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Do Changes in Muscle Architecture Affect Post-Activation Potentiation?

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Do Changes in Muscle Architecture Affect Post-Activation Potentiation?


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Abstract
The purpose of this randomized, cross-over design study was to examine the effect of three different muscle potentiation protocols on acute changes in muscle architecture and vertical jump performance. Eleven experienced, resistance trained men (25±3.6y) completed three potentiation squat protocols using moderate intensity (MI; 75%, 3 sets x 10 repetitions), high intensity (HI; 90%, 3 sets x 3 repetitions) and 100% (1RM; 1 set x 1 repetition) of their 1RM. In addition, all participants completed a control session (CTL) in which no protocol was performed. During each testing session, muscle architecture and vertical jump testing were assessed at baseline (BL), 8min post (8P) and 20min post (20P) workout. Ultrasound measures included cross sectional area (CSA) and pennation angle (PANG) of both the rectus femoris (RF) and vastus lateralis (VL). Following each ultrasound measure, peak vertical jump power (PVJP) and mean (MVJP) power was assessed using an accelerometer. Magnitude based inferences were used to make comparisons between trials. The MI trial resulted in a likely greater increase from BL to 8P and 20P in RF-CSA and VL-CSA, while the HI trial resulted in a likely greater change from BL to 20P in both RF-CSA and VL-CSA. Meanwhile, changes in PVJP and MVJP for the MI trial was likely decreased at BL-8P and BL–20P, while the HI trial was shown to result in a likely or possible decrease compared to CTL at BL-8P and BL–20P, respectively. A likely negative relationship was observed between changes in VL-PANG and MVJP (r = -0.35; p < 0.018) at BL-8P, and between changes in PVJP and RF-CSA (r = -0.37; p < 0.014) at BL-20P. Results of this study were unable to demonstrate any potentiation response from the trials employed, however these protocols did result in acute muscle architectural changes.

Key words: Resistance Exercise, Athletes, Sport, Squats, Performance.

Introduction

Post-activation potentiation (PAP) is a phenomenon by which the force exerted by a muscle is increased due to previous activation (Robbins and Docherty, 2005). Potentiation appears to be dependent on an appropriate training stimulus and a proper rest interval to maximize performance gain and minimize performance impairment due to fatigue (Goosen and Sale, 2000). However, there does not appear to be an accepted training stimulus or rest interval that provides a consistent potentiating effect (Wilson et al., 2013). Muscle potentiation has been induced using various types of exercise protocols. The most common method appears to be through the use of maximal voluntary contractions (Mitchell and Sale, 2011), but PAP has also been stimulated by submaximal efforts as well. Previous studies have suggested that loads of 80% or more of the participant’s one-repetition maximum (1RM) are needed to elicit a potentiation effect to facilitate short term power increases (Gouvea et al., 2013; Matthews et al., 2009; Weber et al., 2008). However, a recent meta-analysis by Wilson and colleagues (2013) have suggested that intensities between 60-84% 1RM are optimal for inducing PAP.

Rest period length is also considered to be important in stimulating muscle potentiation. If recovery from the training stimulus is not complete, the ability to potentiate subsequent power performance is removed (Tillin and Bishop, 2009). Studies have examined rest intervals from immediately post-stimulus to 20 minutes post-stimulus. Gullich and Schmidtmilecher (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 4 to 18 minutes post-exercise, improvements in power or jump height are seen (McAnn and Flanagan, 2010). Based on previous study outcomes, recommendations for a 7-10 or 8-12 minute recovery interval is recommended to enhance the potentiation response to exercise (Gouvea et al., 2013; 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). Muscle pennation angle appears 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 (Kumagai et. al, 2000). Mahfeld 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 (Seynnes et al., 2007). 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

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on vertical jump power performance.

**Methods**

**Participants**

Eleven men (25.18 ± 3.60 y; 1.77 ± 0.07 m; 90.67 ± 12.70 kg) with an average 1RM squat of 178.3 ± 36.7 kg 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 each testing session. In addition, subjects were instructed not to consume any energy or caffeine supplements prior to each testing session.

**Study protocol**

Participants reported to the Human Performance Lab (HPL) on five separate occasions separated by at least one week. During the first visit, participants were familiarized with the technique required to perform the vertical jumps (counter movement jump and vertical jump for height), and baseline ultrasound measures and images were obtained. Participants reported back to the HPL on four additional occasions separated by at least one week to complete testing trials. The squat potentiation protocols administered in this investigation were based on recommendations suggested by previous research (Hoffman et al., 2007; Wilson et al., 2013). During the second visit (1st testing session) participants performed a 1RM squat. This was considered to be the first potentiation trial. The reason for performing this trial first was to determine loads for the subsequent trials. During the next three trials, participants were randomly assigned to one of the three protocols: 1) a moderate intensity (MI) squat protocol using 75% of the participant’s 1RM (3 sets of 10 repetitions); 2) a high intensity (HI) squat protocol using 90% of the participant’s 1RM (3 sets of 3 repetitions); or 3) no workout which served as a control session (CTL). A rest interval of 3-min occurred between each set for both MI and HI trials.

Upon arrival at the HPL, participants rested in the supine position for 15 minutes to account for any fluid shifts. Baseline (BL) ultrasound measures of the vastus lateralis and rectus femoris muscles were then performed. Immediately following BL ultrasound measures, participants performed a standardized dynamic warm-up consisting of 5-minutes 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 eight minutes 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 minutes 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). This protocol was used for the 1RM trial. 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 minute 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.

**Vertical jump testing**

Vertical jump height was assessed using a Vertical Jump Testing station (Usaka 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 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. All three jumps were performed consecutively. The participant was asked to rest themselves following each jump in the starting position and to proceed when ready.

Following vertical jump height testing, participants performed 3 additional CMJ’s with their hands remaining on their hips through the entire range of motion. All three jumps were performed consecutively. Peak (PVJP) and mean (MVJP) vertical jump power was determined from a Tendo™ Power Output Unit (Tendo Sports Machines, Trenčín, Slovak Republic) that was attached at the waist of the participant during the vertical jump assessment. The Tendo™ 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. The ICC for CMJ power was 0.98 (SEM = 62.9 W).

**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. 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 posi-
tion for 15 minutes 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.

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 were 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 off-line (Thomaes et al., 2012) 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 off-line (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 0.98 (SEM = 0.52°) and 0.99 (SEM = 0.33°), 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 off-line (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 = 0.20°) and 0.81 (SEM = 1.28°), respectively.

### Statistical analysis

Data were analyzed using magnitude based inferences, calculated from 90% confidence intervals, as previously described (Batterham and Hopkins, 2006; Cohen, 1988). Change scores were analyzed using the p value from dependent t-test to determine a mechanistic inference utilizing a published spreadsheet (Hopkins, 2007). Qualitative inferences were based upon the chances that the true magnitude of the effect at POST-off-season were substantially greater or smaller than baseline values (PRE), and were assessed as: <1% almost certainly smaller, 1-5% very likely smaller, 5-25% likely smaller, 25-75% possibly greater, 75-95% likely greater, 95-99% very likely greater and >99% almost certainly greater (Hopkins, 2002). If there was a greater than 5% chance that the true value was both greater and smaller, the effect was considered mechanistically unclear. The smallest non-trivial change, or smallest worthwhile change, was set at 20% of the grand standard deviation for all PRE-values (Batterham and Hopkins, 2006).

The relationship between changes in muscle architecture and changes in jump height and power were examined using Pearson product-moment correlation coefficients. Correlation coefficients were further analyzed using a published spreadsheet to determine the magnitude of effect (Hopkins, 2007). 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 and Hopkins, 2006) and were based on the confidence interval range relative to the smallest clinically meaningful effect to be positive, trivial, or negative. 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).

### Results

Comparisons of vertical jump height and jump power are depicted in Table 1. No differences were noted from BL during any of the potentiation protocols or CTL trial. Inferential analysis of the change in jump height between BL and 8P, and between BL and 20P for all trials can be observed in Table 2. At 8P the change in jump height was not statistically significant.

| Table 1. Vertical jump performance comparisons between all trials. Data are means (±SD). |
|---------------------------------|----------|----------|----------|----------|
|                                | CTL      | MI       | HI       | IRM      |
| VJ Height (cm)                 |          |          |          |          |
| BL                             | 66.7 (12.1) | 66.2 (11.2) | 65.6 (10.6) | 66.1 (12.5) |
| 8P                             | 66.3 (12.3) | 62.4 (10.3) | 63.6 (11.0) | 64.6 (12.1) |
| 20P                            | 65.4 (12.0) | 62.7 (10.3) | 62.8 (10.8) | 64.6 (12.5) |
| PVJP (W)                       |          |          |          |          |
| BL                             | 2579 (890) | 2506 (689) | 2423 (599) | 2900 (916) |
| 8P                             | 2584 (991) | 2342 (631) | 2439 (654) | 2792 (916) |
| 20P                            | 2565 (1190) | 2449 (629) | 2470 (602) | 2831 (913) |
| MVJP (W)                       |          |          |          |          |
| BL                             | 1225 (282) | 1323 (359) | 1500 (730) | 1280 (306) |
| 8P                             | 1253 (308) | 1220 (257) | 1307 (330) | 1251 (309) |
| 20P                            | 1223 (289) | 1256 (284) | 1347 (360) | 1247 (316) |

VJ = Vertical Jump; PVJP = Peak Vertical Jump Power; MVJP = Mean Vertical Jump Power; BL = baseline; 8P = 8 minutes post-exercise; 20P = 20 minutes post-exercise
likely decreased in MI compared to HI and 1RM, and very likely decreased compared to CTL. Changes in vertical jump height at HI and 1RM were very likely and likely decreased compared to CTL. At 20P changes in jump height were very likely decreased in MI and HI compared to CTL. In addition, the change in jump height was likely decreased at MI and HI compared to 1RM.

Inferential analysis of the changes in both PVJP and MVJP between trials can be observed in Table 3. At 8P changes in PVJP were likely decreased in MI compared to HI, 1RM and CTL, and possibly decreased for 1RM compared to CTL. At 20P changes in PVJP were likely decreased in MI compared to 1RM and CTL. All other PVJP comparisons were unclear or trivial. Changes in MVJP were likely decreased at 8P in both MI and HI compared to CTL. Changes in 1RM were possibly less than that seen at HI. At 20P changes in MVJP 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.

### Table 2. Magnitude based inferences on between trial comparisons on changes in vertical jump height (cm). Data are means (±SD).

<table>
<thead>
<tr>
<th>Group Comparison</th>
<th>Group 1</th>
<th>Group 2</th>
<th>p-Value</th>
<th>Threshold</th>
<th>Percent</th>
<th>Mean Difference</th>
<th>Inference</th>
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<tr>
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<td>MI vs. CTL</td>
<td>-1.5 (1.5)</td>
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BL = baseline; 8P = 8 minutes post-exercise; 20P = 20 minutes post-exercise

### Table 3. Magnitude based inferences on between trial comparisons on changes in vertical jump power (W). Data are means (±SD).

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<td>MI vs. CTL</td>
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</table>

PVJP = Peak Vertical Jump Power; MVJP = Mean Vertical Jump Power; BL = baseline; 8P = 8 minutes post-exercise; 20P = 20 minutes post-exercise.
Comparisons of muscle architecture changes are depicted in Table 4. No changes were noted from BL during any of the potentiation protocols or CTL trial. Inferential analysis of changes in muscle architecture between trials can be seen in Table 5. At 8P changes in the CSA of the RF was likely greater for MI compared to CTL. At 20P changes in the CSA of the RF were likely greater for MI and HI compared to CTL. All other CSA of the RF comparisons between groups were unclear. Analysis of the changes in the CSA of the VL at 8P was likely and possibly greater in MI and HI, respectively compared to CTL. At 20P changes in the CSA of the VL was 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 were unclear.

Changes in the PANG of the RF at 8P between all trials were unclear, while the change in PANG of the VL was likely greater for MI compared to 1RM. Changes in the PANG during 1RM were very likely decreased compared to that seen during CTL. At 20P the change in the PANG of the RF was likely greater in MI compared to HI, while the change in HI was possibly less than that seen during 1RM. Changes in the PANG at 20P in the VL were likely greater in MI and HI compared to 1RM, while changes in PANG at this time point were likely decreased for 1RM compared to CTL. All other comparisons between groups were unclear.

Magnitude based inferences on Pearson correlation analyses are shown in Table 6. 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 = -0.37) and a possible negative relationship (r = -0.23) 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 (r = -0.35) was observed between changes in PANG between baseline and 8-min post-exercise and mean vertical jump power. No other meaningful correlations were observed.

**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 post-exercise 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 potentiation was not seen during any of the protocols, results did indicate a likely negative relationship between changes in CSA and PANG and changes in vertical jump performance. This suggests that increases in muscle CSA 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., 2013; 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., 2013; 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 the greatest effects on changes in CSA and PANG in both the RF and VL muscles. Although potentiation was not seen during any of the protocols, results did indicate a likely negative relationship between changes in CSA and PANG and changes in vertical jump performance. This suggests that increases in muscle CSA 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.
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 re-
cover from a potentiating exercise protocol, likely related to the greater buffering capacity and resistance to muscle damage seen in the competitive athlete (McHugh et al., 1999; Wilson et al., 2013). In addition, the competitive athlete may benefit from potentiation due to 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., 2013; Mahlfield and colleagues, 2004) reported a significant decrease 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 the other groups 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 and colleagues, (2004) reported a significant decrease (~11%) in PANG 3-6 minutes after a 3-sec isometric maximal voluntary contraction, which was associated

### Table 6. Magnitude Based Inferences on Pearson Correlation Measures between Changes in Jump Performance and Muscle Architecture

<table>
<thead>
<tr>
<th>Group Comparison</th>
<th>r</th>
<th>p-Value</th>
<th>Percent Difference</th>
<th>Inference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vertical Jump Height: BL – 8P</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RFCSA</td>
<td>-.06</td>
<td>.721</td>
<td>4.9</td>
<td>77.8</td>
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<tr>
<td>RFPANG</td>
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<td>.277</td>
<td>.9</td>
<td>57.6</td>
</tr>
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<td>.1</td>
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<tr>
<td>VLPANG</td>
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<td>.118</td>
<td>.2</td>
<td>39.4</td>
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<tr>
<td><strong>Vertical Jump Height: BL – 20P</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RFCSA</td>
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<td>.221</td>
<td>.6</td>
<td>52.6</td>
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<tr>
<td>RFPANG</td>
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<tr>
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<td>.204</td>
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<td>50.8</td>
</tr>
<tr>
<td>VLPANG</td>
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<td>.875</td>
<td>12.6</td>
<td>80</td>
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<tr>
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<td>.821</td>
<td>6.4</td>
<td>79.5</td>
</tr>
</tbody>
</table>

RFCSA = rectus femoris cross-sectional area; RFPANG = rectus femoris pennation angle; VLCSA = vastus lateralis cross-sectional area; VLPANG = vastus lateralis pennation angle; PVJP = Peak vertical jump power; MVJP = Mean vertical jump power; BL = baseline; 8P = 8 minutes post-exercise; 20P = 20 minutes post-exercise.
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., 2013; 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 ultrasound 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 et al., 1993). Fluid shifts have been shown to affect acute changes in skeletal muscle size (Berg et al., 1993), and these changes appear 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; Cerniglia et al., 2007). This may provide some partial explanation to the minimal changes observed in muscle architecture compared to baseline values.

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. In addition, it is possible that 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.

References


Key points

- Three squat protocols using moderate intensity (75% 1-RM; 3 sets x 10 repetitions), high intensity (90% 1-RM, 3 sets x 3 repetitions) and maximal intensity (100% 1RM; 1 set x 1repetition) were unable to potentiate jump height or jump power in experienced, resistance trained men.
- Experienced, resistance trained athletes who are not competitive may be limited in regards to potentiation due to a poor level of conditioning.
- Both the moderate and high intensity potentiation protocols stimulated acute changes in muscle architecture. Greater increases in the CSA of both the RF and VL muscles were noted.
- A different potentiation protocol may have elicited greater changes in muscle architecture.

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Does muscle architecture affect potentiation?

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