Business*. 29(4). 77-78;82.

Professional Publications – Abstracts

Shapiro, R., Cunningham, T., Wallace, B., Norberg, J., Phillips, M., & Miller, M. (2012). Differences in running and walking gait kinematics during Earth and simulated Mars and Lunar gravitational environments: Preliminary investigation. Presented at the 2012 American Society of Biomechanics Annual Meeting, Gainesville, FL.

Wallace, B., Viele, K., & Sugimoto, D. (2011). Analyzing band assisted squat exercise ground reaction forces using landmark registration: a pilot study. Presented at the 2011 NSCA KY State Conference, Louisville, KY.

Wallace, B., Kernozek, T., White, J., Kline, D., Wright, G., Peng, B, & Huang, P. (2008). Quantification of kinetic parameters of various bilateral plyometric exercises. Presented at the 2008 ACSM Annual Meeting, Indianapolis, IN. Reprinted in *Medicine and Science in Sports and Exercise*. 37(5).

White, J., Kline, D., Kernozek, T., Soderstrom, A., Wallace, B., & Wright, G. (2007). Quantification of vertical ground reaction forces of popular bilateral plyometric movements. Presented at the 2007 NSCA National Conference and Exhibition, Atlanta, GA. Reprinted in *Journal of Strength and Conditioning Research*. 21(4). e15.

Wallace, B., Kernozek, T., & Bothwell, E. (2006). Gender differences in the lower extremity kinematics and kinetics of collegiate division three baseball and softball players while performing a modified pro-agility test. Presented at the 2006 NSCA National Conference and Exhibition, Washington, D.C. Reprinted in *Journal of Strength and Conditioning Research*. 20(4). e5. (Podium)

Kernozek, T.W., Torry M.R., Wallace, B.J., & Miller, E.J. (2006). Biomechanics of a failed single legged landing due to fatigue. Presented at 2006 ACSM Annual Meeting, Denver, CO. Reprinted in *Medicine and Science in Sports and Exercise*. 35(5). 677.

Wallace, B., Kernozek, T., & Bothwell, E. (2006). Gender differences in the lower extremity kinematics and kinetics of collegiate division three baseball and softball players while performing a modified pro-agility test. Presented at the 2006 Midwest Graduate Student Biomechanics Symposium, Milwaukee, WI. (Podium)

Wallace, B., Winchester, J., & McGuigan, M. (2004). Effects of elastic bands on force and power characteristics during the back squat exercise. Presented at 2004 NSCA National Conference and Exhibition. Reprinted in *Journal of Strength and Conditioning Research*. 18(4). e370.