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Static Stretching and Preconditioning Exercise Augments Power Output in Recreational Athletes

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STATIC STRETCHING AND PRECONDITIONING EXERCISE AUGMENTS POWER OUTPUT IN RECREATIONAL ATHLETES

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in the College of Education at the University of Kentucky

By

Mark Ryan Mason

Lexington, Kentucky

Advisor: Dr. Mark Abel, Associate Professor of Kinesiology

Lexington, Kentucky

2016

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ABSTRACT OF THESIS

STATIC STRETCHING AND PRECONDITIONING EXERCISE AUGMENTS POWER OUTPUT IN RECREATIONAL ATHLETES

The purpose of this study was to determine the independent and combined effects of performing a preconditioning exercise and antagonist stretching on vertical jump (VJ) performance. Twenty club rugby players performed a VJ in four conditions: control condition, following static stretching, following a preconditioning exercise, and following the combined treatment of static stretching and a preconditioning exercise. Electromyographic (EMG) activity was measured in the gluteus maximus, vastus lateralis, gastrocnemius medialis, and tibialis anterior during the VJ trials. Repeated measures ANOVA were used to compare VJ and EMG outcomes across conditions. A Bonferroni correction was used to account for multiple post-hoc comparisons (significance set at p < 0.0083). Despite a strong trend, there were no independent effects of performing the preconditioning exercise (p = 0.012; Effect size = 0.29) and static stretching (p = 0.050; Effect size = 0.19) on VJ height compared to the control condition. However, the combined treatment increased VJ height 1.59 ± 1.42 cm compared to the control condition (p < 0.001, Effect size = 0.57). There were no significant differences in EMG outcomes between conditions. These findings indicate that performing a preconditioning exercise and statically stretching the antagonist muscles acutely augments lower body power output.

KEYWORDS: Acute Performance, Potentiation, Vertical Jump, Electromyography, Antagonist Stretching

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CHAPTER I

INTRODUCTION

Maximal power development is critical for success in many sports. Power is an important component in numerous biomotor abilities including sprinting, jumping, and throwing. Acutely enhancing power output is advantageous for these skills. There are numerous factors that affect power development including muscle mechanics (force-velocity & length-tension relationships), morphological factors (muscle fiber type & architecture, tendon properties), and neural factors (motor unit recruitment, firing frequency, etc.) (13). Despite the complex interaction of these factors, there is literature that indicates that various neuromuscular exercises may be performed prior to competition or assessment to acutely augment subsequent power development (8, 58).

Post-activation potentiation (PAP) of the agonist muscle and static stretching of the antagonist muscle have independently demonstrated improved power development (7, 8, 23, 28, 51, 58, 63, 67). PAP refers to an increase in the rate of force production resulting from a previous conditioning contraction. PAP has been proposed to occur via phosphorylation of myosin regulatory light chains, increased recruitment of higher order motor units, and a decreased angle of pennation during high intensity muscular contraction (60).

Torque output of a joint is proportional to the activity of agonist and inversely proportional to the activity of the antagonist muscle (4). It may be speculated that a change to the electrical activity to either of these muscle groups could have an effect on the resulting power output. It is common knowledge that an increase in agonist activity produces an increase in force output, but perhaps, given the relationship, a decrease in
antagonist activity could also increase joint torque. It has been shown that static stretching of the antagonist muscle may enhance agonist muscle power production by a reduction in musculotendinous stiffness of the antagonist, reduced motor neuron excitability of the antagonist, and/or autogenic inhibition of the antagonist (54, 58).

Power development is critical for many athletes. It is important to identify practical strategies to enhance power development. The purpose of this study is to investigate the independent and combined effects of static stretching and performing a preconditioning exercise on vertical jump (VJ) performance in recreationally trained male athletes. We hypothesized that statically stretching antagonist muscles (e.g., hip flexors and ankle dorsiflexors) and potentiating agonist muscles (plantar flexors, knee & hip extensors) would increase VJ height compared to a baseline condition.

The following research question was presented for this study:
Can vertical jump performance be augmented by the enhancement of muscular power via preconditioning exercises and static stretching of the antagonist?

This study was delimited to the following factors.

1. The sample was composed of college-aged recreationally trained rugby players.
2. The outcome measures included: VJ height, peak power output, and electromyography.
3. The instruments utilized were: Vertec vertical jump testing apparatus and surface electromyography electrodes.

The following was an assumption of the present study.

1. Each subject provided a maximal effort on every attempt.
Chapter II will review the literature regarding power, static stretching, antagonist stretching, postactivation potentiation, electromyography, and vertical jump performance. This chapter will also review content to provide an understanding of the possible mechanisms that may contribute to the findings of the present study.

Power

Power is work performed per unit of time. Work is the force of an object times the displacement said object is moved in the direction of the force (Work = Force x Displacement). Power is the amount of work performed per unit of time (Power = \( \frac{\text{Force} \times \text{Displacement}}{\text{Time}} \)). To reach a maximum power the ideal balance between work and time must be reached (40, 52). In the sport setting, movements that are thought to be most powerful are maneuvers such as sprinting, jumping, throwing, and kicking.

Force-Velocity Relationship

The force-velocity relationship of muscle is an inherent characteristic that determines the amount of power that can be produced. For a concentric contraction maximum force (F\(_{\text{max}}\)) is produced using heavy loads, whereas maximum velocity (V\(_{\text{max}}\)) is obtained with light loads. Maximum power is generated at an optimal combination of submaximal force and velocity values (42). The optimal combination has been found to be at approximately 30% of F\(_{\text{max}}\) in single muscle fibers and single joint movements (9, 14, 22, 37, 61, 62, 64). The force-velocity relationship reflects different characteristics
with regard to eccentric versus concentric contractions. During an eccentric contraction the absolute tension generated is very high in comparison to the maximal tetanic tension on the muscle and the absolute tension is relatively independent of the lengthening velocity, meaning that there is a plateau in force despite an increase in velocity (43). Whereas, in a concentric contraction force decreases with an increase in velocity (43). Figure 1 is adapted from Lieber 1993.
Figure 1. Force-velocity relationship for skeletal muscle.

Adapted from Lieber, 1993


**Length-Tension Relationship**

The ability of skeletal muscle to produce maximal power is dependent on sarcomere length (18, 26, 44). The optimal length of a sarcomere occurs when actin and myosin overlap is ideal and thus produces maximal active isometric tension (18, 26, 44). When sarcomere lengths are shorter there is a mechanical interruption from actin filaments on opposite sides that blocks myosin binding sites and decreases crossbridge interaction, thus reducing subsequent force production (42). The force production capability of a sarcomere is decreased at longer sarcomere lengths due a reduction in the overlap of contractile proteins (18, 26, 44).

**Motor Unit Recruitment**

The amount of force that can be produced by a muscle is correlated to the number and type of motor units that are activated. The size principle dictates the order in which muscle units are recruited (34, 35). In general, it is accepted that smaller alpha-motor neurons, which innervate smaller motor units, are recruited first due to a lower threshold of activation (34). These smaller motor units are capable of generating smaller amounts of force. It is thought that the size principle can somewhat adapt to the demands of the muscle. The threshold of activation has been observed to be lower for higher order motor units when the muscle is subjected to a ballistic contraction (16, 17). This preferential recruitment for higher order motor units has positive implications for powerful movements such as VJ.
**Firing Frequency**

Motor unit firing frequency (i.e., rate coding) is characterized by the rate at which neural impulses are conducted from the α-motorneuron to the individual muscle fibers (13). Firing frequency has a two-fold effect on force production. The first is an increase in the magnitude of force produced (19). The second way firing frequency can influence the force generated in a motor unit is by increasing the rate of force production. During a ballistic movement the initial firing frequency is high contributing to extreme amounts of force generated rapidly (70).

**Motor Unit Synchronization**

When the threshold to activate multiple motor units is reached in unison more often than expected for independent random processes, motor unit synchronization transpires (50). It has been hypothesized that synchronization of mechanically unstable joints could allow for better transmission of muscular force through the joint for complex movements (48). This would allow for a more direct and efficient conduction of power because less power is lost during the transmission of force through a stable joint when compared to a joint that is not stable.

**Inter-Muscular Coordination**

Inter-muscular coordination refers to the proficiency at which agonist, synergist, and antagonist muscles work together. Agonists and synergists act in cooperation to produce joint actions. At the same time antagonist contraction needs to decrease to allow for the movement to occur. For manifestation of the greatest force possible, these events
need to occur efficiently (57). The amount of co-activation of the antagonist is dependent on several factors including, type of contraction, load, velocity and precision of the movement, and range of motion (27, 38, 53). Co-activation of the antagonist diminishes the force produced by the agonist because the torque generated by the antagonist opposes the action of the prime mover (1, 4, 39). It has been hypothesized that activation of the antagonist could further reduce full activation of the agonist through reciprocal inhibition (49).

**Static Stretching**

It was once believed that static stretching prior to competition could increase range of motion, decrease risk of injury, and improve performance; however, recent research has opposed this supposition (3, 10, 54, 55, 68). It has been reported that static stretching of the prime movers decreases VJ performance (10, 54, 68). The mechanisms responsible for the attenuated performance may include: reduced musculotendinous stiffness, which may impair force production due to an altered length-tension relationship, (10, 54) reduced motor neuron excitability illustrated by a depressed H-reflex, (21, 54) and through autogenic inhibition of the stretched muscle (6, 36, 55).

**Antagonist Stretching**

Recently, one investigation examined the effect of static stretching of antagonist musculature (i.e., tibialis anterior, hipflexors) on VJ performance (58). Sandberg et al. hypothesized that stretching the antagonist muscles may enhance jump performance by inhibiting force produced by the antagonist during co-activation via the mechanisms
previously discussed. Thus statically stretching the antagonist may skew force production in favor of the agonist resulting in superior jump performance. Sandberg et al. found that stretching of the dorsiflexors, knee flexors, and hip flexors produced significantly greater torque production in the knee extensors at $300^{\circ}\cdot s^{-1}$ and significantly increased VJ height and power production (58).

**Postactivation Potentiation**

Postactivation potentiation (PAP) is another neuromuscular mechanism that has been shown to increase power performance (7, 8, 23, 28, 51, 63, 67). PAP may be elicited by performing a preconditioning contraction (e.g., maximal isometric or concentric contraction) and is typically followed by an optimal duration for which PAP exists (28, 67). There are a few proposed physiological mechanisms that may be responsible for this phenomenon. The first of which is phosphorylation of myosin regulatory light chains. In this process the regulatory light chain phosphorylation is catalyzed by myosin light chain kinase. This is activated by the release of Ca$^{2+}$ from the sarcoplasmic reticulum, which will bind to calmodulin downstream (60). It is speculated that this phosphorylation potentiates future contractions by modifying the arrangement of the myosin head and pulling it away from the thick filament backbone (60). The result is an amplified level of myosin cross bridge activity in response to submaximal concentrations of myoplasmic Ca$^{2+}$(56).

PAP is also thought to arise from an increased recruitment of higher order motor units. This is primarily due to a decrease in neurotransmitter failure as a result of several possible responses. These include an increase in the efficacy of the neurotransmitter,
increased quality of neurotransmitter, or a reduction of axonal branch-point failure along afferent neural fibers (20). This could potentially increase the contribution of fast twitch fibers to a contraction, which would enhance power output (29).

Another theory supporting this occurrence is that the angle of pennation of a muscle fiber may decrease during a high intensity muscular contraction. This allows greater transmission of force delivered from the altered muscle (46). It is important to note that the authors’ who proposed this idea agree that there is not much evidence to support this actually contributing to force generation and that the possibility of contribution warrant further research (60).

The effects of PAP on subsequent activity can be altered by several different factors. These include; PAP versus accumulated fatigue, conditioning contraction volume, conditioning contraction type, subject characteristics (muscular strength, fiber-type distribution, training level, and power-strength ratio), and the type of subsequent activity (60). The balance of PAP and fatigue has a major role in the performance of subsequent activity. Immediately following a conditioning contraction, there is a small window where PAP is more of a contributing factor than fatigue, yielding increased performance. Soon afterwards the amount of fatigue present interferes with any performance enhancement of PAP in voluntary movements. The negative effects of fatigue subside more rapidly than the positive effects of PAP, thus there is a second window where performance is enhanced (25, 60).

Conditioning contraction volume plays a role because of this balance of PAP and fatigue and further supports the idea of two potential windows for enhancement of performance (32, 65). Differences in conditioning contraction types have produced mixed
results. It is thought that isometric conditioning contractions cause more central fatigue, but are more likely to activate the peripheral mechanisms of PAP (60). In contrast, dynamic conditioning contractions are thought to cause more peripheral fatigue, but activate central mechanisms of PAP (60). Since the interaction between these conditioning contractions is different, it is speculated that the effects on subsequent powerful movements could be different.

Several subject characteristics have been shown to be a contributing factor to power manipulation. These characteristics change the influence of the PAP-fatigue response. Individuals who are most likely to gain the most enhancements from PAP include; those with more muscular strength, more percentage of type II muscle fibers, higher levels of resistance training, and a smaller power-strength-ratio (60). The subsequent activity is most likely to benefit more from a conditioning contraction that closely mimics the kinematics of that activity. If the conditioning contraction is similar to the explosive activity a subject is performing, the individual is more likely to activate higher order or larger motor units, phosphorylate the myosin regulatory light chain, and change the architecture of the muscles exclusively related to the subsequent activity (60).

**Electromyography**

Electromyography (EMG) can be very useful in sport science. It provides important information into the physiological processes that lead to force generation (15). The EMG signal represents the electric current generated during an action potential during a contraction. There are two main types of EMG used for research purposes. Surface EMG is non-invasive and measures muscle activity via electrodes placed on the
skin. Indwelling EMG is invasive and involves inserting fine wire electrodes directly into the muscle being studied. In general indwelling EMG is used with smaller muscle or when there are multiple muscles in close vicinity and the research is interested in individual muscle activity. This is because of the reduction in crosstalk from other nearby muscles. Surface electrodes are often used due to the ease of application and minimal discomfort to the participants. Surface electrodes are effective for examining gross motor movements. Surface electrodes are placed near the belly or center of the muscle parallel to the muscle fibers (59). This orientation provided the optimal signal transduction to the recording device. Surface EMG is not without drawback. Crosstalk is present in the signal obtained from surface EMG and there are multiple areas where noise can arise. Thoroughly cleaning the skin of debris and ensuring proper adherence of the electrode to the skin can reduce noise while proper placement and sizing of the electrodes can help to reduce crosstalk.

In biomechanics, EMG is particularly important for determining three main objectives. It can be used to signify muscle activation, the relationship with force produced by a muscle, and as an index to assess fatigue as it takes place within a muscle (15). When examining the relationship with force produced by a muscle, there is not a direct measurement or calculation that can tell you how much force is generated based off the EMG activity of any given muscle. The idea that force generally increases with EMG activity only provides a qualitative indication of the relationship between the variables (15). This qualitative relationship can be useful in assessing whether one task generates more force than another for the same individual, but is limited to this extent. It is not
possible to quantitatively analyze precisely how much more or less force is generated between two tasks (15).

**Vertical Jump**

VJ is an essential element to many sports. It can be used as a tool to measure prospective athletic ability, and it is one of the earliest known measurable examples of human power (11). While VJ is an integral asset to sport performance and the ability to increase VJ height can be advantageous, (30, 31) the scope of this paper is pertaining to acute enhancements that would not be advantageous to most conventional sports of a long duration. Acute enhancements of power or VJ height could still be beneficial in single bout sports or athletic assessments. A review of the 2000 NFL Combine revealed that the most significant test to assess draft status and starting salary of the running back position was the VJ test (47). A similar study of the results of the NFL Combine over a six-year period (1999-2004) discovered that the metric displaying the strongest relationship with draft order at the quarterback position was VJ (41).
CHAPTER III

This chapter will explore the methodology necessary to answer the research question of the current study. Chapter III provides a detailed description of the study design, sample, procedures, and statistical analysis used to answer the research question.

METHODOLOGY

Experimental Approach to the Problem

A within-subjects design was used to determine the effects of performing static stretching and a preconditioning exercise on power output. The dependent variables in this study were VJ height and electromyographic activity in the gluteus maximus, vastus lateralis, gastrocnemius medialis, and tibialis anterior during a VJ. The stretching and exercise treatments served as the independent variables. Participants performed a VJ five minutes after the following treatments: control condition (no treatment), stretching treatment, preconditioning exercise treatment, and a combined treatment of both stretching and exercise. The order of the treatments was randomized. The study consisted of three testing sessions for each participant. Session 1 served to familiarize subjects with the testing procedures. Two of the four conditions were performed in Session 2 and the remaining conditions were performed at least two days later in Session 3. The time between each treatment and performing the VJ was standardized across subjects (Figure 2). In addition, during the absence of stretching treatment, preconditioning exercise or both a recovery period was administered to control for the effect of time on potentiation. This was performed to ensure there were no differences in potentiation between trials due to time differences. Figure 2 is a flow chart with the summary of procedures.
TA: Tibialis anterior, FID: Functional isometric deadlift.

Figure 2. Summary of procedures.
Subjects

A convenience sample of 20 healthy, recreationally trained male rugby players was recruited to participate in this study. All participants were members of a Division I collegiate rugby team or a Division III men’s rugby team. The participants’ mean age was 23.9 ± 3.5 years, height = 180.1 ± 5.3 cm, and body mass = 90.1 ± 11.7 kg. All participants completed a PAR-Q form to ensure they were free from musculoskeletal, cardiovascular, and metabolic disorders. The study procedures were approved by the University’s Institutional Review Board. Each subject provided written informed consent prior to participation in the study.

Procedures

Stretching Treatments

The stretching treatments were concluded six minutes and thirty seconds before the VJ test to control for time differences between condition procedures. The stretching treatment emphasized the static stretching of the hip flexors and ankle dorsiflexors. The stretching treatment consisted of a static lunge stretch. This stretch targets the muscles required for hip flexion, which serves as the antagonistic to the main joint action during the hip extension motion on the upward movement phase of a VJ. This particular hip flexor stretch was chosen because it can be performed by the individual without assistance and it isolates the hip flexors. This stretch specifically targets the iliopsoas group (iliacus, psoas major, and psoas minor). Three repetitions were performed and each stretch was held for 30 seconds (3). Participants were instructed to kneel onto one knee on a padded surface in the “90°/90°” lunge position (Figure 3; the trailing hip is extended
to 90° and the trailing knee is flexed to approximately 90°, which is in contact with the ground and supporting the majority of the bodyweight, while the lead leg is flexed at both the hip and knee approximately 90°, and the foot of which is flat on the floor in front of the body). The subject was instructed to internally rotate his leg to emphasize the stretch on the hip flexors and decrease the stretch of the rectus femoris, which contributes to knee extension (58). Each subject was instructed to assume an appropriate upright posture, place the stretch side hand on the hip to be stretched, the other on a nearby support for balance, and drive that hip forward until the point of mild discomfort. This procedure was performed on each leg alternating the right and left hip. This stretch is demonstrated in Figure 3 below.
Figure 3: Psoas stretch prior to vertical jump.
To stretch the ankle dorsiflexors, participants were positioned supine on a plinth. The feet were extended over the table, unrestricted in space. The investigator passively stretched the ankle dorsiflexors by manually placing the ankle in plantar flexion. The ankle dorsiflexors were stretched to a point of mild discomfort. The dorsiflexor stretch is illustrated in Figure 4.
Figure 4: Tibialis anterior stretch prior to vertical jump.
Maximum Voluntary Isometric Contraction

A MVIC was conducted on the deadlift exercise (i.e., preconditioning exercise) five minutes prior to the VJ test for the treatment trials to induce PAP in all leg extensor muscle groups (hip extensors, knee extensors, ankle plantarflexors). Three repetitions of MVIC’s were performed with 20 seconds of rest between repetitions. Each repetition was held for 5 seconds. The subject performed the functional isometric deadlift exercise by standing in an athletic stance on a strap and holding the attached handles, simulating a deadlift. A goniometer was used to ensure the subject’s knees were bent to 45°. The subject then pressed as hard as possible with his legs from the deadlift position while keeping the strap firmly in his hands preventing vertical movement. Figure 5 below depicts the functional isometric deadlift use for this study.
Figure 5: Functional isometric deadlift used as a preconditioning exercise.
*Vertical Jump Test*

The VJ test was performed using a Vertec device (Sports Imports, Columbus, OH, USA) using a protocol described by Bean (5). The VJ test has been found to be highly reliable (ICC=0.94) (12, 69). The investigator explained and demonstrated the proper position for obtaining the standing reach measure. Specifically, the participant stood under the apparatus with the feet together and reaching as high as possible with the dominant arm while keeping the feet flat on the floor. The palm of the hand was placed against the Vertec device. The investigator recorded the standing reach height as the highest vane touched by the top of the longest finger. The investigator also explained and demonstrated the VJ and reach. Specifically, the participant began by moving the feet to a comfortable jumping position. One countermovement (dip) of the legs plus one swing of the arms was made immediately prior to the push-off phase of the jump. While in the air the subject touched the highest vane possible. The subject performed a minimum of three VJ attempts for each condition with 20-30s of recovery between attempts. The investigator recorded all VJ attempts to the closest 1.27cm (device sensitivity). If the subject continued to improve on the third trial, subsequent trials were provided until no further improvements were observed on two consecutive trials. The Harman peak power equation \( W = 61.9 \text{ (cm) Jump Height} + 36.0 \text{ Body Mass (kg)} + 1,822 \) \( (r=0.88) \) was used to calculate peak power using vertical jump displacement (cm), subject’s body mass (kg), and constants developed by Harman and colleagues (33).
Electromyography

Peak surface electromyography (EMG) signals were obtained using SENIAM guidelines for preparation and placement from the gluteus maximus (GM), vastus lateralis (VL), medial gastrocnemius (MG), and tibialis anterior (TA) during VJ. The electrodes were 99.9% Ag-CI bars, which are 10 x 1 mm in diameter with an interelectrode distance of 10 mm. EMG detection was performed through differential means, input impedance of \( > 10^{15} \Omega // 0.2 \mu \text{F} \), Common Mode Rejection Ratio (CMRR) \( \geq 100 \text{ dB} \), a Signal to Noise Ratio (SNR) \( \leq 1.2 \mu \text{V} \), and the Gain was set at 1000 Hz. Some participants had the gain set at 10000Hz for the (GM) to increase the signal due to adipose tissue in this area. Sampling of the EMG was completed at 1550 Hz through a 16-bit A-D board. Data were collected on a computer using Cortex software (Motion Analysis, Santa Rosa, CA) and processed using Visual3D software (C-Motion Inc., Germantown, MD). Filtering was performed using a Butterworth band pass filter with a bandwidth of 20-500Hz. The peak EMG signal was calculated by using a moving RMS function (30 ms) to rectify and smooth the EMG signal.

Statistical Analyses

Basic statistics were used to describe the data (mean ± standard deviation). Relative difference scores were used to compare treatment conditions versus the control condition and were calculated as follows: Relative difference = \( \frac{\text{Treatment condition} - \text{Control condition}}{\text{Control condition}} \times 100 \). The absolute difference between treatment versus control conditions was calculated as: Absolute difference = Treatment condition – Control condition. Separate repeated measures analysis of variance
(ANVOA) were used to identify significant differences in VJ height and EMG activity between the four conditions (control condition, stretching treatment, preconditioning exercise treatment, combined treatment). Paired samples t-tests were used in the post-hoc analysis to determine which conditions differed. Due to the use of multiple comparisons, a Bonferroni correction was used to control for the inflation of Type I error. Thus, the level of significance was set at $p < 0.0083$ ($0.05 / 6$ comparisons). Post-hoc effect size estimates were reported with partial eta squared. All analyses were conducted using the Statistical Package for Social Sciences (SPSS 18.0; IBM, Somers, NY, USA).
CHAPTER IV

RESULTS & DISCUSSION

This chapter will review the results of the current study as well as provide discussion of the findings. Chapter IV will compare the findings of the current study to the results of previous works in similar experiments. This chapter will also discuss possible mechanisms that may be responsible for the findings and explain how these mechanisms may be responsible for supporting or refuting the findings of relevant investigations.

**Vertical Jump Height**

Table 1 displays the mean vertical jump displacement for each condition. There was a significant within-subjects effect for vertical jump height ($F(3,17) = 9.125, p = .001$; Effect size = 0.617; Power = 0.98) indicating that only the combined treatment produced a greater vertical jump height compared to the control condition. Despite strong trends, there were no other significant differences between the independent treatments and the control condition ($p = 0.012$ to 0.050; Table 2).
Table 1. Comparison of vertical jump height between experimental and control conditions in 20 male subjects.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>VT Displace (cm)</th>
<th>Relative Diff. (%)</th>
<th>Absolute Diff. (cm)</th>
<th>ES</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>51.13 ± 11.56</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Stretch</td>
<td>51.96 ± 10.79</td>
<td>1.61</td>
<td>0.83</td>
<td>0.188</td>
<td>0.512</td>
</tr>
<tr>
<td>Precon. Ex.</td>
<td>52.53 ± 10.92</td>
<td>2.73</td>
<td>1.40</td>
<td>0.288</td>
<td>0.749</td>
</tr>
<tr>
<td>Combination*</td>
<td>52.72 ± 10.63</td>
<td>3.10</td>
<td>1.59</td>
<td>0.568</td>
<td>0.997</td>
</tr>
</tbody>
</table>

*Significant difference from control (p < 0.001). Precon. Ex.: Preconditioning exercise; ES: Effect size.
Table 2. Post-hoc comparison of mean VJ displacement (cm) between conditions.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean ± SD</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control vs. Stretch</td>
<td>-0.83 ± 1.76</td>
<td>0.050</td>
</tr>
<tr>
<td>Control vs. Precon. Ex.</td>
<td>-1.40 ± 2.25</td>
<td>0.012</td>
</tr>
<tr>
<td>Control vs. Combination</td>
<td>-1.59 ± 1.42</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stretch vs. Precon. Ex.</td>
<td>-0.57 ± 2.08</td>
<td>0.234</td>
</tr>
<tr>
<td>Stretch vs. Combination</td>
<td>-0.76 ± 1.19</td>
<td>0.010</td>
</tr>
<tr>
<td>Precon. Ex. vs. Combination</td>
<td>-0.19 ± 1.81</td>
<td>0.643</td>
</tr>
</tbody>
</table>

*Level of significance set to p < 0.0083. Precon. Ex.: Preconditioning exercise.
Table 3 displays the peak power output for each condition. There was a significant within subjects effect for peak power output ($F(3,17) = 9.125, p = 0.001$; Effect size = 0.617; Power = 0.983) indicating that the Combined treatment produced greater power than the Control condition ($p < 0.001$). Despite strong trends, there were no other significant differences between the treatment and control conditions ($p = 0.012$ to 0.05; Table 4).
Table 3. Comparison of peak power output between experimental and control conditions in 20 male subjects.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Power Output (W)</th>
<th>Relative Diff (%)</th>
<th>Absolute Diff (W)</th>
<th>ES</th>
<th>Power</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>8232.4 ± 634.1</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Stretch</td>
<td>8283.5 ± 617.7</td>
<td>0.62</td>
<td>51.10</td>
<td>0.188</td>
<td>0.512</td>
</tr>
<tr>
<td>Precon Ex.</td>
<td>8318.9 ± 608.0</td>
<td>1.05</td>
<td>86.47</td>
<td>0.288</td>
<td>0.749</td>
</tr>
<tr>
<td>Combination*</td>
<td>8330.7 ± 600.7</td>
<td>1.19</td>
<td>98.27</td>
<td>0.568</td>
<td>0.997</td>
</tr>
</tbody>
</table>

*Significant difference from control (p < 0.001). Precon Ex.: Preconditioning exercise; ES: Effect size.
Table 4 displays the post-hoc comparison of peak power output between conditions. After the Bonferroni correction the only significant difference was a 98.27W increase in peak power output in the combined treatment compared to the control.
Table 4. Post-hoc comparison of peak power output (W) between conditions.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean</th>
<th>SD</th>
<th>p-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control vs. Stretch</td>
<td>-51.10</td>
<td>±109.03</td>
<td>0.050</td>
</tr>
<tr>
<td>Control vs. Precon. Ex.</td>
<td>-86.47</td>
<td>±139.47</td>
<td>0.012</td>
</tr>
<tr>
<td>Control vs. Combination</td>
<td>-98.27</td>
<td>±87.89</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stretch vs. Precon. Ex.</td>
<td>-35.38</td>
<td>±128.73</td>
<td>0.234</td>
</tr>
<tr>
<td>Stretch vs. Combination</td>
<td>-47.17</td>
<td>±73.92</td>
<td>0.010</td>
</tr>
<tr>
<td>Precon. Ex. vs. Combination</td>
<td>-11.79</td>
<td>±111.98</td>
<td>0.643</td>
</tr>
</tbody>
</table>

*Level of significance set to p < 0.0083. Precon. Ex.: Preconditioning exercise.
Table 5 reflects the relative frequency that each trial exhibited the highest vertical displacement across subjects. This analysis was conducted to determine if performing multiple trials of the vertical jump within a given condition may have produced additional PAP.
Table 5. Relative frequency (%) that each trial number resulted in the highest vertical jump by condition across 20 male subjects.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Stretch</th>
<th>Precon. Ex.</th>
<th>Combination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trial 1</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>30</td>
</tr>
<tr>
<td>Trial 2</td>
<td>50</td>
<td>30</td>
<td>40</td>
<td>25</td>
</tr>
<tr>
<td>Trial 3</td>
<td>35</td>
<td>45</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>Trial 4</td>
<td>5</td>
<td>10</td>
<td>15</td>
<td>10</td>
</tr>
<tr>
<td>Trial 5</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Precon. Ex.: Preconditioning exercise.
Peak Muscle Activation

Table 6 represents the peak muscle activation for each condition as a percent of the reference jump. There were no significant differences in peak muscle activation between any of the conditions.
### Table 6: Comparison of muscle activation levels during vertical jump by condition.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Control</th>
<th>Stretch</th>
<th>Precon. Ex.</th>
<th>Combination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right Vastus Lateralis</td>
<td>98.52</td>
<td>102.25</td>
<td>110.28</td>
<td>92.34</td>
</tr>
<tr>
<td>Right Gastrocnemius</td>
<td>101.45</td>
<td>106.23</td>
<td>112.12</td>
<td>87.26</td>
</tr>
<tr>
<td>Right Gluteus Maximus</td>
<td>98.91</td>
<td>104.88</td>
<td>112.35</td>
<td>90.09</td>
</tr>
<tr>
<td>Left Vastus Lateralis</td>
<td>94.89</td>
<td>102.76</td>
<td>108.24</td>
<td>82.90</td>
</tr>
<tr>
<td>Left Gastrocnemius</td>
<td>101.45</td>
<td>106.23</td>
<td>112.12</td>
<td>87.26</td>
</tr>
<tr>
<td>Left Gluteus Maximus</td>
<td>97.48</td>
<td>103.45</td>
<td>110.92</td>
<td>89.74</td>
</tr>
</tbody>
</table>

*All values represent mean +/- standard deviation for a given variable. Ex: Precon. Ex. Precon. Ex.*
The primary purpose of this study was to examine the independent and combined effects of performing static stretching of antagonist musculature and a preconditioning exercise on vertical jump performance. To the best of our knowledge, this is the first study to examine the additive effects of these strategies for increasing power production. The primary finding of this study was a significant increase in vertical jump displacement (1.59cm) and VJ power (98.27W) when both techniques were employed in sequence. The literature regarding each treatment type and the potential neuro-physiological mechanisms are discussed below.

Although the present study did not identify a significant independent effect of performing a preconditioning exercise on VJ performance, there was a strong trend toward enhancing performance (p = 0.012). One possible explanation for this trend is PAP. PAP is stimulated by performing a preconditioning contraction (e.g., near maximal isometric or concentric contraction) (8). To our knowledge, there is only one previous study using functional isometric exercise to elicit a PAP response and this study further assessed the influence of resistance training status on potentiation. Specifically, Berning et al. reported a significant increase in VJ height in resistance trained men at both 4 (+2.4cm) and 5 minutes (+2.6cm) post-functional isometric squat performed at 150% of 1 repetition maximum (RM)(8). However, untrained men performing the same conditioning contractions with a functional isometric squat did not experience any change in VJ performance (8). These findings suggest that resistance trained individuals may experience PAP. Unfortunately, we did not identify the current resistance trained status of participants in the present study. All participants had a history of resistance training, but not all were current lifters. The findings of Berning et al. (8) may suggest that the
average improvement in VJ height and VJ power may have been attenuated by the
participants not currently resistance training in the present study.

There is an optimal window of opportunity to utilize the increased rate of force
development experienced during PAP (28, 67). Several peripheral and central PAP
mechanisms have been proposed that may be responsible for the enhanced power
development. Peripherally, PAP is thought to be due to phosphorylation of myosin
regulatory light chains. In this process the regulatory light chain phosphorylation is
catalyzed by myosin light chain kinase. This is triggered by the release of Ca$^{2+}$ from the
sarcoplasmic reticulum. The Ca$^{2+}$ will subsequently bind to calmodulin (60). If this is the
case, the phosphorylation potentiates future contractions by modifying the position of the
myosin head and pulling it away from the thick filament backbone (60). The result is an
increased level of myosin cross bridge activity in response to submaximal concentrations
of myoplasmic Ca$^{2+}$ (56).

Centrally, PAP is speculated to result from an increased recruitment of higher
order motor units due to a decrease in neurotransmitter failure. This decrease is a result of
an increase in the efficacy of the neurotransmitter, increased quality of neurotransmitter,
or a reduction of axonal branch-point failure along afferent neural fibers (20). Evidence
suggests this could lead to a larger activation of Type IIx muscle fibers, thus increasing
the amount of power developed (29).

The magnitude of PAP is influenced by a variety of factors including:
accumulated fatigue, conditioning contraction volume, conditioning contraction type,
subject characteristics, and the type of subsequent activity (60). Immediately after a
conditioning contraction, the initial effects of PAP are greater than the negative effects of
fatigue. Subsequently, there is a brief period where power increases. Fatigue then becomes the more dominant factor leading to a decrease in performance. However, the negative effects of fatigue diminish more rapidly than the positive effects of PAP, thus a second window is present where performance is enhanced about 5-10 minutes post-exercise (25, 60).

Since the relative contribution between PAP and fatigue is crucial, conditioning contraction volume is a major factor that additionally supports the indication of two performance enhancement windows (32, 65). It has been speculated that different types of conditioning contractions lead to differences in where the PAP and fatigue mechanisms are more prevalent. Isometric conditioning contractions, as used in the present study, may produce more central fatigue (i.e., reduction in neural drive to the muscle), but are more likely to activate the peripheral mechanisms of PAP (i.e., increased percentage of phosphorylation of myosin regulatory light chains) (60). In contrast, dynamic conditioning contractions are thought to cause more peripheral fatigue (i.e., reduced force generating capacity due to action potential failure, excitation-contraction coupling failure, or impairment of cross-bridge cycling in the presence of unchanged or increased neural drive), but activate central mechanisms of PAP (i.e., increased higher order motor unit activation) (60). It can be postulated that these differences in conditioning contractions could lead to potential differences in the performance of subsequent ballistic movements. It could also be speculated that there is a lack of significance in the EMG data of the present study due to the type of conditioning contraction used. Any changes on the peripheral level would be attributed to efficiency of the contraction instead of muscle activation. These peripheral changes would not
manifest in increased or decreased EMG activity, but still lead to increased performance. Tillin et al. mused the idea that isometric conditioning contractions would lead to central fatigue and peripheral mechanisms of PAP. (60) If this supposition is correct, we would expect non-significant EMG when using an isometric conditioning contraction and expect a change in EMG data if a concentric conditioning contraction where used.

Subject characteristics may influence the PAP-fatigue response and potentially become a contributing factor to the resulting manipulation of power. Individuals who possess greater muscular strength, higher percentage of type II muscle fibers, have more resistance training experience, and a smaller power-strength ratio may benefit more from the performance enhancement associated with PAP (60). Furthermore, greater PAP is experienced when the conditioning contraction simulates the gross motor movement of the subsequent activity (60). Unfortunately, assessments of these characteristics were not conducted in the present study to substantiate these findings.

The majority of highest attempts occurred on the second or third attempt. It could be possible that this is due to a learning effect of repeated jumps. This is one possible explanation for the lack of representation of jump one as the best performance. The author of the current study suggest that this is more like due to PAP resulting from the prior jump because each subject followed this trend for each condition.

Although the present study did not identify a significant independent effect of static stretching the antagonist on VJ performance, there was a strong trend (p = 0.050). Sandberg et al. (58) found a significant increase in VJ height, VJ power, and knee extension torque at 300°/s while performing the same static stretches as used in the present study. Sandberg et al. reported a small effect size for VJ height and power while
finding a moderate effect size for knee extension torque at 300°/s (58). Although our findings were not significant, we did identify a trend supporting the findings of Sandberg and company (58). When comparing the result of the present study to the Sandberg et al. (58) study, it is important to note that Sandberg et al. (58) used a 90 second rest period between stretching and the experimental trials. Because our study had interest in the combined effects of static stretching and preconditioning exercise, where fatigue is an additional variable, we had to alter the timeline procedures. Specifically, participants in the present study had 6 minutes and 30 seconds from the conclusion of the stretches to the first VJ trial. This increased amount of time may have allowed partial recovery of the mechanisms responsible for the increase in VJ height.

There are several factors that may be responsible for the effects of static stretching of the antagonist on vertical jump performance. The general idea behind this is that static stretching of the antagonist will decrease the ability of the antagonist to produce a resistive force; therefore, increasing the ratio of force produced by the agonist. First, static stretching may decrease the musculotendinous stiffness of the stretched muscle, thus decreasing the resistance applied by the antagonist muscle. For instance, Fowles et al. (24) found a decrease in passive torque of the plantarflexors 5 minutes after an acute bout of maximally tolerable passive stretch. Reduced musculotendinous stiffness can lead to an altered length-tension relationship (24). After an intense stretch the musculotendinous unit has been observed to acutely increase length at any given joint angle. This length change could orient muscle fascicles such that they are on the descending limb of the length-tension relationship (2). This would result in less than optimal force generation upon stimulation. There is potential that a passive stretch could
cause mechanical breakage of stable myosin-actin crossbridge sites (45). This would reduce the passive tension of the muscle and lead to less force generation during contraction, therefore, effectively increasing the relative contribution of force produced by the agonist muscle.

Statically stretching the antagonist muscle may also enhance power production in the agonist by producing a suppressed H-reflex in the antagonist. Reduced motor neuron excitability is illustrated by a depressed H-reflex (21, 54). It has been proposed that the depressed H-reflex is a result of the reduction in excitatory drive from the Ia afferent nerve endings on to the \(\alpha\)-motorneurons (2). Avela et al. observed a \(~50\%\) reduction in the H-reflex in the stretched muscle measured at the end of a repeated and prolonged passive stretching treatment of 1 hour in duration. The results were interpreted to indicate an increased compliance of the muscle, leading to a reduced response of the muscle spindle. Consequently, this reduction initiated a disfacilitation of the \(\alpha\)-motorneuron pool in the stretched muscle (2).

Some investigators postulate a reduction in force through autogenic inhibition of the stretched muscle via activation of the Golgi tendon organ (GTO) (36, 55). Others exclude this possibility because the GTO is sensitive to contraction of muscle, but not typically sensitive to passive stretch (2, 24). In the event the GTO is activated during a maintained stretch, the inhibitory effects are momentary.

The current study did not identify significant differences in the EMG activity between conditions. Due to the inherent properties of EMG analysis, the ability to find statistically significant findings decreases as a movement becomes more dynamic as a result of additional noise in the system and spatial filtering (15). However, previous
investigations have noted significant differences in EMG outcomes on VJ performance (36, 66). Wallmann et al. found a 17.9% increase in mean EMG activation in the gastrocnemius following three – 30 second static stretches, but a decrease of 5.6% on VJ performance. Although counterintuitive, these statistically significant findings imply that static stretching of the agonist may lead to greater muscle activation, but decreased performance (66). Hough et al. (36) examined the effects of no stretch, static stretch, and dynamic stretch of agonist (vastus medialis) on mean EMG activation and VJ performance. The only significant finding of the EMG portion of the study was an increase in mean EMG activity of the vastus medialis in the static stretching condition compared to the dynamic stretching condition. Hough et al. also found a difference between all three groups in VJ performance (static stretch < no stretch < dynamic stretch) (36).

There were several limitations to the present study. The EMG data had high variability that diminishes the statistical power and increases the probability of type II error. This is likely due to the utilization of EMG during a highly ballistic movement. Dynamic movements inherently create more noise in the system producing variability. Secondly, the participants were advised to omit physical activity before and between sessions, but we cannot be certain of the adherence to this request. The resolution of the Vertec measuring device is 1.27cm. This leaves a window of opportunity open for omitting minute differences. The Harman equation (33) was used to calculate peak power output. This equation uses body mass and the current authors only measured body mass on the first day of testing. This same measurement was used to calculate power output on both days of testing for all treatments. This introduces a possibility of error in the
calculation for the non-measured day of testing. However, all participants were tested within a three-day period, so we expect the fluctuation in body mass to be minimal for each participant. Lastly, we did not measure the hip/torso angle during the functional isometric deadlift. All participants were familiar with performing a deadlift, but it is widely known that deadlift form can be different among experienced lifters. Although the participants were instructed on proper form by a National Strength and Conditioning Association Certified Strength and Conditioning Specialist (CSCS), the level of activation between the quadriceps and hamstrings could vary between subjects based on deadlift form. This could result in different levels of potentiation between participants.
CHAPTER V

SUMMARY & CONCLUSIONS

In summary, this study was developed to determine whether or not the use of static stretching and preconditioning exercises could be used to acutely augment power to enhance performance on a vertical jump test. Previous authors have shown that static stretching and preconditioning exercises can independently enhance vertical jump performance. The results of this study were unable to reproduce the effects of each of these neuromuscular manipulations independently, but was successful in increasing performance on vertical jump test when combined.

Practical Applications

Static stretching of the antagonist muscle and performing a preconditioning exercise resulted in an increased VJ height and power output. It appears advantageous to perform these methods of power manipulation prior to performing an explosive movement. It can be speculated that these results may transfer to other single-bout power events (high jump, long jump, shot-put, etc.). Athletes who participate in explosive power sports may benefit from similar procedures. However, it is important that athletes practice these procedures prior to competition because the effects vary by subject characteristics, time periods, and preconditioning exercises. The protocol to elicit the greatest results likely varies from individual to individual.

Further research should evaluate these procedures on other muscle groups and measures of power output. Furthermore, future research should explore how subject characteristics (muscular strength, fiber-type distribution, training level, and power-
strength ratio) interact with these procedures. Potential opportunities lie in determining the possible mechanisms responsible for the noted increase in power.

In conclusion, despite the presence of strong (nonsignificant) trends of using static stretching and preconditioning exercise techniques to enhance vertical jump performance and peak power output, the combined implementation of these techniques produced significant additive effects. These techniques appear to be complimentary in nature and may enhance power performance. The results of the present study indicate utilization of both static stretching of the antagonist and preconditioning exercise techniques in succession will lead to the greatest enhancement in vertical jump height and peak power output.
REFERENCES


VITA

The author, Mark Ryan Mason, was born in Princeton, KY on August 22, 1987 and raised in Cerulean, KY. He attended Hopkinsville Community College from 2005 – 2007, and received an Associates Degree in Science and an Associate Degree in Arts. He attended University of Kentucky from 2007 – 2010, and received a Bachelor of Science in Kinesiology. He began his work towards a Master’s of Science in Exercise Physiology at the University of Kentucky in the Fall of 2012.