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DO CHANGES IN MUSCLE ARCITECTURE EFFECT POST-ACTIVATION POTENTIATION?

by

DANIELLE REARDON A.A. Associates of Arts, Valencia College 2010 B.S. Exercise Science, University of Central Florida 2012

A thesis submitted in partial fulfillment of the requirements for the degree of Masters of Science in Applied Exercise Physiology in the Institute of Exercise Physiology and Wellness in the College of Education and Human Performance at the University of Central Florida Orlando, Florida

Fall Term 2013

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ABSTRACT

Purpose: To examine the effect of three muscle potentiation protocols on changes in muscle architecture and the subsequent effect on jump power performance.

Methods: Maximal (1RM) squat strength (Mean SD=178.3 \pm 36.6kg), vertical jump power, and muscle architecture were obtained in 12 resistance trained men (25.2 \pm 3.6y; 90.67 \pm 12.7kg). Participants randomly completed three squatting protocols at 75% (3 x 10 reps), 90% (3 x 3 reps) or 100% (1 x 1) of their 1RM, or no workout (CON), with each protocol being separated by one week. During each testing session ultrasound and vertical jump testing were assessed at baseline (BL), 8min post (8P) and 20min post (20P) workout. Ultrasound measures of the rectus femoris (RF) and vastus lateralis (VL) muscles included; cross sectional area (CSA) and pennation angle (PNG). Following each ultrasound, peak (PVJP) and mean (MVJP) vertical jump power (using hands for maximum jump height) were measured using an accelerometer.

Results: Magnitude based inferences analysis indicated that in comparison to CON, 75% resulted in a likely greater change in RF-CSA and VL-CSA (BL-8P and BL–20P), 90% resulted in a likely greater RF-CSA and VL-CSA (BL–20P), and 100% resulted in a very likely or likely decrease in VL-PNG at BL-8P and BL–20P, respectively). Meanwhile, changes in PVJP and MVJP for the 75% trial was likely decreased at BL-8P and BL–20P; and for the 90% trial MVJP was likely decreased at BL-8P and BL–20P. Analysis of the magnitude of the relationships indicated a likely negative relationship between VL-PNG and MVJP (r = -0.35; p < 0.018) at BL-8P, while at BL–20P, a negative relationship was observed between PVJP and RF-CSA (r = -0.37; p < 0.014).

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Conclusion: Acute increases in muscle size and acute decreases in pennation angle did not result in any potentiation in vertical jump power measures. Although the inverse relationships observed between muscle architecture variables and power suggests a potential effect, the change in position (i.e. movement from standing to supine for ultrasound measures) may negate, as a result of potential fluid shifts or muscle relaxation, the potentiating effects of the exercise. It is also possible that the fatiguing nature of the squat protocols in trained but not competitive participants may have also contributed to the results. I lovingly dedicate my thesis work to my family. Mom, Dad, Judy, and Sarah you have supported me through this whole process not letting distance hinder your support in anyway, giving me pep talks, and words of wisdom through not only this project but my whole academic career. Included in that bunch is my boyfriend, Ian. You have witnessed, helped, and supported, me through some of the most challenging times of my life. Thank you for everything, especially being so understanding and my rock when I needed it. I love you all very much, and couldn't ask for a better family.

ACKNOWLEDGMENTS

This research project would not have been possible without the support of many people. I want to sincerely express my gratitude to my faculty advisor, Dr. Jay Hoffman who has been abundantly helpful and offered invaluable assistance, support and guidance through the whole project. Deepest gratitude is also due to the remaining members of my thesis committee, Assoc. Prof. Dr. Jeff Stout, Dr. Maren Fragala, and Dr. David Fukuda without whose knowledge, assistance and words of advice this study would not have been successful. Exceptional thanks also to the graduate and undergraduate students in the human performance lab; for sharing literature, vital assistance, experience and friendship. I would also like to convey thanks to the University of Central Florida and Faculty for providing the financial means and outstanding laboratory facilities to complete this study. Last and far from least, I want to express love and gratitude to my family; for their support, understanding & endless love, through the duration of my study and graduate career.

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LIST OF ACRONYMS/ ABBREVIATIONS

1RM	One Repetition Maximum
ANOVA	Analysis of Variance
BL	Baseline
СМЈ	Counter Movement Jump
CSA	Cross Sectional Area
CTL	Control
HI	High Intensity (90% 1RM)
HPL	Human Performance Laboratory
ICC	Intraclass Correlation Coefficient
MI	Moderate Intensity (75% 1RM)
MRLC	Myosin Regulatory Light Chain
MVJP	Mean Vertical Jump Power
MVC	Maximal Voluntary Contraction
PANG	Pennation Angle
PAP	Post Activation Potentiation

PVJP	Peak Vertical Jump Power
RF	Rectus Femoris
SEM	Standard error of Measurement
VAS	Visual Analog Scale
VJ20P	Vertical Jump 20 min Post Squat
VJ8P	Vertical Jump 8 min Post Squat
VJPRE	Vertical Jump Pre Squat
VL	Vastus Lateralis

CHAPTER ONE: INTRODUCTION

Post-activation potentiation (PAP) is a phenomenon by which the force exerted by a muscle is increased due to its previous contractions (Robbins, 2005). Potentiation appears to be dependent on an appropriate training stimulus and a proper rest interval to ensure fatigue is not instigating performance impairments (Goosen and Sale, 2000). Many studies show conflicting results regarding rest intervals and training intensity (Wilson, 2013). The conflicting results reported amongst studies are highly dependent upon the individual performing the task. There are many differences between untrained, trained and competitive athletes residing within their muscle physiology and architecture that can and has influenced the PAP response. Consequently, the variability between subjects makes administering an optimal PAP protocol difficult, as potentiation is not a "one size fits all" phenomena. It has even been argued that PAP is more of a muscle phenomenon or an observation rather than something that can be trained or altered (Sale, 2002). Potentiation has been induced in many different ways. The most common method is from the use of maximum voluntary contractions (Mitchell and Sale, 2011), but PAP has also been prompted by submaximal efforts. Previous studies examining PAP determined that loads of 80% or more of 1RM are needed to elicit a potentiation effect to facilitate short term power increases (Gouvea et al., 2012; Matthews, O'Conchuir and Comfort, 2009; Weber, Brown, Coburn & Zinder, 2008). However, a recent meta-analysis by Wilson et.al (2013) has suggested that intensities of 60-84% 1RM are optimal for inducing PAP.

Rest period length is also considered very important in stimulating muscle potentiation. Fatigue seems to be dominant in the early stages of recovery, potentially diminishing subsequent power performance (Tillin and Bishop, 2009). Studies have examined rest intervals from immediately post stimulus to 20 min. post stimulus. Gullich and Schmistbleicher (1996) reported no change, or a decrease in the rate of force development, when power was assessed immediately following the potentiation stimulus. However, as the rest interval increases from 4to 18 min. post-exercise, improvements in power or jump height are seen (Mcann, Flanagan, 2010). Based on previous study outcomes, recommendations for a 7-10 or 8-12 min. recovery interval is recommended to be used to enhance the potentiation response to exercise (Gouve et al., 2012; Wilson et al. 2013).

The mechanism responsible for muscle potentiation has not been fully elucidated. It has been suggested that priming the neurological system by enhancing motor unit activation is one possible mechanism (Tillin and Bishop, 2009). However, acute changes in muscle architecture may also contribute to the potentiation response (Tillin and Bishop, 2009). Changes in muscle pennation angle appear to have a significant role on muscle power performance (Earp, et al., 2010). Larger pennation angles are reported to be associated with a greater potential for generating power (Earp, et al., 2010), yet the force per cross section has been reported to decrease (Ikegawa et. al, 2008). In contrast, a smaller pennation angle has been associated with faster sprinting ability (Kumangi et. al, 2000). Mahlfeld and colleagues (2004) reported that a decrease in pennation angle occurs for 3-6 minutes following maximal voluntary contractions. Furthermore, increased muscle thickness measures have also been correlated highly with the ability to produce force (Seyennes, Boer and Narici, 2007). A greater fascicle length represents longer sarcomeres or more sarcomeres in line (Earp, et al., 2010). Thus, as fascicle length increases, so does the velocity of the movement and the force that can be applied at higher

velocities. However, acute changes in muscle architecture and its role in muscle potentiation are not fully understood. Thus, the purpose of this study was to examine the effect of three muscle potentiation protocols on changes in muscle architecture and the subsequent effect on jump power performance.

CHAPTER TWO: LITERATURE REVIEW

Post-Activation Potentiation

Post- activation potentiation (PAP) is a phenomenon by which, the contractile history of skeletal muscle influences subsequent contractions of the same muscle group, typically increasing peak force and rate of force development (Tillin and Bishop, 2009). The mechanisms responsible for eliciting the PAP response reside either within the muscle; increasing Ca²⁺ sensitivity and rate of cross bridge attachment, or an acute change in muscle architecture, or at the spinal cord by enhancing neural drive to increase higher end motor unit recruitment. An optimal protocol for inducing this response has yet to be determined. This is likely related to the high degree of inter-individual variability attributed to differences in training status and the potentiation stimulus (i.e., exercise type, intensity, volume, rest interval). The purpose of this review is to focus on the mechanisms of the PAP, as well as what are the most optimal variables for maximizing its response.

Mechanisms Attributed to Post-Activation Potentiation

Muscle Phosphorylation

As discussed, there are several mechanisms that have been attributed to PAP. Acute changes within the muscle resulting from previous exercise have been suggested to have a potentiating effect. One of these mechanisms enhancing PAP is related to muscle contraction induced myosin regulatory light chain phosphorylation (Cabrera, Morales, Greer, & Pettitt, 2009). It is suggested that previous muscle activity can enhance the sensitivity of the actin and myosin

myofilaments to Ca^{2+} released from the sarcoplasmic reticulum, resulting in greater force output for each successive twitch contraction (Lorenz, 2011). A myosin molecule is composed of two heavy chains, the amino termini of each chain, classified as the myosin head, contains two regulatory light chains (MRLC) (Vandenboom, Grange, Houston, 1993; Szczesna, 2003). Each MRLC contains a specific site for phosphate to bind to. The enzyme, myosin light chain kinase (MLCK) is activated by increases in Ca^{2+} concentration, and is responsible for phosphorylation of the MRLC. Upon muscle activation, Ca²⁺ concentrations elevate, activate MLCK and increase MRLC phosphorylation. The phosphorylation of MLCK increases the rate at which myosin cross bridges move from a non- force producing state to a force producing state (Grange, Vandenboom, Houston, 1993 and Sweeney Bowman, and Stull, 1993). Enhanced force production as a result of MRLC phosphorylation is due to the increased Ca²⁺ sensitivity, and the increased rate of attachment of cross bridges (Rassier and MacIntosh, 2000), thereby improving the ability of the sarcomere to produce more force within a smaller window of time (Sweeney and Stull, 1990). Several studies have demonstrated a positive relationship between the magnitude of potentiation and the magnitude of MRLC phosphorylation (Klug et al., 1982; Manning & Stull, 1989; 1982; Moore & Stull, 1982). Still, others have been unable to support the effect of MRLC phosphorylation and muscle potentiation. Smith and Frye (2007) reported no significant difference in leg extension performance or change in muscle phosphorylation 7min following a 10-sec isometric maximal voluntary contraction. Whether this was a sufficient PAP stimulus is debatable, yet it does suggest that other mechanisms may contribute to PAP.

Muscle Architecture

The ability of a muscle to generate maximal power appears to be influenced by a series of morphological factors, one of the most important being the architecture of the muscle fibers (Cormie, McGuigan and Newton, 2011). These architectural variables include pennation angle (PANG) and cross sectional area (CSA). Irrespective of fiber type, the maximal force generated by a single muscle fiber is directionally proportional to its CSA (Gollnick & Bayley, 1986; Edgerton, Roy, Gregor, et al, 1986; McComas, 1996; Bodine, Roy, Meadows, et al. 1982; Partridge & Benton, 1981). An increase in muscle fiber CSA is generally attributed to an increase in the size and number of myofibrils within each muscle fiber (Komi, 1973; MacDougall, 1986; MacDougall, 1992). Muscle fibers with greater CSA can generate a higher maximal power output than muscle fibers with smaller CSA (Jones, Rutherford, Parker, 1989; MacIntosh & Holash, 2000; Malisoux, Francaux, Nielens, et al.2006; Rutherford & Jones, 1986; Widrick, Stelzer, Shoepe et al., 2002, Herbert & Gandevia , 1995; Shoepe, Stelzer, Garner, et al. 2003).

In addition to a training response, which would have little effect on PAP, acute changes in muscle CSA have also been reported (Jajtner, Hoffman, Gonzalez, et al., unpublished data; Storey, Wong, Smith, and Marshall, 2012). The mechanism responsible for these acute changes are likely related to the post-exercise hyperemic response following the acute training stimulus (Collier et al., 2010; Fahs et al., 2011). Whether this mechanism is contributing to an enhanced PAP is not well-understood. To date, there are no studies found that have examined this specific question. Interestingly, if muscle CSA is enhanced by an acute exercise response, it would likely alter muscle pennation angle as well.

The pennation angle (PANG) of a muscle is formed by the fascicles and the inner aponeurosis (Folland and Williams, 2007), and is defined as the angle between the muscles' fascicles and the line of action (Spector, Gardiner, Zernicke, et al. 1980; Huijing, 1985; Powell, Roy, Kanim, et al. 1984). PANG will physiologically affect the force- velocity relationship and force transmission from the muscular contraction to the tendons and bones (Folland and Williams, 2007; Fukunanga & Ito et al., 1997). PANG has been shown to be related to force and power ability within a muscle. A larger PANG is associated with greater force capability, while decreases in PANG are associated with greater improvements in speed or power performance (Abe et al., 1998; Earp, Kraemer, Newton, Comstock, Fragala, Lewis, Hill & Penwell, 2010). Increases in force capability with a larger PANG is related to a decreased length the muscle has to shorten to produce the same amount of force (Muhl, 1982), but a slowed contraction velocity from an increase in PANG may imply a negative impact on power production (Spector, Gardiner, Zernicke, et al. 1980). On the other hand, a small PANG operates at a biomechanical advantage, allowing more sarcomeres to be aligned in series, resulting in a rapid transmission of force from the muscle to the tendon, thus increasing contractile RFD and contractile impulse (Storey, Wong, Smith, & Marshall, 2012).

Acute change in PANG resulting from an acute bout of exercise is not well understood. Considering the potential hyperemic response associated with resistance exercise, it would stand to reason that changes in muscle CSA would also alter muscle PANG as well. Mahlfield, Franke, and Awiszus (2004) reported a significant decrease (~11%) in PANG 3-6 minutes after a 3-sec isometric maximal voluntary contraction. However, the change in PANG was associated with only a 0.9% increase in force transmission to the tendons, but no effects on power were

reported. Nevertheless a decrease in PANG was related in an increase in force transmission, and these types of changes to muscle architecture are postulated to contribute to the PAP response (Tillin and Bishop, 2009). However, to date there does not appear to have been any studies examining acute changes in PANG and its potential role in PAP.

Neurological Adaptation

Although much of the existing literature suggests that the mechanism responsible for PAP is localized within the muscle, others have indicated that PAP may also be a function of acute neural adaptation (Gullich & Schmidtbleicher, 1996; Kitago, Mazzocchio, Luizzi et al., 2004; Trimble & Harp, 1998;; Van Boxtel, 1986; and Zucker & Regehr; 2002). By performing heavy loaded exercises right before a light power exercise, it is thought that there would be greater activation and preparation for maximal effort with the lighter load (Verkhoshansky, 1983). A mechanism termed the Hoffman reflex (H-reflex) is considered to be the electrical analogue of the stretch reflex (Brooke et al., 1997; and Scheppati, 1987). The amplitude of the H- reflex is a function of the number and size of recruited motor neurons (Hugon, 1973). Variation in the H- reflex amplitude is relative to the constant intensity of stimulation and constant efferent motor response (M-wave), indicating synaptic modification occurring at the spinal cord (Misiaszek, 2003). The H-reflex is thought to occur by altering the excitability of the motor neurons, varying the amount of neurotransmitter released by afferent terminals, or varying the intrinsic properties of the motor neurons (Misiaszek, 2003). It is thought that the H-reflex works in a similar manner to the size principle and would be most beneficial to the type II or fast twitch fibers (Zehr, 2002; Henneman, Somjen & Carpenter, 1965).

In the presence of a potentiated reflex response an individual can optimize the reflex contribution to neural drive, thus increasing the efficacy of subsequent voluntary contractions (Hodgson, Docherty and Robbins, 2005). Heavy resistance training prior to a plyometric exercise may increase synaptic excitability within the spinal cord, which in turn results in increased post-synaptic potentials and subsequent increased force generating capacity of the involved muscle groups (Rassier, and Herzog, 2002). Gullich and Schmidtbleicher (1996) demonstrated a significant potentiation response, as a result of the H-reflex in elite speed- strength trained athletes, but not in physical education students (untrained) following isometric maximal voluntary plantarflexions.

Variability in Post-Activation Potentiation

In attempt to maximize PAP, many studies have been conducted in order to define an optimal stimulus for a given individual. Collectively, the studies conducted report inconsistencies in their findings, which can be attributed to the high degree of inter-individual variability. The number of combinations that can be made between subject characteristics and the potentiation stimulus (i.e., exercise type, intensity, volume, rest interval) complicates this matter even further.

Muscle Fiber Type Distribution:

There are many variables suggested to affect PAP. The most important variable may be the distribution of muscle fiber type within a given muscle (Lorenz, 2011). Muscle fiber type can affect the magnitude of post activation potentiation. The highest magnitudes of PAP have been recognized in muscles that are predominantly type II muscle fibers (Hamada, Sale, and MacDougall, 2000; Miyamoto, Fukunaga , and Kawakami, 2009). Hamada and colleagues (2000) reported significant differences in PAP in participants with differing percentages of fast twitch fibers. Those participants with a higher percentage of type II muscle fibers experienced a greater PAP compared to participants with a lower percentage of type II muscle fibers. Type II muscle fibers display the greatest increase in MRLC phosphorylation following a contraction (Moore and Stull, 1984). MRLC is a mechanism in which potentiation can be achieved through increased Ca2+ sensitivity and increased rate of cross bridge attachment (Hodgson, Docherty and Robbins, 2005). Thus, it would appear that athletes with a higher percentage of type II fibers would be more sensitive to the potential ergogenic effects of PAP. In an examination of NCAA Division I track and field, strength/power athletes, Weber and colleagues (2008) reported on significant improvements in jump performance following a 5-RM squat protocol. Considering that these were likely athletes with a high percentage of type II fibers, these athletes may have been optimal to experience a PAP response.

Training Status

Studies have shown athletes who participate in sports that demand maximal intensity, strength/powerful movements (sprinting, jumping, and throwing) would elicit the greatest magnitude of PAP as well as reap the greatest benefits (Lorenz, 2011). The potential for PAP appears to differ between trained and untrained individuals. However, the optimal PAP response may be seen in trained, competitive athletes (Wilson, Duncan, Marin, Brown, Loenneke, Wilson, S., Jo, and Lowrey, 2013). A trained athlete is one that can be defined as having years of experience, but not necessarily be of a high level of performance, whereas a trained, competitive athlete is one that is experienced but is also competing. In a review, Wilson and colleagues

(2013) compared several studies examining individuals of varying training experiences; untrained (sedentary), recreationally trained (active, but not resistance training), resistance trained (at least 1 year training experience) and competitive athletes (>3 years resistance training experience) and suggested PAP is most effective in resistance trained and competitive strength/power athletes with at least one year of resistance training experience. Others have suggested that competitive strength/power athletes with at least 3 years of resistance training experience would be most sensitive to the effects of PAP (Chiu, Fry, Weiss, Schilling, Brown, and Smith, SL 2003).

One study compared the effects of a heavy load back squat training program (5x1 with 90% 1RM, 5-7 min rest between sets) on subsequent jump power performance on competitive and recreationally trained athletes (Chiu et al., 2003). They found significantly greater power potentiation (1-3% increase in countermovement jump (CMJ) performance and depth drop height) in the competitive athletes, but a 1-4% decline in performance was noted in the recreationally trained athletes.

Muscular Strength

There appears to be a relationship between strength level and potential for CMJ potentiation. Kilduff, Bevan, and Kingsley, (2007) reported a moderate correlation (r=0.63) between 1 RM strength and CMJ potentiation after a high intensity activity (1x3RM back squat). The potential potentiating effect related to strength levels was also supported by Gourgoloulis Aggeloussis, and Kasimatis (2003), who compared individuals who could squat >160kg to those who squatted <160kg. A 4% increase (p< 0.05) was reported in CMJ height following 5 sets of

back squats in the stronger group, while the group who squatted < 160kg only increased 0.4% in CMJ height (p>0.05).

There is evidence that suggests that the power to strength ratio may also play a role in inducing PAP (Schneiker et al., 2006). In a comparison of two groups, Schneiker and colleagues compared one group with a power/strength ratio <19 W/kg to a second group with a power/strength ratio >19W/kg. Each group performed one set of a 6RM back squat and then performed a loaded CMJ 2-4 minutes following the squat exercise. The group with the lower power/strength ratio was shown to have a negative significant correlation ($r^2 = -0.91$), while the group with the higher power/strength ratio showed no relationship (Schneiker, 2006). These results suggest that athletes who are powerful and strong may be experience greater potentiating effects from previous exercise than athletes who were powerful and less strong.

Methods for Potentiation

PAP has typically been induced by using a near maximal, or maximal, voluntary dynamic or isometric contractions, and has been shown to increase peak force and rate of force development during subsequent contractions (Tillin and Bishop, 2009). Sale (2002) suggests the degree in which the PAP response is realized is likely related to its prior contractions. However, inconsistent findings have been reported with the administration of different contraction types. Research on isometric MVC'S have shown significant increases in subsequent explosive activity (French DN, Kraemer WJ, Cooke CB, 2003; Gullich, Schmidtbleicher. 1996) while others report no change in performance (Behm, Button, Barbour et al. 2004; Gossen, Sale, 2000; Robbins, Docherty, 2005). Similar to isometric contractions, dynamic contraction protocols also show conflicting results, some report significance in subsequent power performance (Gourgoulis, Aggeloussis, Kasimatis, et al., 2003; Rahimi, 2005; Saez Saez de Villarreal, Gonzalez-Badillo, Izquierdo, 2007; Batista, 2007; Chatzopoulos, Michailidis, Giannakos, et al., 2007; Kilduff, Bevan, Kingsley, et al., 2007; Young, Jenner, Griffiths, 1998) while others do not (Chiu, Fry, Weiss, et al., 2003; Ebben, Jenson RL, Blackard, 2000; Hanson, Leigh, Mynark, 2007; Jensen, Ebben, 2003; Mangus, Takahashi, Mercer, et al., 2003).

An isometric contraction is one in which muscle remains contracted for a period of time but there is no movement. In contrast, during a dynamic contraction, there is rhythmical contraction and relaxation of a muscle which does result in movement. Rixion, Lamont and Bemben (2007) were one of the few studies to compare isometric (maximal voluntary contraction back squat) to dynamic movements (1 x 3RM of the back squat) on subsequent CMJ performance. They reported that isometric contractions elicited a greater PAP response in CMJ height than the dynamic contraction (2.9% versus no change, respectively), while no differences were seen between contraction types in peak CMJ power (8.7% and 8.0% improvements, respectively).

Differences in contraction type on PAP may also be related to muscle proprioception activation. For instance, dynamic exercise includes an eccentric phase, while isometric exercises do not. The eccentric phase increases muscle spindle firing, which activates group 1a neural fibers (Taylor Butler, and Gandevia, 2000). This could lead to enhanced neural volley at the spinal cord and consequently a decrease in transmission failure from 1a neural fibers to adjacent α - motor units, and thus activation of higher end motor units for subsequent activity (Tillin and Bishop, 2009). Isometric contractions activate a greater number of motor units than dynamic

contractions (Duchateau and Hainaut, 1984), consequently, activating a greater number of motor units, resulting in a greater percentage of MRLC phosphorylation, as well as potentially greater changes to muscle architecture (Tillin and Bishop, 2009). Unfortunately the interactions of these mechanisms with PAP have not been clearly defined, and further research is necessary.

Exercise Type

Acute increases in explosive force in the upper and lower body have been observed with the use of maximal voluntary contractions. Dynamic, multi-joint, strength exercises (i.e., back squat) are used in the majority of potentiation studies. However, the use of exercises that elicit a greater power output (cleans versus squats) may produce differing results. McCann and Flanagan (2010) compared the back squat to a power exercise (hang clean) to determine if a greater velocity movement would induce a better PAP response. Their results indicated that the hang clean was not as effective in eliciting PAP as the back squat. This supports the work of McBride and colleagues (2005) who reported that a loaded CMJ was unable to potentiate 40m sprint performance. In contrast, others have suggested that integration of power exercises into a warm-up (4 sets of 4 power snatches with 75% -85% of the subjects 1RM) can increase the standing long jump by 3.9 cm (Radcliff and Radcliff, 1996), while Gilbert and Lees (2005) reported improvements in vertical jump height following a heavy back squat of 1RM. Weber Brown, Coburn, and Zinder, (2008) reported that performing a heavy back squat (85% 1RM) before a set of squat jumps significantly enhanced acute jump performance and ground reaction forces compared to a five-repetition squat jump. Similarly, Young, Jenner, and Griffiths, (1998) observed significant acute enhancement of power performance (2.8%) in 10 resistance trained men during the loaded CMJ, following a 5RM half squat protocol. Considering the contrasting

results from these studies, it appears that both traditional strength and power exercises are effective in eliciting a PAP response. It may be dependent upon specific athletes or athlete preference.

Volume/ Intensity:

The greatest augmentation of power performance in trained individuals appears to occur when multiple sets are used compared to a single set for PAP (Wilson et al., 2013). However, this may be mediated by training experience. In untrained individuals, the use of multiple sets may decrease subsequent power output, while in the experienced trained individuals multiple sets appears to augment power output (Wilson et al., 2013). This is likely related to the lack of conditioning in individuals with limited training resulting in greater fatigue and likely negates the potentiating effect. Gouvea, Fernandes, Cesar, Silva, and Gomes, (2013) examined 14 studies including 165 participants who were training for at least 6 months, and suggested that single sets (lower volume) with higher intensities (80-100% 1RM) produced greater potentiation effects than the multiple set moderate volume protocols, especially in the well trainedcompetitive athlete population. These contrasting conclusions from recent reviews are difficult to explain, but may be related to differences in study selection between the two meta-analyses.

In studies examining trained athletes, Weber et al. (2008) reported an acute potentiating effect in lower body muscular power following a squat protocol of 1x5RM (85% 1RM) in twelve NCAA D1 track and field athletes. In addition, Hoffman, Ratamess, Faigenbaum, Mangine, and Kang (2007) observed a 3% increase (p < 0.05) in vertical jump height and significant improvements in peak power 5 minutes following a 1RM (maximal) squat exercise in American college football players. Others have shown significant improvements in jump performance 8-min following a squat protocol (3x3 at 87% 1RM) in professional rugby players (Kilduff, 2008). Multiple set protocols using exercise intensities exceeding 80% 1RM have leading to PAP have also been shown in volleyball, and other non-described athletes (Chiu, 2003; Crewther, Kilduff, Cook, Middleton, Bunce, and Yang, 2011; Saez Saez de Villarreal, Gonzalez-Badillo, and Izquierdo, 2007). The efficacy of multiple versus single set potentiation protocols may be similar to the differences observed and discussed between trained and untrained individuals. It is likely that the trained athlete can perform more efficiently following a multi-set protocol due to physiological adaptations associated with training such as: increased buffering capacity and resistance to muscle damage (Kendrick, 2008; Skulachev, 2000; McHugh, 1999).

Exercise intensity is an important variable that also contributes to the potentiating effect. A recent meta-analysis concluded that individuals with less than 1 year training experience should only perform a single set (vs. multiple) with moderate intensities (60-84% 1RM). While, individuals with more than 1 year of training experience, will benefit from the same intensity (60-84% 1RM), but with the use of multiple (vs. single) sets prior to performing a criterion power task (Wilson et al, 2013). In contrast, a meta-analysis by Gouvea and collegues (2012), suggested that intensities ranging from 80- 100% 1RM elicits the greatest performance enhancements. Although these studies have reported increases in jump performance, following interventions with loads ranging from 80- 100% 1RM, there were also several studies, using similar loads that produce no significant difference in jump performance (Kilduff et al., 2007; Jones and Lees, 2003; Esforme., Cameron, and Bampouras, 2010; Khamoui, Brown, Coburn, Judelson, Uribe, Nguyen, Tran, and Eurich, 2009; Deutsch and Lloyd, 2008). The

inconsistencies in the literature regarding training intensity are likely attributed to the differences in training status among the studies. Similar to what was discussed before, differences in training status likely contributes to a greater fatigue in multi-set, high intensity training in less experienced or non-competitive or non-conditioned individuals (regardless of training experience) than athletes that are highly conditioned and competitive.

Rest Intervals

Investigations examining PAP have been conducted using rest intervals between the exercise stimulus and performance measure as soon as immediately post (Jensen and Ebben, 2003) up to six hours post-stimulus with equivocal results. Gourgoulis et al. (2003) reported a significant increase in CMJ height immediately following two back squats performed with 90% 1RM (Gourgoulis et al., 2003). These results suggest that PAP can be achieved immediately after the stimulus, however Tillin and Bishop (2009) suggest that potentiation declines within the first 60 seconds following the stimulus followed by a slow, more gradual decline in potentiation. In contrast, others have suggested a decrease in performance when the actual performance measures occur between 10 - 15 seconds following the potentiating stimulus (Crewther et al., 2011; Gilbert et al. 2001; Gullich and Schmidtbliecher, 1996; Jensen & Ebben, 2003; Kilduff et al., 2008). Even recovery periods lasting between 3 – 7 minutes may not be sufficient to see a potentiating effect in competitive athletes (Comyns, Harrison, Hennessy, Jensen, 2006; Jensen et al., 2003; Weber et al., 2008). However, when recovery time is extended the potentiating response is observed with more consistency.

It appears that a rest intervals of 8 - 12 minutes elicits the greatest potentiating responses (Gouvea et al., 2012), while others recommend 7 – 10 minutes (Wilson et al., 2013). One study compared CMJ power performance at 0.25, 4, 8, 12, 16, and 20 minutes post stimulus (3RM back squat) (Kilduff et al., 2007). These investigators demonstrated a window of opportunity regarding potentiation effects following 8 minutes of recovery (Kilduff, 2007). These investigators followed up with a subsequent study and reported similar results (Kilduff et al., 2008). Crewther and colleagues (2011) administered a squat protocol (1 x 3RM) and measured CMJ performance at 0.25, 4, 8, 12, and 16 minutes post-squat. Significant performance improvements were noted at 4, 8 and 12 minutes post, but a significant decrement in performance was noted 16 minutes post-stimulus. Wilson et al. (2013) in their meta-analysis concluded that PAP dissipates likely with rest intervals longer than 30 minutes. However, there was little to no data to support that extended recovery period.

PAP and Fatigue:

During repetitive stimulation, two opposing processes are ongoing inside a muscle cell; one that enhances muscular performance (PAP), and one that decreases muscle performance (fatigue). Optimal performance enhancements occur when fatigue is minimized and potentiation is optimal (Hodgson, 2005). The challenge is in creating a protocol that optimizes the balance between fatigue and potentiation. If the rest interval is too short the effects of fatigue may outweigh the effects of potentiation, on the other hand, if the rest interval is too long, potentiation effects may be diminished. Both situations will produce less than optimal results. To

date, an optimal rest interval for PAP remains elusive. An important aspect of skeletal muscle properties found regarding both fatigue and PAP is the differing time courses of recovery.

Considering the cascade of events associated with muscle contraction, failure could occur in a number of different steps. Primarily, a decrease in peak or mean free calcium (Ca^{2+}) in the myoplasm has been observed as a consequence of fatigue from previous activity (Allen, 1989; Westerblad and Allen, 1996; and Westerblad, duty and Allen, 1993). In addition, a decrease in Ca²⁺ sensitivity may occur of repetitive contraction (Westerblad, Lee, Lännergren and Allen 1991). This may occur due to a decrease in the Ca^{2+} / troponin binding affinity or a decrease in force produced by each cross bridge during contraction (Rassier, 2000). The decrease in Ca^{2+} sensitivity and Ca^{2+} / troponin binding affinity consequently, results in a decrease in force production for subsequent power performance, as muscular contraction depends on the presence of Ca^{2+} . For a given submaximal contraction, the contrasting effects of fatigue and potentiation acting synchronously, may result in enhanced, decreased or no change in subsequent contractions depending on the relative change in increased Ca^{2+} sensitivity and decreased Ca^{2+} concentration (Rassier, 2000). Clearly, fatigue and potentiation have opposing effects on force production and it is the net balance between the processes that determines potentiating potential (Hodgson, 2005). As discussed earlier, volume (multiple versus single sets), intensity and rest intervals contribute to varying degrees to both fatigue and potentiation. These factors appear to be dependent upon the subjects' training status (untrained, trained, and competitive trained). It does appear that fatigue abates at a faster rate than PAP, and thus the optimal rest interval will be defined as the point where the processes that enhance force remain existent while the processes that diminish force dissipate.

CHAPTER THREE: METHODS

Participants

Eleven men (25.18 \pm 3.60 y; 90.67 \pm 12.70 kg) with an average 1RM squat of 178.3 \pm 36.7 volunteered to participate in this study. Following an explanation of all procedures, risks, and benefits, each participant gave his informed consent prior to participation in this study. The Institutional Review Board of the University approved the research protocol. For inclusion in the study, participants must have had no positive risk factors on the administered PAR-Q; had at least one year of resistance training experience; and have been able to back squat at least their body weight. Participants were instructed not to perform any lower body exercise for at least 72 hours prior to testing sessions. In addition, subjects were instructed not to consume any energy or caffeine supplements prior to each testing session.

Study protocol

The study protocol is depicted in Figure 1. Participants reported to the Human Performance Lab (HPL) on five separate occasions separated by at least one week. During the first visit, participants became familiar with the technique required to perform the vertical jumps (counter movement jump and vertical jump for height), as well as obtain baseline ultrasound measures and images. Participants reported back to the HPL on four additional occasions separated by at least one week to complete testing trials. During the second visit (1st testing visit) participants were tested on their one repetition max (1RM) squat protocol. The reason for having this trial in advance was to determine loads for percentages of the later visits. During the three subsequent visits, participants were randomly assigned to one of the three protocols: 1) a moderate intensity (MI) squat protocol using 75% of the participant's 1RM; 2) a high intensity (HI) squat protocol using 90% of the participant's 1RM; 3) no workout which served as a control session (CTL).

Upon arrival at the HPL, participants rested in the supine position for 15 min. to account for any fluid shifts. Baseline (BL) ultrasound measures of the vastus lateralis and rectus femoris were then performed. Immediately following BL ultrasound measures, participants performed a standardized dynamic warm-up consisting of 5-min. on a cycle ergometer, ten body weight squats, ten body weight walking lunges, ten dynamic walking hamstring stretches, and ten dynamic walking quadriceps stretches. Participants then performed their first vertical jump testing (VJPRE), followed by their designated squat protocol. Following the squat protocol, participants rested supine for 8 min. during which ultrasound measures were again measured. Participants then performed the vertical jump testing protocol (VJ8P). A third ultrasound measure was performed following the second jump protocol. Participants then perform an additional jump test (VJ20P) at 20 min post exercise intervention. All testing occurred at the same time of day and was monitored by a Certified Strength and Conditioning Specialist.

Maximum Strength Testing

The 1RM squat assessment was performed using methods previously described (Hoffman, 2006). Each participant performed a warm-up set using a resistance that was approximately 40-60% of their perceived maximum, and then performed 3-4 subsequent attempts to determine the 1RM. A 3-5 min. rest period was provided between each attempt. Trials not meeting the range of motion criteria for each exercise were discarded. The squat

exercise required the participant to place an Olympic weightlifting bar across the trapezius muscle at a self-selected location. Each participant descended to the parallel position (that was monitored closely by the certified staff), which was attained when the greater trochanter of the femur reached the same level as the knee. The participant then lifted the weight until full knee extension.

Potentiation Protocols

During the moderate intensity protocol (MI), participants performed 3 sets of 10 repetitions at 75% 1RM. During the high intensity protocol (HI), participants performed 3 sets of 3 repetitions at 90% 1RM. A rest interval of 3-min occurred between each set for both MI and HI trials. During the 1RM protocol, participants performed the 1 repetition maximum (1RM) testing protocol previously described, and during the control protocol (CTL) participants performed the same testing routine with the exception of the squat intervention. The squat protocols that were administered were chosen based off of previous potentiation studies that elicited the greatest potentiation results using those intensities (Wilson et al, 2013).





Visual Analog Scale

Before every vertical jump testing series, participants were instructed to assess their subjective feelings of power using a 15-cm visual analog scale (VAS) at the time points; BL, VJ8P, and VJ20P. The scale was anchored by the words "Lowest" and "Highest" to represent extreme ratings where the greater measured value represented the greater feeling. The question was structured as "My level of power is". The validity and reliability of VAS has been previously established (Lee et al., 1991).
Vertical Jump Testing

Vertical jump height was assessed using a Vertical Jump Testing station (Uesaka Sport, Colorado Springs, CO). Before testing, each participant's standing vertical reach height was determined by colored squares located along the vertical neck of the device. These squares correspond with similarly colored markings on each horizontal tab, which indicate the vertical distance (in inches) from the associated square. Vertical jump height was determined by the indicated distance on the highest tab reached following 3 maximal countermovement jump (CMJ) attempts. Peak (PVJP) and mean (MVJP) vertical jump power was determined from a TendoTM Power Output Unit (Tendo Sports Machines, Trencin, Slovak Republic) that was attached at the waist of the participant during the vertical jump assessment. The TendoTM unit consists of a transducer that measured velocity (m/s), defined as linear displacement over time. Subsequently, the velocity of each jump was calculated and power determined.

Following vertical jump height testing, participants performed 3 additional CMJ's with their hands remaining on their hips through the entire range of motion. PVJP and MVJP was recorded for each jump using the TendoTM unit and used for subsequent analysis. Test-retest reliability for the TendoTM unit in our laboratory has consistently shown r > 0.90.

Ultrasonography

Measurements of pennation angle (PANG), and cross sectional area (CSA), were collected via non-invasive ultrasonography. All measures were collected on the rectus femoris (RF) and vastus lateralis (VL) of the dominant leg. This technique uses sound waves at fixed frequencies to create in vivo, real time images of the deep limb musculature. For all visits, participants were instructed to wear shorts to expose the superficial dermis of the anterior and lateral thigh. Participants rested in a supine position for 15 min. to with a rolled towel beneath the knee to allow for a 10-15° bend as measured by a goniometer. A 12 MHz linear probe scanning head (General Electric LOGIQ P5, Wauwatosa, WI, USA) was used to optimize spatial resolution (Thomaes et al., 2012). The probe was coated with water soluble transmission gel (Aquasonic 100 ultrasound transmission gel, Parker Laboratories, Inc. Fairfield, New Jersey) and positioned on the surface of the skin to provide acoustic contact without depressing the dermal layer to collect the image. Measures of muscle cross-sectional area (CSA) were obtained using a sweep of the muscle in the extended field of view mode with gain set to 50 dB and image depth to 5cm, while longitudinal images of pennation angle (PANG) were taken using B-mode ultrasound (Cadore et al., 2012). Following scanning, all images were analyzed offline using ImageJ (National Institutes of Health, Bethesda, MD, USA, version 1.45s), an image analysis software available through the National Institute of Health. For these analyses, a known distance of 1cm shown in the image was used to calibrate the software program (Chapman et al., 2008).

The anatomical location for all ultrasound measures was standardized for each muscle in all participants. For RF measurements, the participant was placed supine on an examination table, according to the American Institute of Ultrasound in Medicine, with the legs extended but relaxed and with a rolled towel beneath the popliteal fossa allowing for a 10° bend in the knee as measured by a goniometer (Bemben, 2002). For VL measurements, the participant was placed on their side with the legs together and relaxed allowing for a 10° bend in the knee as measured by a goniometer. CSA was determined using the same images for the RF and VL muscles. Measurements of the RF was taken in the sagittal plane parallel to the long axis of the femur and

scanning occurred in the axial plane, perpendicular to the tissue interface at 50% of the distance between the anterior, inferior iliac to the proximal border of the patella. VL was measured at 50% of the distance from the most prominent point of the greater trochanter to the lateral condyle. Three consecutive images were analyzed and averaged using the polygon tracking tool in the ImageJ software to obtain as much lean muscle as possible without any surrounding bone or fascia for CSA. The ICCs for rectus femoris and vastus lateralis CSA were 0.98 (SEM = 0.52 cm²) and 0.99 (SEM = .33 cm²), respectively.

Measures of PANG were taken at the same site described for CSA (Abe et al., 1998), but with the probe oriented longitudinal to the muscle tissue interface for both the RF and VL. Within each muscle, three consecutive images were analyzed and averaged offline (Thomaes et al., 2012). Muscle fiber PANG was determined as the intersection of the fascicles with the deep aponeurosis. ICCs for RF and VL PANG were 0.99 (SEM = $.20^{\circ}$) and 0.81 (SEM = 1.28°), respectively.

Statistical Analysis

A Kolmogorov-Smirnov test was used to test the normal distribution of the data. Significance was set at $p \le 0.05$. In addition, data were analyzed using magnitude based inferences, interpreted through the analysis of the magnitude of the relationships (Batterham & Hopkins, 2006; Cohen, 1988). Statistical Software (SPSS; V. 20.0, SPSS Inc., Chicago, IL) was used to calculate Pearson product-moment correlation coefficients, which along with the sample size were input into the correlation coefficient statistic on a published spreadsheet (Batterham & Hopkins, 2006) to determine the magnitude of the effect. The threshold values for positive or negative correlations were set at 0.1, which was previously reported to be the smallest clinically important correlation (Cohen, 1988). Inferences on correlations were determined as positive, trivial, or negative according to methods previously described (Batterham & Hopkins, 2006) and were based on the confidence interval range relative to the smallest clinically meaningful effect to be positive, trivial, or negative. The percent chances of a positive or negative outcome was evaluated with the following scale: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely; 25–75%, possible; 75–95%, likely; 95–99% very likely; and >99% almost certain. If the likely range substantially overlapped both positive and negative values, it was inferred that the outcome was unclear (Hopkins, Batterham, Marshall, & Hanin, 2009). In the event of a positive or negative result, the correlation was re-examined at 0.3 and 0.5 threshold values to determine if the low correlation was in fact, a moderate or high correlation respectively (Cohen, 1988).

CHAPTER FOUR: RESULTS

Comparisons of vertical jump height, power and countermovement jump power can be seen in Table 1. No performance improvements were noted in any of the potentiation protocols. Interestingly, performances at 8 minutes and 20 minutes post-exercise tended to decline or not change following these different exercise protocols.

		MI	HI	CON	1RM
VJ Height	Pre	26.06 ± 4.41	25.81 ± 4.18	26.25 ± 4.76	26.01 ± 4.94
(cm)	8P	24.58 ± 4.04	25.03 ± 4.33	26.12 ± 4.86	25.43 ± 4.76
	20P	24.67 ± 4.05	24.72 ± 4.27	25.76 ± 4.74	25.44 ± 4.92
PVJP (w)	Pre	2506 ± 689	2423 ± 599	2579 ± 890	2900 ± 916
	8P	2342 ± 631	2439 ± 654	2584 ± 991	2792 ± 916
	20P	2449 ± 629	2470 ± 602	2565 ± 1190	2831 ± 913
MVJP (w)	Pre	1323 ± 359	1500 ± 730	1225 ± 282	1280 ± 306
	8 P	1220 ± 257	1307 ± 330	1253 ± 308	1251 ± 309
	20P	1256 ± 284	1347 ± 360	1223 ± 289	1247 ± 316

 Table 1: Vertical Jump Performance Comparisons between All Testing Protocols

VJ = vertical jump; PVJP = peak vertical jump power; MVJP = mean vertical jump power.

Magnitude based inferences comparing the change (Δ) scores in vertical jump height from baseline to 8-min post exercise are depicted in Table 2. Changes in jump height between these time points were likely decreased in MI compared to HI and 1RM, and very likely decreased compared to CTL. Changes in CTL were possibly greater at this time than that seen at HI. Similarly, changes in vertical jump height at HI and 1RM were likely decreased compared to CTL. No other changes were noted.

Magnitude based inferences comparing the Δ scores in vertical jump height from baseline to 20-min post exercise are shown in Table 3. Changes in jump height between these time points were very likely decreased in MI and HI compared to CTL. In addition, changes in jump height

were likely decreased at MI and HI compared to 1RM. No other changes were noted.

				Ind. SE					
Jump				Diff/				Mean	
height	Group	Group	P -	Thre	Posi	Trivi	Negati	Differe	Interpretatio
	1	2	Value	sh	tive	al	ve	nce	n
MI vs	-1.5 ±	-0.7 \pm	0.127	0.21	3	9.6	87.4	-0.8 \pm	Likely
HI	1.5	0.7						0.8	Decreased
MI vs	-1.5 ±	-0.1 ±	0.014	0.21	0.3	1.3	98.4	-1.4 ±	Very Likely
CTL	1.5	0.6						0.9	Decreased
MI vs	-1.5 ±	$-0.7 \pm$	0.101	0.21	2.2	8.7	89.2	$-0.8 \pm$	Likely
1RM	1.5	0.7						0.8	Decreased
HI vs	$-0.7 \pm$	-0.1 ±	0.011	0.21	0.1	4.1	95.9	$-0.6 \pm$	Very Likely
CTL	0.7	0.6						0.4	Decreased
HI vs	$-0.7 \pm$	$-0.7 \pm$	0.044	0.21	0	100	0	0 ± 0	Almost
1RM	0.7	0.7							Certainly
									Trivial
CTL	-0.1 ±	$-0.7 \pm$	0.076	0.21	88.1	10.9	1	$0.6 \pm$	Likely
vs	0.6	0.7						0.6	Increased
1RM									

 Table 2: Magnitude Based Inferences on Changes in Vertical Jump Height between Baseline and
 8-Min Post-Exercise

PVJP	Grou	Grou	P -	Ind.	Positi	Trivi	Negati	Mean	Interpretati
	p 1	p 2	Value	SE	ve	al	ve	Differe	on
				Diff/				nce	
				Thre					
				sh					
MI vs	-1.4 ±	-1.1 ±	0.492	0.19	13.1	27.3	59.6	-0.3 ±	Unclear
HI	1.2	0.8						0.74	
MI vs	$-1.4 \pm$	-0.3 \pm	0.025	0.19	0.5	2.5	97	-1.1 ±	Very Likely
CTL	1.2	0.9						0.78	Decreased
MI vs	-1.4 ±	-0.7 \pm	0.092	0.19	1.8	8.9	89.3	-0.7 \pm	Likely
1RM	1.2	0.7						0.68	Decreased
HI vs	-1.1 \pm	-0.3 \pm	0.025	0.19	0.3	3.7	95.9	-0.8 \pm	Very Likely
CTL	0.8	0.9						0.57	Decreased
HI vs	-1.1 ±	-0.7 \pm	0.044	0.19	0.2	13.9	85.9	-0.4 \pm	Likely
1RM	0.8	0.7						0.32	Decreased
CTL vs	-0.3 ±	-0.7 ±	0.313	0.19	70	23.1	7	0.4 ±	Unclear
1RM	0.9	0.7						0.67	

 Table 3: Magnitude Based Inferences on Changes in Vertical Jump Height between Baseline and 20-Min Post-Exercise

Magnitude based inferences comparing the Δ scores in PVJP from baseline to 8-min post exercise are depicted in Table 4. Changes in PVJP between these time points were likely decreased in MI compared to HI, 1RM and CTL. Changes in CTL were possibly greater at this time than that seen at HI.

PVJP	Group 1	Group	P -	Ind.	Positi	Trivi	Negati	Mean	Interpretati
		2	Value	SE	ve	al	ve	Differe	on
				Diff/				nce	
				Thre					
				sh					
MI vs	$-402.2 \pm$	90.8 ±		116.				-490 ±	Likely
HI	735.8	758.6	0.14	7	3.50	9.10	87.40	550	Decreased
		105.4							
MI vs	-402.2 \pm	±		116.				$-510 \pm$	Likely
CTL	735.8	259.8	0.04	7	0.80	4.90	94.40	410	Decreased
		-32.1							
MI vs	-402.2 \pm	<u>±</u>		116.		12.3		$-370 \pm$	Likely
1RM	735.8	308.9	0.147	7	3.20	0	84.50	430	Decreased
		105.4							Most
HI vs	$90.8 \pm$	±		116.		100.		-15 ±	Likely
CTL	758.6	259.8	0.05	7	0.00	00	0.00	12	Trivial
		-32.1							
HI vs	$90.8 \pm$	±		116.				$120 \pm$	
1RM	758.6	308.9	0.953	7	50.10	4.50	45.40	3600	Unclear
		-32.1							
CTL vs	$105.4 \pm$	±		116.		40.8		140 ±	Possibly
1MR	259.8	308.9	0.272	7	56.70	0	2.50	210	Increased

Table 4: Magnitude Based Inferences on Changes in Peak Vertical Jump Power betweenBaseline and 8-Min Post-Exercise

PVJP = peak vertical jump power

Magnitude based inferences comparing the Δ scores in PVJP from baseline to 20-min post exercise can be seen in Table 5. Changes in PVJP between these time points were likely decreased in MI compared to 1RM and CTL. All other comparisons were unclear or trivial.

				Ind.					
				SE					
				Diff					
				/				Mean	
		Group	P -	Thre	Positi	Trivi	Negati	Differe	Interpretati
PVJP	Group 1	2	Value	sh	ve	al	ve	nce	on
MI vs	$-302 \pm$	$97.5 \pm$	0.21	111.	5.70	12.4	81.90	$-400 \pm$	Unclear
HI	680.3	764.4		6		0		530	
MI vs	$-302 \pm$	$58.2 \pm$	0.11	111.	2.10	11.2	86.60	$-360 \pm$	Likely
CTL	680.3	242.1		6		0		380	Decreased
MI vs	$-302 \pm$	53.1 ±	0.14	111.	3.10	12.3	84.70	$-360 \pm$	Likely
1RM	680.3	346.7		6		0		400	Decreased
HI vs	$97.5 \pm$	$58.2 \pm$	0.12	111.	0.50	99.5	0.00	39 ±	Very
CTL	764.4	242.1		6		0		42	Likely
									Trivial
HI vs	97.5 ±	53.1 ±	0.87	111.	40.50	30.6	28.90	44 ±	Unclear
1RM	764.4	346.7		6		0		480	
				_		_			
CTL	$58.2 \pm$	53.1 ±	0.97	111.	20.70	60.7	18.60	5.1 ±	Unclear
VS	242.1	346.7		6		0		220	
1RM									

Table 5: Magnitude Based Inferences on Changes in Peak Vertical Jump Power between Baseline and 20-Min Post-Exercise

PVJP = peak vertical jump power

Magnitude based inferences comparing the Δ scores in MVJP from baseline to 8-min post exercise can be observed in Table 6. Changes in MVJP between these time points were likely decreased in both MI and HI compared to CTL. Changes in 1RM were possibly less than that seen at HI. All other comparisons between groups for this measure were unclear or possibly trivial.

Table 6: Magnitude Based Inferences on Changes in Mean Vertical Jump Power between Baseline and 8-Min Post-Exercise

				Ind.					
				SE					
			P -	Diff/				Mean	
	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
MVJP	1	Group 2	ue	sh	ve	al	ve	nce	n
	-200.3								
MI vs	<u>±</u>	-161.1		74.0		25.7		-39 ±	
HI	478.8	± 545.8	0.86	5	30.50	0	43.80	380	Unclear
	-200.3								
MI vs	<u>+</u>	$31.2 \pm$		74.0		12.3		$-230 \pm$	Likely
CTL	478.8	92.7	0.13	5	2.50	0	85.10	250	Decreased
	-200.3								
MI vs	±	-13.7 ±	0.23	74.0		17.9		-190 ±	
1RM	478.8	110.3	4	5	5.30	0	76.80	270	Unclear
	-161.1								
HI vs	\pm	$31.2 \pm$		74.0		15.2		-190 ±	Likely
CLT	545.8	92.7	0.13	5	2.10	0	82.80	210	Decreased
	-161.1								
HI vs	\pm	-13.7 ±	0.26	74.0		23.7		$-150 \pm$	Possibly
1MR	545.8	110.3	3	5	4.90	0	71.40	220	Decreased
CTL vs	31.2 ±	-13.7 ±	0.31	74.0		73.9		45 ±	Possibly
1MR	92.7	110.3	4	5	25.50	0	0.60	75	Trivial

MVJP = mean vertical jump power

Magnitude based inferences comparing the Δ scores in MVJP from baseline to 20-min post exercise are depicted in Table 7. Changes in MVJP between these time points were likely decreased in MI compared to CTL, and possibly decreased in HI compared to CTL. All other comparisons between groups for this measure were unclear or possibly trivial.

				Ind.					
			Р-	Diff/				Mean	
	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
MVJP	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	-166.2	$-80.6 \pm$	0.72	79.1	25.10	23.9	51.10	-86 ±	Unclear
HI	±	643.6	6	2		0		420	
	471.5								
MI vs	-166.2	21.7 ±	0.21	79.1	3.90	19.0	77.00	-190 ±	Likely
CTL	±	77.9		2		0		250	Decreased
	471.5								
MI vs	-166.2	-24.9 ±	0.35	79.1	7.90	25.9	66.20	-140 ±	Unclear
1RM	±	93.6	1	2		0		260	
	471.5								
								100	
HI vs	$-80.6 \pm$	21.7 ±	0.22	79.1	2.10	36.5	61.40	$-100 \pm$	Possibly
CTL	643.6	77.9		2		0		140	Decreased
TTT 1	00.6	24.0	0.61	70.1	11.70	16.0	41.50	56	T T 1
HI VS I	-80.6 ±	-24.9 ±	0.61	/9.1	11.70	46.8	41.50	-56 ±	Unclear
	643.6	93.6	2	2		0		190	
CTL ve	21.7 +	_24.9 ±	0.21	70.1	10.30	80.6	0.10	<i>1</i> 7 +	Likely
		-2 4 .7 ±	0.21	2	19.50	00.0	0.10	+/ <u>+</u>	
	//.9	93.6	9	2		U		63	I rivial
		1	I			I	1	1	

Table 7: Magnitude Based Inferences on Changes in Mean Vertical Jump Power between Baseline and 20-Min Post-Exercise

MVJP = mean vertical jump power

Comparisons of muscle architecture changes in the RF and VL muscles can be seen in Table 8.

		MI	HI	CTL	1RM
RFCSA	Pre	16.8 ± 3.0	17.2 ± 2.9	17.5 ± 3.0	16.9 ± 2.7
(\mathbf{cm}^2)	8P	17.4 ± 3.1	17.6 ± 3.2	17.8 ± 3.2	17.4 ± 2.6
	20P	17.3 ± 2.9	17.5 ± 2.9	17.5 ± 3.0	17.1 ± 2.7
RFPNG	Pre	15.4 ± 2.2	15.5 ± 2.1	15.5 ± 2.3	15.3 ± 2.8
(°)	8P	15.4 ± 2.6	15.1 ± 3.3	16.1 ± 4.0	14.8 ± 3.0
	20P	16.8 ± 3.29	14.8 ± 1.5	16.7 ± 4.3	15.4 ± 2.9
VLCSA	Pre	40.0 ± 6.7	39.6 ± 6.5	40.2 ± 6.3	38.2 ± 7.4
(cm^2)	8P	41.6 ± 7.0	40.6 ± 6.5	40.6 ± 6.2	39.5 ± 7.4
	20P	41.5 ± 7.2	40.2 ± 6.7	39.9 ± 6.5	39.3 ± 7.2
VLPNG	Pre	14.6 ± 2.4	14.1 ± 3.3	14.1 ± 2.2	15.6 ± 3.8
(°)	8 P	15.3 ± 2.6	15.3 ± 3.5	15.8 ± 2.7	15.1 ± 3.6
	20P	16.1 ± 3.4	15.3 ± 2.9	14.8 ± 2.6	14.4 ± 3.9

Table 8: Muscle Architecture Comparisons between All Testing Protocols

RFCSA = rectus femoris cross-sectional area; RFPNG = rectus femoris pennation angle; VLCSA

= vastus lateralis cross-sectional area; VLPNG = vastus lateralis pennation angle.

Magnitude based inferences comparing the Δ scores in CSA of the RF from baseline to 8min post exercise are depicted in Table 9. Changes in CSA between these time points were likely greater in MI compared to CTL. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
			P -	Diff/				Mean	
CSA-	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
RF	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	0.6 ±	0.3 ±	0.42	0.12	68.90	18.2	13.00	0.3 ±	Unclear
HI	0.6	0.6				0		0.62	
MI vs	0.6 ±	0.3 ±	0.16	0.12	81.00	16.3	2.70	0.3 ±	Likely
CTL	0.6	0.3				0		0.35	Increased
MI vs	0.6 ±	$0.4 \pm$	0.70	0.12	56.10	16.7	27.20	0.2 ±	Unclear
1RM	0.6	0.8				0		0.89	
HI vs	0.3 ±	0.3 ±	0.15	0.12	0	100	0	0 ± 0	Almost
CTL	0.6	0.3							Certainly
									Trivial
HI vs	0.3 ±	$0.4 \pm$	0.67	0.12	17.50	35.8	46.70	-0.1 ±	Unclear
1RM	0.6	0.8				0		0.4	
						_			
CTL vs	0.3 ±	0.4 ±	0.50	0.12	7.30	47.9	44.80	-0.1 ±	Unclear
1RM	0.3	0.8				0		0.25	

Table 9: Magnitude Based Inferences on Changes in Cross-Sectional Area of the Rectus Femoris Muscle between Baseline and 8-Min Post-Exercise

CSA-RF = cross-sectional area of the rectus femoris

Magnitude based inferences comparing the Δ scores in CSA of the RF from baseline to 20-min post exercise can be observed in Table 10. Changes in CSA between these time points were likely greater in HI compared to CTL. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
			P -	Diff/				Mean	
CSA -	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
RF	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	0.5 ±	0.2 ±	0.24	0.11	77.00	17.3	5.70	0.3 ±	Unclear
HI	0.6	0.6				0		0.43	
MI vs	0.5 ±	0 ± 0.5	0.09	0.11	90.90	7.00	2.00	0.5 ±	Likely
CTL	0.6							0.48	Increased
MI vs	0.5 ±	0.2 ±	0.27	0.11	75.90	17.5	6.60	0.3 ±	Unclear
1RM	0.6	0.5				0		0.45	
HI vs	0.2 ±	0 ± 0.5	0.09	0.11	77.90	21.5	0.60	0.2 ±	Likely
CTL	0.6					0		0.19	Increased
HI vs	0.2 ±	$0.2 \pm$	0.59	0.11	0	100	0	0 ± 0	Almost
1RM	0.6	0.5							Certainly
									Trivial
CTL vs	0 ± 0.5	$0.2 \pm$	0.44	0.11	11.70	25.0	63.30	-0.2 ±	Unclear
1RM		0.5				0		0.44	

Table 10: Magnitude Based Inferences on Changes in Cross-Sectional Area of the Rectus Femoris Muscle between Baseline and 20-Min Post-Exercise

CSA-RF = cross-sectional area of the rectus femoris

Magnitude based inferences comparing the Δ scores in CSA of the VL from baseline to 8-min post exercise are depicted in Table 11. Changes in CSA between these time points were likely and possibly greater in MI and HI, respectively compared to CTL. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
				Diff					
			P -	/				Mean	
CSA-	Group	Group	Val	Thr	Positi	Triv	Negati	Differ	Interpretati
VL	1	2	ue	esh	ve	ial	ve	ence	on
MI vs	1.6 ±					33.4		0.6 ±	
HI	2.4	1 ± 0.6	0.47	0.47	56.30	0	10.20	1.4	Unclear
MI vs	$1.6 \pm$	$0.5 \pm$				19.6		$1.1 \pm$	Likely
CTL	2.4	1.4	0.22	0.47	76.10	0	4.30	1.5	Increased
MI vs	$1.6 \pm$	$1.1 \pm$				23.9		$0.5 \pm$	
1RM	2.4	3.9	0.73	0.47	50.80	0	25.30	2.5	Unclear
HI vs		$0.5 \pm$				45.7		$0.5 \pm$	Possibly
CTL	1 ± 0.6	1.4	0.23	0.47	52.90	0	1.40	0.69	Increased
HI vs									Most
1RM		$1.1 \pm$				100.		-0.1 ±	Likely
	1 ± 0.6	3.9	0.29	0.47	0.00	00	0.00	0.16	Trivial
CTL vs	0.5 ±	1.1 ±				25.4		-0.6 ±	
1RM	1.4	3.9	0.64	0.47	20.70	0	54.00	2.2	Unclear

Table 11: Magnitude Based Inferences on Changes in Cross-Sectional Area of the Vastus Lateralis Muscle between Baseline and 8-Min Post-Exercise

CSA-VL= cross-sectional area of the vastus lateralis

Magnitude based inferences comparing the Δ scores in CSA of the VL from baseline to 20-min post exercise can be seen in Table 12. Changes in CSA between these time points were likely greater in both MI and HI compared to CTL. Comparison between changes in MI and HI revealed possible greater changes in MI compared to HI. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
			P -	Diff/				Mean	
CSA -	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
VL	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	1.5 ±	0.7 ±	0.24	0.43	70.70	25.4	4.00	$0.8 \pm$	Possibly
HI	2.1	0.9				0		1.1	Increased
MI vs	1.5 ±	-0.2 ±	0.04	0.43	93.90	5.50	0.70	1.7 ±	Likely
CTL	2.1	1.5	3					1.4	Increased
MI vs	1.5 ±	1 ± 3.3	0.66	0.43	52.40	26.6	21.00	0.5 ± 2	Unclear
1RM	2.1					0			
HI vs	0.7 ±	-0.2 ±	0.04	0.43	86.30	13.5	0.20	0.9 ±	Likely
CTL	0.9	1.5	3			0		0.72	Increased
HI vs	$0.7 \pm$	1 ± 3.3	0.13	0.43	0.10	75.1	24.90	-0.3 ±	Likely
1RM	0.9					0		0.33	Trivial
CTL vs	-0.2 ±	1 ± 3.3	0.30	0.43	8.30	17.0	74.70	-1.2 ±	Unclear
1RM	1.5					0		2	

Table 12: Magnitude Based Inferences on Changes in Cross-Sectional Area of the Vastus Lateralis Muscle between Baseline and 20-Min Post-Exercise

CSA-VL= cross-sectional area of the vastus lateralis

Magnitude based inferences comparing the Δ scores in PANG of the RF from baseline to 8-min post exercise can be observed in Table 13. Changes in PANG between these time points were unclear.

				Ind.					
				SE					
				Diff					
			P -	/				Mean	
PANG	Group	Group	Val	Thre	Positi	Triv	Negati	Differe	Interpretatio
-RF	1	2	ue	sh	ve	ial	ve	nce	n
MI vs		-0.4 \pm	0.72			32.1			
HI	0 ± 2.5	2.3	9	0.51	46.20	0	21.70	0.4 ± 2	Unclear
MI vs		$0.6 \pm$				28.3		-0.6 ±	
CTL	0 ± 2.5	3.5	0.63	0.51	18.80	0	52.90	2.1	Unclear
MI vs		-0.5 ±	0.61			34.6		0.5 ±	
1RM	0 ± 2.5	1.8	5	0.51	49.70	0	15.80	1.7	Unclear
HI vs	-0.4 ±	0.6 ±				17.2		-1 ±	
CTL	2.3	3.5	0.63	0.51	23.40	0	59.40	3.5	Unclear
HI vs	-0.4 ±	-0.5 ±	0.43			99.8		0.1 ±	Most Likely
1RM	2.3	1.8	7	0.51	0.20	0	0.00	0.22	Trivial
CTL vs	0.6 ±	-0.5 ±	0.35			21.6			
1RM	3.5	1.8	8	0.51	69.20	0	9.30	1.1 ± 2	Unclear
			-	0.01		-		· -	

Table 13: Magnitude Based Inferences on Changes in Pennation Angle of the Rectus Femoris Muscle between Baseline and 8- Min Post-Exercise

PANG-RF = pennation angle of the rectus femoris

Magnitude based inferences comparing the Δ scores in PANG of the RF from baseline to 20-min post exercise can be seen in Table 14. Changes in PANG between these time points were likely greater in MI compared to HI. In addition, changes in PANG at these time points for HI was possibly lower than that seen at 1RM. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
				Diff					
			Р-					Mean	
PANG	Group	Group	Val	Thre	Positi	Triv	Negati	Differe	Interpretatio
- RF	1	2	ue	sh	ve	ial	ve	nce	n
MI vs	1.4 ±	-0.7 ±	0.06					2.1 ±	Likely
HI	3.1	1.9	9	0.60	90.80	8.00	1.10	1.9	Increased
MI vs	1.4 ±	$1.2 \pm$				39.2		0.2 ±	
CTL	3.1	3.5	0.86	0.60	36.40	0	24.40	1.9	Unclear
MI vs	1.4 ±					20.1		1.4 ±	
1RM	3.1	0 ± 3.1	0.30	0.60	72.70	0	7.20	2.3	Unclear
HI vs	-0.7 ±	1.2 ±						-1.9 ±	
CTL	1.9	3.5	0.86	0.60	40.90	4.30	54.80	19	Unclear
HI vs	-0.7 ±					40.6		-0.7 ±	Possibly
1RM	1.9	0 ± 3.1	0.14	0.60	0.50	0	59.00	0.78	Decreased
CTL vs	1.2 ±					22.5		1.2 ±	
1RM	3.5	0 ± 3.1	0.42	0.60	65.80	0	11.70	2.5	Unclear

Table 14: Magnitude Based Inferences on Changes in Pennation Angle of the Rectus Femoris Muscle between Baseline and 20-Min Post-Exercise

PANG-RF = pennation angle of the rectus femoris

Magnitude based inferences comparing the Δ scores in PANG of the VL from baseline to 8-min post exercise are depicted in Table 15. Changes in PANG between these time points were likely and very likely greater in MI and CTL, respectively compared to CTL. All other comparisons between groups for this measure were unclear.

				Ind.					
				SE					
			P -	Diff/				Mean	
PANG-	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
VL	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	0.7 ±	1.3 ±	0.46	0.34	12.60	24.5	62.90	-0.6 ±	Unclear
HI	1.6	1.7				0		1.4	
MI vs	$0.7 \pm$	1.1 ±	0.58	0.34	15.40	31.0	53.60	-0.4 ±	Unclear
CTL	1.6	1.7				0		1.2	
MI vs	$0.7 \pm$	-0.5 ±	0.07	0.34	90.10	8.40	1.40	$1.2 \pm$	Likely
1RM	1.6	1.5	9					1.1	Increased
HI vs	1.3 ±	1.1 ±	0.58	0.34	35.20	57.7	7.20	0.2 ±	Unclear
CTL	1.7	1.7				0		0.6	
HI vs	1.3 ±	-0.5 ±	0.85	0.34	55.80	2.60	41.60	$1.8 \pm$	Unclear
1RM	1.7	1.5	8					17	
CTL vs	1.1 ±	-0.5 ±	0.02	0.34	96.30	3.20	0.50	1.6 ±	Very Likely
1RM	1.7	1.5	7					1.2	Increased

Table 15: Magnitude Based Inferences on Changes in Pennation Angle of the Vastus Lateralis Muscle between Baseline and 8-Min Post-Exercise

PANG-VL= pennation angle of the vastus lateralis

Magnitude based inferences comparing the Δ scores in PANG of the VL from baseline to 20-min post exercise can be seen in Table 16. Changes in PANG between these time points were likely greater in MI and CTL, respectively compared to CTL. All other comparisons between groups for this measure were unclear.

				Ind.					
			Р-	SE Diff/				Mean	
PANG-	Group		Val	Thre	Positi	Trivi	Negati	Differe	Interpretatio
VL	1	Group 2	ue	sh	ve	al	ve	nce	n
MI vs	1.5 ±	1.3 ±				39.4		0.2 ±	
HI	2.5	2.2	0.85	0.55	36.90	0	23.80	1.8	Unclear
MI vs	1.5 ±	1.4 ±				43.7		0.1 ±	
CTL	2.5	2.6	0.92	0.55	31.70	0	24.70	1.6	Unclear
MI	15.	11.	0.04					26	T :11
MI VS	1.5 ±	-1.1 ±	0.04					2.0 ±	Likely
IRM	2.5	3.2	6	0.55	94.50	4.60	0.90	2.1	Increased
HI vs	1.3 ±	1.4 ±				43.7		-0.1 ±	
CTL	2.2	2.6	0.92	0.55	24.70	0	31.70	1.6	Unclear
TT	1.2	11.	0.02					2.4.	
HI VS	$1.3 \pm$	-1.1 ±	0.93					2.4 ±	
IRM	2.2	3.2	9	0.55	52.30	1.40	46.30	54	Unclear
CTL vs	1.4 ±	-1.1 ±	0.05					2.5 ±	Likely
1RM	2.6	3.2	9	0.55	93.30	5.50	1.20	2.2	Increased
			1						

Table 16: Magnitude Based Inferences on Changes in Pennation Angle of the Vastus Lateralis between Baseline and 20-Min Post-Exercise

PANG-VL= pennation angle of the vastus lateralis

Magnitude based inferences on Pearson correlation analyses are shown in Table 17. A likely negative relationship (r= -0.30) was observed between changes in CSA of the VL between baseline and 8-min post-exercise and changes in vertical jump height at the same time points. A likely negative relationship (r = -0.369) and a possible negative relationship (r = -0.229) was seen between changes in peak vertical jump power and changes in the CSA of the RF and VL, respectively between baseline and 20-min post-exercise. A likely negative relationship (p = -0.354) was observed between changes in PANG between baseline and 8-min post-exercise and mean vertical jump power. No other meaningful correlations were observed.

	Vertical Jump Height Baseline-8-Min Post							Vertical Jump Height Baseline-20-Min Post						
Varia ble	r	p- val ue	Positi ve	Triv ial	Negat ive	Interpret.	r	p- valu e	Posit ive	Triv ial	Negat ive	Interpret		
RCS A	0.05 5	0.7 21	4.9	77. 8	17.3	Likely trivial	- 0.18 8	0.22	0.6	52. 6	46.8	Possibly trivial		
RPN G	- 0.16 8	0.2 77	0.9	57. 6	41.5	Possibly trivial	- 0.17 6	0.25 2	0.7	55. 6	43.7	Possibly trivial		
VCS A	- .300 *	0.0 48	0.1	24. 7	75.3	Likely Negative	- 0.19 5	0.20 4	0.5	50. 8	48.7	Possibly trivial		
VPN G	- 0.23 9	0.1 18	0.2	39. 4	60.4	Possibly Negative	0.02	0.87 5	12.6	80	7.3	Unclear		
	Peak	Vertio	al Jum	o Powe Post	r Baselin	e – 8 min	Peak '	Vertica	l Jump	Power Post	Baseline	e – 20 min		
RCS A	0.15 6	0.3 12	1.1	60. 4	38.6	Possibly trivial	- .369 *	0.01 4	0	11. 9	88.1	Likely Negativ e		
RPN G	0.09 8	0.5 26	25.2	72. 1	2.7	Possibly trivial	- 0.03 5	0.82	6.4	79. 5	14.1	Unclear		
VCS A	- 0.09 7	0.5 33	2.7	72. 3	25	Possibly trivial	- 0.22 9	0.13 5	0.3	42	57.7	Possibly Negativ e		
VPN G	0.09 1	0.5 57	23.8	73. 2	3	Possibly trivial	- 0.04 6	0.76 7	5.6	78. 7	15.8	Unclear		
	Mean	Verti	cal Jum	p Powe Post	er Baselii	ne – 8 min	Mean Vertical Jump Power Baseline – 20 min Post							
RCS A	- 0.00 4	0.9 77	9.3	80. 6	10.2	Unclear	0.08 5	0.58 3	22.6	74. 2	3.3	Possibly trivial		
RPN G	0.04 6	0.7 65	15.8	78. 7	5.6	Unclear	0.01 3	0.93 5	11.2	80. 4	8.4	Unclear		
VCS A	- 0.05 8	0.7 11	4.7	77. 5	17.7	Likely trivial	0.00 3	0.98 5	10	80. 6	9.4	Unclear		
VPN G	- .354 *	0.0 18	0	14. 2	85.8	Likely negative	- 0.03 5	0.82	6.4	79. 5	14.1	Unclear		

Table 17: Magnitude Based Inferences on Pearson Correlation Measures on Comparisonsbetween Changes in Muscle Architecture and Jump Performance

CHAPTER FIVE: CONCLUSION/ DISCUSSION

The primary purpose of this study was to compare recommended potentiation protocols on subsequent jump performance, and to relate how acute changes in muscle architecture influence these effects in experienced, resistance trained participants. The main findings of this study showed that none of the potentiation protocols (MI, HI, and 1RM) resulted in any jump performance improvements. Interestingly, performances at 8 minutes and 20 minutes postexercise tended to decline or not change following all three protocols. However, muscle architecture responses did appear to be sensitive to the different potentiation protocols. The MI potentiation protocol did appear to have the greatest effects on changes in CSA and PANG in both the RF and VL muscles. Although no potentiation was noted in any of the protocols, results did indicate likely negative relationships between changes in CSA and PANG and changes in vertical jump performance. This suggests that greater increases in acute muscle swelling reduced the magnitude of performance decrements. Considering the acute changes observed in muscle architecture, the lack of any performance improvements may be related to either the conditioning level of the subjects, or possibly to the methodology employed in this study.

The lack of a response to any of the potentiation protocols contrasts with previous recommendations emanating from several meta-analyses (Gouvea et al., 2012; Wilson et al., 2013). In addition, previous research has also demonstrated a significant increase in vertical jump performance following 1-RM testing (Hoffman et al., 2007). These studies were the basis behind the development of the potentiation protocols employed in the present study. Although existing evidence does indicate that potentiation is more sensitive to the experienced individual

(Gouvea et al., 2012; Wilson et al., 2013), there also appears to be a difference between experienced and those who are experienced and competitive (Wilson et al., 2013). The present study recruited experienced resistance trained men, many of whom were former strength/power athletes. Although all were lifting weights on a regular basis, none of the participants were presently competing. Competitive athletes appear to have an advantage for performance potentiation that is related to their level of conditioning (Chiu et al., 2003; Khamoui et al., 2009; Kilduff et al., 2007).

The potentiation protocols used appeared to have fatigued the participants far greater than anticipated. Anecdotally, many of the subjects expressed their fatigue following all of the protocols. One participant, a former competitive athlete with a 1-RM squat of 238.6 kg even remarked that he was "spent" following the 1RM protocol, and that he felt that he would have experienced a greater potentiation effect when he was competing. It has been suggested that highly conditioned athletes have a greater ability than recreationally trained athletes to recover from a potentiating exercise protocol, likely related to the greater buffering capacity and resistance to muscle damage seen in the competitive athlete (McHugh, Connolly, Eaton, and Gleim, 1999; Wilson et al., 2013). In addition, the competitive athlete may benefit from the PAP due to a more efficient high end motor unit recruitment (Tillin and Bishop, 2009). Although potentiation and fatigue can occur within the same stimulus, Rassier and MacIntosh, (2000) suggest that there may be an optimal recovery period to reduce fatigue and for potentiation to be realized. In consideration of the importance of appropriate recovery time, we incorporated the most widely accepted rest interval time for potentiation (7-12 minutes) (Gouvea et al., 2012; Wilson et al., 2013). Despite this recovery time incorporated between all protocols, no PAP

response was observed. This may be a function of a high degree of variability among individuals using PAP. McCann and Flannagan (2010) examined rest intervals of 4 and 5 minutes following the squat (5RM) and power clean (5RM) exercises on vertical jump performance. Although significant improvements were noted at 4 minutes post-exercise for the group, when the data was analyzed separately the 5 minute rest interval was superior for many subjects compared to the 4 min rest interval.

Many factors such as training volume and intensity can increase fatigue and decrease the PAP response. The high volume, low intensity protocol used in MI may have resulted in a volume overload, while the high intensity, low volume used in the HI and 1RM protocols may have resulted in an intensity overload. Although all protocols appeared to result in a fatigue response that did not dissipate in time to enhance potentiation, the mechanisms generating the fatigue may have differed between the protocols. It is likely that the recreational resistance training that all subjects were presently performing was not sufficient to stimulate physiological adaptation that could result in a potentiation response.

The protocols did appear to stimulate acute changes in muscle architecture. Both MI and HI did appear to result in a greater increase in CSA of both the RF and VL muscles. This is consistent with others that reported significant elevations in muscle size following acute resistance exercise (Csapo et al., 2011). However, changes in PANG were not consistent between the protocols. PANG did appear to decline in 1RM compared to MI and CTL in the VL, which would be consistent with a potential for improved power output (Abe et al., 2000). However, the magnitude of change may not have been sufficient to cause performance improvements. Mahlfield, Franke, and Awiszus (2004) reported a significant decrease (~11%)

in PANG 3-6 minutes after a 3-sec isometric maximal voluntary contraction, which was associated with a greater power output. The changes in PANG reported by Mahlfield and colleagues (2004) did exceed the changes observed in this present study.

As discussed earlier, the protocols employed in this present study were based upon the recommendations from two separate, recently published meta-analyses (Guovea et al., 2012; Wilson et al., 2013). Although we have discussed several issues regarding the lack of potentiation observed that has support in literature, there are two additional factors that may have also contributed to the results. This is the first study that we are aware of that used muscle ultrasound measures to explain potential mechanisms resulting in PAP. However, the methodology employed in using the ultrasound may have potentially contributed to the lack of effect. Upon arrival each subject was instructed to lay supine for 15-min on an examination table prior to the baseline ultrasound images being taken. Following the squat intervention at 8 and 20 min post squat more ultrasounds images were again taken in the same supine position. It is likely that the movement from a standing position to a supine position for each measurement resulted in a fluid shift skewing the ultrasound data, and possibly affecting subsequent jump performance. Evidence does show that changes in body position from standing to supine can lead to changes in intra-muscular fluid levels which may influence the accuracy of muscle measures when using an ultrasound (Berg, Tedner, and Tesch, 1993). Fluid shifts have been shown to affect acute changes in skeletal muscle size (Berg et al., 1993), and these changes appear to relative to the time spent in the supine position, with the most profound decreases in size occurring within the first 15–20 min of lying down (Berg et al., 1993; Cernigliam

Delmonico, Lindle, Hurley, Rogers, 2007). This may provide some partial explanation to the minimal changes observed in muscle architecture compared to baseline values.

In conclusion, although the results of this study demonstrate little to no significant PAP response, we did observe some acute muscle architectural changes. The lack of potentiation reported could be attributed to high intra-individual variability, and the sensitivity of the PAP response to the potentiating stimulus. These findings suggest more information on which stimulus is appropriate for a given population is imperative in understanding this phenomenon. In the future perhaps a different training stimulus or different subject population would augment a greater PAP response. In addition, it is possible a greater change in muscle architecture would have been observed with the use of alternate methodology regarding the positioning of the participants during ultrasound scanning. It is clear that further investigation is warranted concerning acute muscle architecture changes and how those changes affect PAP.

APPENDIX: IRB APPROVAL LETTER



University of Central Florida Institutional Review Board Office of Research & Commercialization 12201 Research Parkway, Suite 501 Orlando, Florida 32826-3246 Telephone: 407-823-2901 or 407-882-2276

www.research.ucf.edu/compliance/irb.html

Approval of Human ResearchFrom:UCF Institutional Review Board #1 FWA00000351, IRB00001138To:Danielle ReardonDate:July 23, 2013

Dear Researcher:

On 7/23/2013 the IRB approved the following human participant research until 7/22/2014 inclusive: Type of Review: Submission Response for UCF Initial Review Submission Form

Expedited Review

Project Title: CHANGES IN MUSCLE ARCITECTURE MAY EFFECT POST-ACTIVATION POTENTIATION

Investigator: Danielle Reardon IRB Number: SBE-13-09470

Funding Agency:

Grant Title: Research ID: N/A

The scientific merit of the research was considered during the IRB review. The Continuing Review Application must be submitted 30days prior to the expiration date for studies that were previously expedited, and 60 days prior to the expiration date for research that was previously reviewed at a convened meeting. Do not make changes to the study (i.e., protocol, methodology, consent form, personnel, site, etc.) before obtaining IRB approval. A Modification Form <u>cannot</u> be used to extend the approval period of a study. All forms may be completed and submitted online at <u>https://iris.research.ucf.edu</u>.

If continuing review approval is not granted before the expiration date of 7/22/2014, approval of this research expires on that date. <u>When you have completed your research, please submit a</u> <u>Study Closure request in iRIS so that IRB records will be accurate</u>.

<u>Use of the approved, stamped consent document(s) is required.</u> The new form supersedes all previous versions, which are now invalid for further use. Only approved investigators (or other approved key study personnel) may solicit consent for research participation. Participants or their representatives must receive a copy of the consent form(s).

In the conduct of this research, you are responsible to follow the requirements of the <u>Investigator Manual</u>. On behalf of Sophia Dziegielewski, Ph.D., L.C.S.W., UCF IRB Chair, this letter is signed by:

Signature applied by Patria Davis on 07/23/2013 04:18:25 PM EDT

IRB Coordinator

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