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Acute Effects of Antagonist Stretching on Jump Height and Knee Extension Peak Torque

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ACUTE EFFECTS OF ANTAGONIST STRETCHING ON JUMP HEIGHT AND KNEE EXTENSION PEAK TORQUE

by

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ABSTRACT

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A great deal of research has shown decrements in force and power following static stretching. There has been little research investigating the acute effects of static stretching of the antagonist on the expression of strength and power. The purpose of this study was to investigate the effects of static stretching of the antagonist muscles on a variety of strength and power measures. Sixteen active males were tested for vertical jump height and isokinetic torque production in a slow knee extension (KES) at 60°/s and a fast knee extension (KEF) at 300°/s. Electromyography was taken during knee extension tests for the vastus lateralis and the biceps femoris muscles. Participants performed these tests in a randomized counterbalanced order with and without prior antagonist stretching. All variables for stretching and non-stretching treatments were compared using paired *t* tests at an alpha of .05. Paired samples *t* tests revealed a significant ($p = .034$) difference between stretch KEF and non-stretch KEF conditions.

There was no significant ($p > .05$) difference between KES stretch and non-stretch conditions. Vertical jump height was significantly ($p = .011$) higher for the stretching treatment than the non-stretching treatment. Vertical jump power was also significantly higher ($p = .005$) in the stretch versus the non-stretch condition. Paired samples *t* test indicated no significant ($p > .05$) difference between testing conditions for electromyography, represented as a percentage of maximal voluntary contraction (MVC). These results suggest that stretching the antagonist hamstrings prior to high speed isokinetic knee extension increases torque production. It also demonstrated that stretching the hip flexors and dorsi flexors may enhance jump height and power. Practitioners may use this information to acutely enhance strength and power performances.

(61 pages)

PUBLIC ABSTRACT

Acute Effects of Antagonist Stretching on Jump Height and Knee Extension Peak Torque

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John B. Sandberg

There has been a great deal of research investigating the effects of stretching preceding strength training, jumping and running. Static stretching, as the name implies, involves stretching a muscle and holding the stretch with minimal or no movement for a given duration. Several studies have shown that static stretching before strength training or jumping can actually result in poorer performances. Static stretching may reduce the nervous system's ability to recruit muscles or it may reduce the ability of the muscle to produce force directly.

All major skeletal muscles have an opposing or antagonist muscle that acts in opposition to it. For example the quadriceps muscles extend (increase the angle between the foot and upper thigh) the knee and the hamstrings perform the opposite movement (knee flexion). Previous research has investigated the effects of stretching on the muscle primarily involved in the movement. No known research has attempted to determine the effects of stretching the antagonist muscle for a given movement. Because antagonist muscles provide a braking force to the movement of their opposing muscles, stretching the antagonist muscle could reduce this braking force. This could potentially enhance

strength and power following antagonist stretching.

The current study investigated the hypothesis that stretching the antagonist muscles would improve strength and jumping ability. Sixteen active males were tested for vertical jump height and knee extension strength on a machine that controlled lifting speed. The participants tested knee extension strength at a fast and slow speed. Electromyography, a measure of a muscle's electrical activity, was taken during knee extension tests for the quadriceps and the hamstring muscles. This test was done to determine if antagonist stretching affects the nervous system's ability to recruit the quadriceps and hamstrings during knee extensions. Each test was performed with and with out antagonist stretching.

Stretching the antagonist muscles resulted in a significant improvement in knee extension strength at the fast speed. It also significantly improved jumping ability. Antagonist stretching did not result in a significant difference for electromyography or knee extension strength at the slow speed. Antagonist stretching may provide a method for enhancement for high velocity activities such as jumping. Strength and conditioning practitioners could use this information to implement antagonist stretching during training and competition to increase performance and/or overload during training.

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CHAPTER 1

INTRODUCTION

Traditionally, stretching has been used as a part of pre-exercise and competition warm-up (Young & Behm, 2002). Various forms of stretching have been utilized to increase the range of motion around a joint (Bandy, Irion, & Briggler, 1997). Stretching has been proposed as a method to reduce the risk of or prevent injury (Johagen, Nemeth, & Eriksson, 1994; Safran, Seaber, & Garrett, 1989) and improve performance (Wordell, Smith, & Winegardner, 1994). In recent years, however, the practice of including stretching as a warm up modality has been questioned. A review conducted by Shrier (1999) concluded that it is unlikely that pre-activity static stretching prevents injury. There is also a great deal of evidence indicating that pre-activity static stretching has a negative impact on strength and power performances (Church, Wiggins, Moode, & Crist, 2001; Cornwell, Nelson, & Sidaway, 2002; Fowles, Sale, & MacDougall, 2000; Kokkonen, Nelson, & Cornwell 1998; Nelson & Kokkonen, 2001; Power, Behm, Cahill, Carrol, & Young, 2004; Young & Elliot, 2001).

To the author's knowledge there has been no published research investigating the effects of static stretching for the antagonist musculature on subsequent expression of strength and power. Therefore, the purpose of this study was to investigate the effects of static stretching for the antagonist muscles on peak torque of the knee extensors recorded at a slow knee extension (KES) of 60°/s and a fast knee extension (KEF) of 300°/s, and vertical jump height (VJ) and power (VJP). The study also sought to determine whether

antagonist stretching would affect neural activity in the agonist and antagonist musculature.

Research Questions

1. Will static stretching of the antagonist muscles of the quadriceps (i.e., hamstrings) improve isokinetic peak torque of the knee extensors?

2. Will static stretching of the hip flexors and dorsi flexors prior to performing a vertical jump improve jump height and power?

3. Will static stretching of the hamstrings prior to performance of a knee extension affect neural activity of the vastus lateralis and biceps femoris.

Research Hypotheses

Stretching of the antagonist muscles prior to strength and power performance could potentially lead to enhanced performance. Gains in strength might be accompanied with an increase in neural activity of the agonist and neurological inhibition of the antagonist (Carolan & Cafarelli, 1992; Häkkinen et al., 1998). Stretching the agonists prior to a given movement may decrease the agonist muscle strength and power, perhaps through decreased neural drive (Cornwell et al., 2002; Fowles et al., 2000; Vujnovich & Dawson, 1994). Conversely, stretching the antagonists may result in their inhibition and reciprocally facilitate increased activity of the agonists, with subsequent improvements in strength and power related performance.

If this hypothesis is proven true it could have strong practical implications.

Individuals in sports and activities characterized by brief maximal efforts (e.g. throwers, jumpers, and sprinters in track and field, power lifters and weight lifters) could potentially benefit by including antagonist stretching prior to performances requiring high force and/or power. Antagonist stretching could also be used during training to increase the overload placed on the agonist musculature via enhanced motor unit recruitment.

The major muscles involved in the knee extension are the quadriceps group (vastus lateralis, vastus medialis, vastus intermedius, and rectus femoris) (Baechle & Earle, 2000). The antagonist musculature to the quadriceps is the hamstring group, which includes the semitendinosus, semimembranosus, and biceps femoris. It has been proposed anecdotally that increasing the range of motion of the quadriceps prior to a leg curl exercise will increase the amount of motor units used in the hamstrings during this exercise (Poliquin, 2010). It is hypothesized that stretching the hamstrings, the antagonist musculature of the quadriceps, will decrease the quadriceps force production capabilities. This may facilitate the action of the agonist musculature, thereby improving knee extension performance.

The hip extensors (gluteals and hamstrings) are vital to jumping performance (Bobbert & van Zandwijk, 1999; Goodwin et al., 1999). The plantarflexors are also involved in jumping (Goodwin et al., 1999; Hay & Nohara, 1990). It is hypothesized that stretching the antagonist musculature of the hip extensors and plantar flexors (i.e., hip flexors and dorsi flexors) will decrease their force production capabilities, thereby improving VJ performance via enhanced activity of the hip extensors and plantar flexors.

Pre-activity static stretching has been shown to reduce electrical activity of the

muscle being stretched (Cornwell et al., 2002; Fowles et al., 2000; Vujnovich & Dawson, 1994). Therefore, it is hypothesized that electrical activity of the hamstrings will decrease following stretching. A decrease in electrical activity of the antagonist musculature has been shown to increase electrical activity of agonist musculature (Mayhew, Norton, & Sahrmann, 1983). It is therefore hypothesized that electrical activity of the quadriceps will increase following stretching and this will be evident by an increase isokinetic knee extension torque.

Limitations/Delimitations

One of the limitations of this study is the potential confounding factor of the dual role of the rectus femoris as a hip flexor and knee extensor during the VJ assessment (Van De Graaff, 1998).

Definition of Terms

Autogenic inhibition: Reflex inhibition of a motor unit in response to excessive tension in the muscle fibers it supplies. Muscle tension is monitored by the Golgi tendon organs. Autogenic inhibition is a protective mechanism, preventing muscles from exerting more force than the bones and tendons can tolerate.

Electromyography (EMG): Measurement of action potentials in muscle fibers.

Hoffmann reflex (H reflex): A response that is elicited artificially by electrical stimulation of a peripheral nerve and selective activation of the Group Ia (largest diameter) afferents. The afferent volley activates motor neurons and elicits an EMG and force response. The

H reflex is used as a test of the level of excitability of the motor neuron pool (Enoka, 2002).

Integrated electromyography (IEMG): The sharp peaks (high frequencies) present in a rectified EMG can be smoothed by integration, an electronic process that consists of filtering the high frequency content of the signal (Enoka, 2002).

Musculotendinous unit: The combination of muscle and associated connective tissue structures that is involved in transmitting the force exerted by the muscle fibers to the skeleton.

Rectified EMG: Rectification consists of taking the absolute EMG signal and removing or flipping over the negative phases of the EMG signal. This creates a more easily read signal (Enoka, 2002).

Series elastic component: The component of the Hill model (a model describing the components of a single muscle fiber) that accounts for the elasticity of muscles in a series with the contractile element (Enoka, 2002).

CHAPTER II

LITERATURE REVIEW

The first section of the literature review will give an overview of adaptations to the agonist and antagonist musculature during training. The implications of these adaptations to antagonist stretching will be discussed. The following section will review the effects of pre-activity static stretching on various measures of strength and power. The possible mechanisms for reductions observed in strength and power following static stretching will be explored. The final section will discuss antagonist stretching prior to the vertical jump. The potential limitation of the rectus femoris dual role in hip flexion and knee extension will be addressed here. The literature review will then summarize the theoretical basis for this study and state its purpose.

Adaptations to Antagonist Musculature Following Resistance Training

Concurrent neural adaptations to both the agonist and antagonist muscles are important to facilitate greater torque and power development (Carolan & Cafarelli, 1992; Häkkinen et al., 1998). The net external force applied during a movement is proportional to the force produced by the agonist and inversely proportional to the force produced by the antagonist muscles (Baratta et al., 1988; Draganich, Jaeger, & Kralj, 1989). Therefore, inhibiting the force produced by the antagonist muscles may allow the agonist muscles to apply greater force and power production.

Häkkinen et al. (1998) trained 11 older men (M = 73 years old SD = 3), 10 middle

aged men (*M* = 40 years old *SD* = 2), 10 older women (*M* = 67 years old *SD* = 3), and 11 middle aged women (*M* = 39 years old *SD* = 3) for 6 months in a periodized strength training program involving heavy resistance training and lighter explosive training. Maximal isometric leg extension torque at a knee angle of 107 degrees and squat jump height were measured pre- and post-treatment. The electromyogram (EMG) activity of the vastus medialis, vastus lateralis, and long head of the biceps femoris was also taken during isometric strength tests pre- and post-treatment. All groups demonstrated significant increases in maximum isometric force, rate of force development, and squat jump height. All groups also demonstrated significant increases in maximum integrated EMGs of the agonist vastus medialis and vastus lateralis. The older group also had significant reductions in integrated EMGs of the antagonist biceps femoris. Increases in activity of the agonist along with concurrent decreases in activity of the antagonist in isometric actions have also been observed in younger individuals following strength training (Carolan & Cafarelli, 1992).

Other studies have demonstrated that acute reductions in the activity of the antagonist muscles may augment power production of the agonist muscles. Baker and Newton (2005) examined the effects of alternating opposing movement patterns (bench press throws and bench pulls) on power output. Participants were randomly assigned to either a traditional group, which performed two sets of five repetitions of bench throws with 3 min rest between, or an antagonist group which performed a set of a heavy bench pulls between the sets of bench throws. Power outputs for the bench press throws were measured with a specially designed Smith weight machine on each set for both groups.

The antagonist group demonstrated significantly greater ($p \leq .05$) power output than the traditional group. Acute strength increases have been demonstrated previously in studies that alternated antagonist and agonist muscles groups in resistance exercise workouts (Burke, Pelham, & Holt, 1999). Baker and Newton theorized that the augmentation to power in their study may have been due to enhanced reciprocal inhibition of the antagonist.

As the agonist muscle contracts, spindle intrafusal fibers are activated sending signals from 1a afferents to the spinal cord. This may stimulate the activation of an inhibitory interneuron, resulting in a decrease in the excitability of motor neurons innervating the antagonist muscle (Katz, Penicaud, & Rossi, 1991). Mayhew et al. (1983) assessed the relative participation of the hip extensors and abdominal musculature in 5 men and 6 women during a unilateral straight leg raise (USLR). EMG activity of the rectus abdominis, external obliques and medial hamstrings were recorded bilaterally. Each participant performed a supine USLR three times with two different modes, preferred and relaxed. The preferred mode was performed first in which the participants were asked to perform the USLR through a comfortable range of motion. The relaxed mode was performed by instructing the participants to relax the contralateral extremity while the USLR was executed. Results showed that 9 of the 11 participants' medial hamstrings were active during the USLR with the preferred mode. When participants were instructed to relax the contralateral limb, abdominal activity significantly increased ($p < .001$). This study indicates that when the antagonist hip extensors are activated the agonist abdominal EMG activity decreases. It also indicates that verbal instruction to

relax the antagonist musculature will increase activity of the agonist muscles. An increase in activity of the antagonist may result in a decrease in activity of the agonist through reciprocal inhibition.

Pre-activity static stretching has been shown to acutely decrease strength, power, and EMG activity of agonist muscles (Church et al., 2001; Cornwell et al., 2002; Fowles et al., 2000; Herda, Cramer, Ryan, McHugh, & Stout, 2008; Kokkonen et al., 1998; Nelson & Kokkonen, 2001; Power et al., 2004; Young & Elliot, 2001). A decrease in antagonist activity has been shown to increase EMG activity of the agonist (Mayhew et al., 1983). If one could acutely decrease the strength and power of the antagonist muscles through static stretching it could theoretically cause an increase in strength and power of the agonist muscles, via reciprocal inhibition.

Possible Mechanisms for Reductions in Strength and Power Following Stretching

There are two primary mechanisms that may contribute to the decreases in strength and power performances following stretching of agonist muscles. The first proposed mechanism is that stretching causes transient mechanical changes at the level of the musculotendinous unit, most likely a reduction in stiffness. Stretching could potentially alter the length-tension relationship increasing the time needed to "take up the slack" in more compliant series elastic components (Caldwell, 1995). Wilson, Murphy, and Pryor (1994) found a high correlation between concentric and isometric bench press strength and musculotendinous stiffness. However, other research indicates a negative

correlation between musculotendinous stiffness and stretch shorting performances of the lower body (Walshe & Wilson, 1997; Walshe, Wilson, & Murphy, 1996). Walshe and Wilson (1997) and Walshe et al. (1996) hypothesized that the forces transmitted from the skeletal system to the musculature of stiffer participants reduced their ability to attenuate higher eccentric loads due to less effective contractile dynamics and greater levels of reflex inhibition.

Another proposed mechanism for the acute decrease in force and power capabilities following stretching is reduced neural drive. Stretching has been shown to cause autogenic inhibition (H-reflex depression) (Vujnovich & Dawson, 1994). A decrease in the sensitivity of sensory receptors (e.g., spindle fibers) could potentially decrease central drive to alpha motor neurons (Bigland-Ritchie, Furbush, Gandevia, & Thomas, 1992). The mechanisms behind declines in force and power following stretching are still not fully understood.

Herda et al. (2008) postulated reductions in strength following static stretching are due to mechanical factors. They investigated the effects of static versus dynamic stretching on peak isometric torque, electromyography and mechanomyography (MMG), a measure of mechanical oscillation in the muscle that may reflect mechanical adaptations, of the biceps femoris at 61˚, 81˚, and 101˚. Four repetitions of three static stretches were held for 30s each, whereas four sets of dynamic stretching exercises were performed for 12-15 repetitions with each set lasting 30 s. Peak torque decreased after static stretching at 81˚ ($p = .019$) and 101˚ ($p = .001$) but not at 61˚. Peak torque did not change ($p > .05$) after dynamic stretching. EMG amplitude remained unchanged with

static stretching ($p > .05$) but increased after dynamic stretching ($p < .001$) at 101˚ and at 81˚ ($p < .001$). Mechanomyography amplitude increased in response to static stretching at 101˚ ($p = .0003$) but not other angles. Dynamic stretching increased ($p \leq .05$) MMG amplitude at all joint angles. Because there was no change in EMG activity the authors suggested that decreases in strength following static stretching are due to mechanical rather than neural factors in the biceps femoris. The MMG amplitude however was not decreased for any static stretching conditions and actually increased at 101˚.

In contrast to Herda et al. (2008), Fowles et al. (2000) found EMG activity was significantly decreased for the first 15 min following static stretching, and force decrements were greatest during this time. Electrical activity did, however, return to normal after 15 min while force decrements remained for 60 min. These authors theorized that neural factors played a bigger role in strength decreases early, but as time passed, the reduction in maximum voluntary contractions originated peripherally in the muscle.

Cornwell et al. (2002) tested the explosive capabilities of the plantar flexors following static stretching. Static passive stretching exercises were performed for 30 s, 3 times on each leg. They found a reduction in stiffness and EMG activity, but concluded the change in stiffness was unlikely sufficient to cause changes in strength and power. They theorized that the decreases to performance were likely due to neural factors. Knudson, Bennett, Corn, Leick, and Smith (2001) found no difference in the kinematics of the vertical jump with or without static stretching. These authors hypothesized that if stiffness was reduced, changes in knee angles would have been witnessed during

jumping. Stretching likely reduces neural drive to the muscle stretched.

Antagonist Stretching for the Vertical Jump

It is difficult to distinguish a true antagonist in a complex movement such as the VJ. Hip extension, knee extension, and planar flexion are all important movements in jumping (Goodwin et al., 1999). Stretching the hip flexors has however been anecdotally observed to enhance jumping ability (Poliquin, 2004). This may be because hip extensors play a vital role in jumping. Bobbert and van Zandwijk (1999) studied the importance of stimulation dynamics for force development in vertical jumping. They assessed 21 males in maximum height jump squats. As a measure of the signal dynamics rise time (RT), the time taken by the signal to increase from 10% to 90% of its peak value, was used. Rise time was calculated from smoothed rectified electromyograms (SREMG) of seven lower extremity muscles, net moments about the hip, knee, and ankle joints, and components of the ground reaction force vector. A strong correlation (r = .88) was found between RT of SREMG of the gluteus maximus and RT of the vertical component of the ground reaction force vector. It was speculated that for an effective transfer from joint extensions to vertical motion, the center of mass needs a forward component during push off. Given the starting position of the squat jump, only the hip extensor muscles are able to generate such a forward acceleration of the center of mass. Therefore, the greater the RT of the hip joint moment and RT of the gluteus maximus SREMG, the greater the RT of the vertical component of the ground reaction force.

Active lengthening of the hip flexors has been proposed as a way to

simultaneously increase flexibility of short hip flexors while concomitantly increasing the function of the antagonist hip extensors (White & Sahrmann, 1994). Winters et al. (2004) found that both passive and active stretching of the hip flexors increases active hip extension. Although no strength or EMG measures were taken it is likely that increased hip flexor flexibility improved hip extension function. Efficient hip extension is important to VJ performance (Bobbert & van Zandwijk, 1999). To the author's knowledge the effects of stretching the hip flexors on jumping ability has not been investigated in reviewed research. If however hip extension could be enhanced through stretching the hip flexors this could theoretically improve VJ.

Additionally, some evidence suggests that lengthening the rectus femoris prior to jumping may not have a detrimental effect on performance. A three dimensional model of the take off leg in the running long jump, running vertical jump, and standing vertical jump was developed to compute the muscle-tendon lengths of seven muscle groups, the gluteus maximums, hamstrings, vastus medialis and lateralis, rectus femoris, soleus, and gastrocnemius (Hay & Nohara, 1990). The data obtained from digitizing the films were used to determine how the lengths of these muscles changed during the takeoff. All muscle groups in all three jump types either shortened or lengthened and then shortened during takeoff, with the exception of the rectus femoris. The rectus femoris was the only muscle which lengthened throughout the takeoff in all jumps. Consequently, lengthening of the rectus femoris prior to performance of the vertical jump is unlikely to have a negative effect on performance despite its dual role in hip flexion and knee extension.

Hakkinen and Komi (1985) investigated the effects of 24 weeks of training

involving various jumps with and without light weights on jump height. Changes in integrated electromyography (IEMG) for the vastus lateralis, vastus medialis, and rectus femoris muscles during training were also measured. Training resulted in a 21% increase in jump height. Significant ($p < .05$) increases in IEMG of the vastus medialis and vastus lateralis were observed in squat jumps during the training. No significant changes were observed in the IEMG of the rectus femoris. This study indicates that neural adaptations to the vastus medialis and vastus lateralis may be more important to improved jumping ability than adaptations to the rectus femoris. Although this study was investigating chronic not acute adaptations to training, it could be theorized that reductions in force and power, via static stretching, to the vastus medials and vastus lateralis would have a greater negative effect on VJ performance than reductions in strength and power to the rectus femoris from stretching. Attempts will be made in the methods to minimize any stretch at the knee joint during stretching of the hip flexors.

Stretching the antagonist could theoretically reduce the opposing torque of the antagonist muscle groups, resulting in greater net torque for the agonist muscle groups, thereby enhancing performance. It has been proposed anecdotally that stretching the antagonist will acutely enhance strength and power related performances by means of decreased neural drive to the antagonist, thereby facilitating the effectiveness of the agonist muscle groups (Poliquin, 2004, 2010). The purpose of this study was to investigate the effects of antagonist stretching on a variety of variables that measure strength and power.

CHAPTER III

METHODS

Experimental Approach to the Problem

To determine if stretching the antagonist musculature affects performance, participants were tested for peak knee extension torque at 60˚/s and 300˚/s and for VJ. All tests were performed with and without preceding antagonist stretching for each participant. The study used a within group design, and the treatment was provided in a randomized counter-balanced order. Participants underwent the KES and KEF on the same day with or without the stretching treatment; they then received the opposite treatment 1 to 3 days later. The KES and KEF was performed in randomized order on the first testing day. The opposite order was repeated with the opposite treatment on the second testing day. The VJ was tested by itself on two separate days with or without stretching treatment with one to three days between each treatment.

Participants

A power analysis revealed that a minimum of 11 participants would be necessary to theoretically achieve statistical significance with a power of .80 and an alpha of .05. Sixteen active males participated in the study. Participants engaged in resistance training a minimum of two times a week for the previous 6 months. All participants were free from musculoskeletal, cardiovascular, and metabolic disorders at the time of the study (Appendix A). The participants were given written informed consent (Appendix B). The

study was approved by the university's institutional review board.

Stretching Treatments

All stretches for the KES, KEF, and VJ, were held for 30 s and repeated three times with 20 s rest between stretches. Previous research has recommenced holding static stretches for duration of 30 s (Bandy et al., 1997; Chan, Hong, & Robinson, 2001). A 90 s rest period was provided between stretching and knee extension and VJ tests.

Stretches prior to the knee extension tests emphasized the hamstring group. The participant laid supine on a training table. While stabilizing the opposing limb, the investigator put one hand on the participant's heel and the other hand just above the knee. The investigator then pushed the participant's heel and took them into knee extension and hip flexion (see Figure 1).

The stretching treatment prior to the vertical jump emphasized the stretching of the hip flexors and dorsi flexors. To stretch the hip flexors, the participant was positioned in a half-kneel position. For comfort a foam pad was placed under the knee of the kneeling limb. The participant was instructed to keep an erect upper torso. The hip that was posterior was then extended by contracting the gluteals. The participant was then instructed to internally rotate the leg, or turn his foot out (see Figure 2). Internal rotation stretches the hip flexors because their insertion point is the lesser trochanter of the femur (Lorenz, 2007).

Figure 1. Hamstring stretch performed prior to knee extension.

Because the knee was placed in only partial flexion, a greater emphasis of stretch was placed at the hip than the knee. Therefore, the rectus femoris, which has a dual role of hip flexion and knee extension, was lengthened more at the origin (anterior superior iliac spine) than the insertion (tibial tuberosity via the quadriceps tendon).

Figure 2. Hip flexors stretch performed prior to vertical jump.

To stretch the dorsi flexors, the subjects lay supine on a training table with their feet hanging freely off the edge of the table. An investigator put the foot into plantar flexion by pulling on the toes and pushing on the heel (see Figure 3).

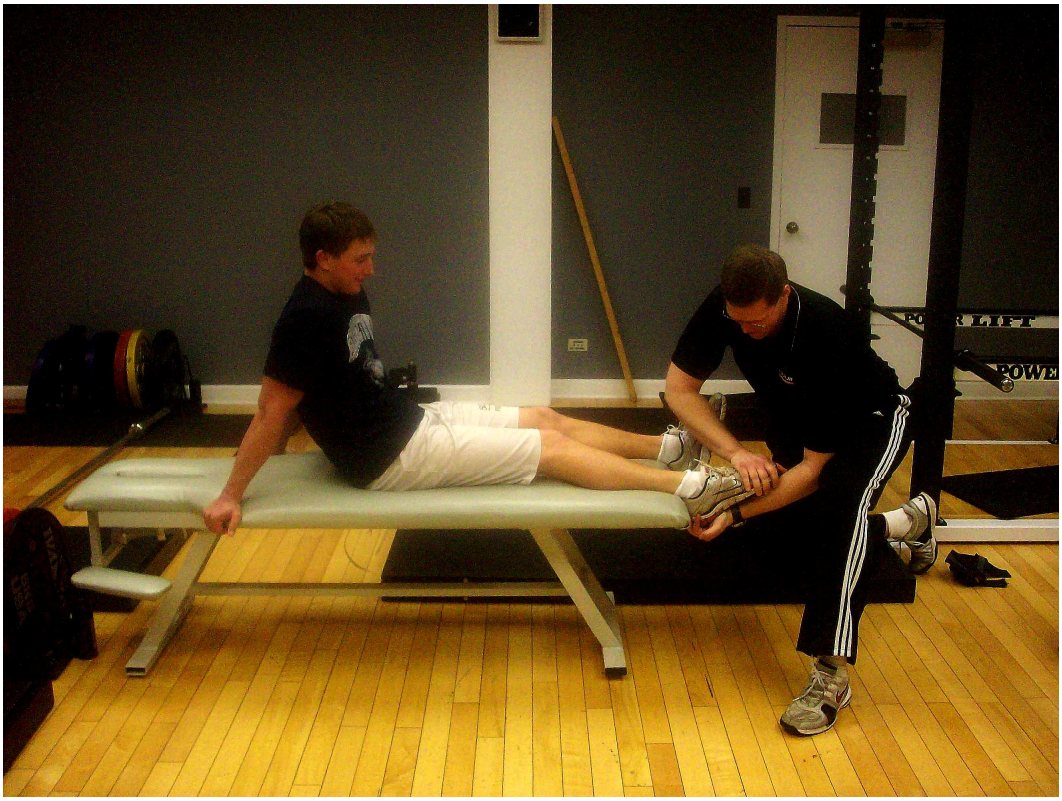

Figure 3. Dorsi flexor stretch performed prior to vertical jump.

The dorsi flexors were stretched to a point of mild discomfort. The dorsi flexors were stretched first followed by the hip flexors.

Isokinetic Testing

Knee extensors peak torque was measured on a Biodex isokinetic dynamometer (Biodex, Shirley, NY). Calibration was performed prior to testing. Participants were tested in a seated position with straps placed over their waist and distal thigh for stabilization. The tibial pad was placed and secured approximately two finger widths proximal to the medial maleolus on the dominant leg. The axis of rotation of the

dynamometer was aligned with the medial epicondyle of the knee. Concentric peak torque of the knee extensors was recorded at 60˚/s and 300˚/s. Each testing velocity was performed in a randomized order with 10 min rest between maximal tests. During stretching treatment, the stretching protocol was repeated before each maximal attempt at each testing velocity. Five maximal attempts were made and the highest value was used for data analysis. A similar five-repetition isokinetic protocol has been shown to be reliable (r = .95) for both 60°/s and 300˚/s (Feiring, Ellenbecker & Derscheid, 1990).

Electromyography

Electromyography (EMG) (Biopack MP 150, Goleta, CA) was performed on the vastus lateralis and the long head of the biceps femoris during knee extension tests. Positioning and placement of electrodes was determined using procedures described by Herda et al. (2008). Before applying the EMG electrodes, the skin at the placement sight was shaved, rubbed with alcohol, and slightly abraded to ensure good surface contact and to reduce skin resistance. Bipolar surface electrodes (2.5 cm interelectrode distance) were placed at the approximate center of each muscle belly. A ground electrode was applied to the tibial tuberosity. For the biceps femoris, the electrodes were placed at 50% of the distance from the ischial tuberosity to the lateral epicondyle of the tibia. For the vastus lateralis, the electrodes were placed at 50% of the distance between the greater trochanter and the lateral epicondyle of the femur. The positions of the electrodes were marked with a small ink mark on the skin. The precise distance was also recorded and used for electrode placement in all conditions. The electrodes were placed before commencing the

stretching treatments.

The raw EMG signals were pre amplified 100 times at the electrode site, then further amplified for a total gain of 5,000 with a band width of 10-500 Hz. A low pass filter was used with a cut off frequency of 250 Hz. A high pass filter was used with a cut off frequency of 25 Hz. As recommended by Basmajian and DeLuca (1985), the EMG signal was smoothed by integration by root mean square.

It has been proposed that if EMGs are to be compared between trials that require the reapplication of electrodes, between muscles, or individuals that they should be normalized (Burden, 2010; DeLuca, 1997). This was done by taking the EMG from each treatment and comparing it to a reference contraction of the same muscle with the same electrode placements. To represent reference contraction, a maximal voluntary contraction (MVC) was performed with the dynamometer set at 45˚ of knee flexion. Participants were tested in a seated position the same as described above with torque testing. Velocity was set at 30˚/s and participants performed 3 maximal knee extensions with a 5 s isometric hold at the terminal range of motion. Participants were instructed to exert maximal effort against the tibial pad. The MVC was performed then participants rested 10 min before performing stretching and randomized isokinetic testing. The peak EMG voltage for the vastus lateralis and biceps femoris for KES stretch, KES non-stretch, KEF stretch, and KEF non-stretch was divided by the peak EMG voltage for the MVC. The EMG results are represented as a percentage of MVC. A summary of the time line for isokinetic knee extension and EMG testing is shown in Figure 4.

Vertical Jump Testing

The vertical jump test was performed using a Vertec device (Sports Imports, Columbus, OH) according to Harman, Garhammer, and Pandorf (2000). The investigator adjusted a vertical column with vanes low enough that the participant could register a standing reach measurement. The participant then stood so that the dominant hand reached straight upward and directly below the center of the vanes. The highest vane that could be pushed forward while standing flat-footed determined the standing reach height. The same reach height was used for both trials. The vertical column was then raised to accommodate the jumping ability of the participant. The participant was allowed a counter movement with no approach step. The participant then jumped to the highest vane possible. Participants were allowed to jump until they were unable to touch a higher vane on two consecutive trials. Jump height was determined by subtracting the distance between the highest vane touched and the standing reach. Jump height and body mass were used to calculate absolute vertical jump power using the Harman equation (Harman, Rosenstein, Frykman, Rosenstein, & Kraemer, 1991). This test was chosen because it is a commonly used field test to measure power. The test has been found to have high reliability (r = .94) (Young, MacDonald, Heggen, & Fitzpatrick, 1997).

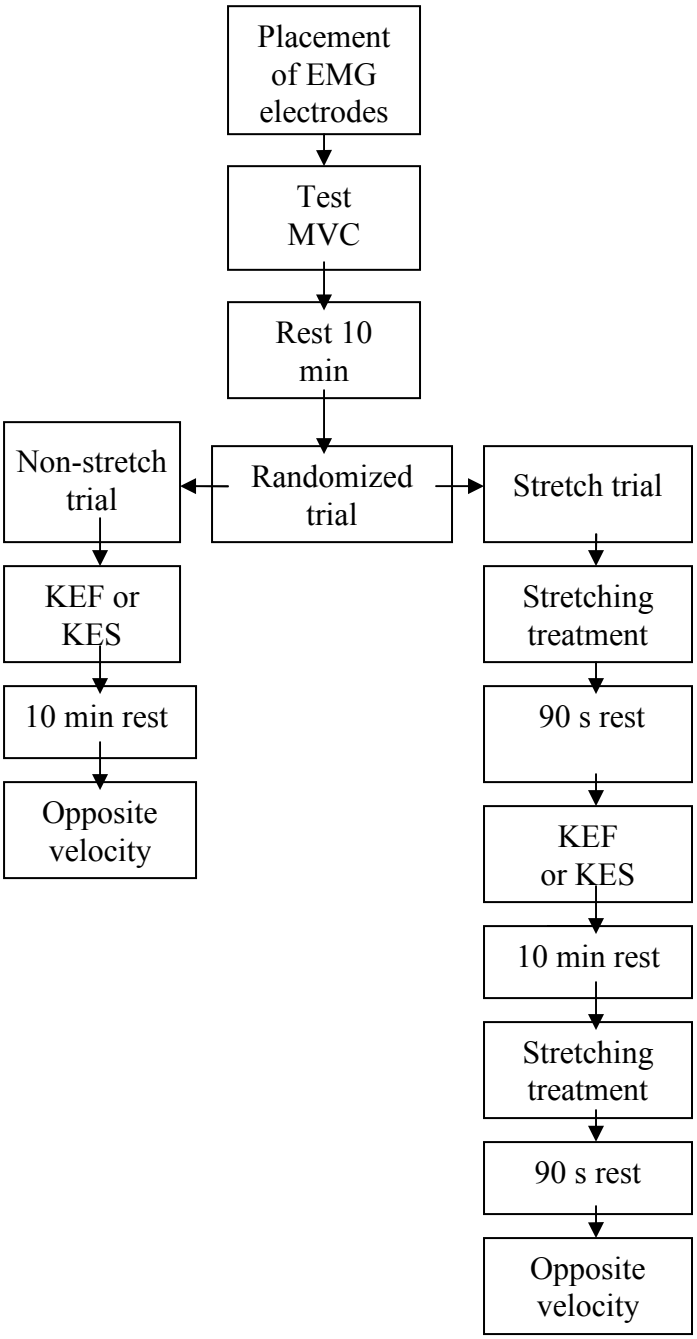

Figure 4. Summary for isokinetic knee extension trials.

Statistical Analyses

Statistical significance for the mean difference between the stretch and non-stretch trials for the KES, KEF, VJ height, vertical jump power, and percentage of MVC for EMG activity for the vastus lateralis and the long head of the biceps femoris during knee extension tests were determined from paired *t* tests. Probability was set at $p \leq .05$. Effect sizes were also calculated using Cohen's *d* for each dependent variable. All analyses were executed using Statistical Package for Social Sciences (SPSS 13.0).

CHAPTER IV

RESULTS

A total of 18 participants volunteered for the study. Two dropped out due to scheduling issues. Thus, 16 participants (M = 22.5 years, SD = 49 years) finished all stretching and non-stretching trials for KES, KEF, and VJ. The average height and weight of the study sample was M = 180.3 cm, SD = 10.1 cm and M = 84.9 kg, SD = 19.5 kg, respectively.

Torque

The results for knee extension torque are summarized in table 1. Stretching the antagonist produced a significantly greater torque for the KEF but not the KES. According to Rhea (2004), the effect sizes for both trials were trivial for recreationally trained subjects.

Vertical Jump Height and Power

Both VJ and VJP were significantly higher following the stretching protocol (see Table 1). The effect sizes were trivial (Rhea, 2004) for both variables.

Electromyography

Paired samples t test indicated no significant ($p > .05$) difference between trials for electromyography, represented as a percentage of MVC. The results for all conditions

for the vastus lateralis are summarized in Figure 5.

Table 1

Isokinetic Knee Extension Torque and Vertical Jump

	Trial				
Treatment	Stretch	Non stretch	%Change	*P*	*ES*
KEF (Nm)	102.2 ± 26.8	93.5 ± 33.4	13.9	.032 *	0.26
KES (Nm)	176.7 ± 52.1	162.9 ± 46.3	8.5	.086	0.29
VJ (cm)	59.8 ± 13.3	58.6 ± 13.3	2.0	.011 *	0.09
VJP (W)	8571.7 ± 597	8487.4 ± 615	1.0	.005 *	0.14

Note. All trials represented as Mean ± Standard Deviation. * $p \leq .05$.

The results for all conditions for the biceps femoris are summarized in Figure 6.

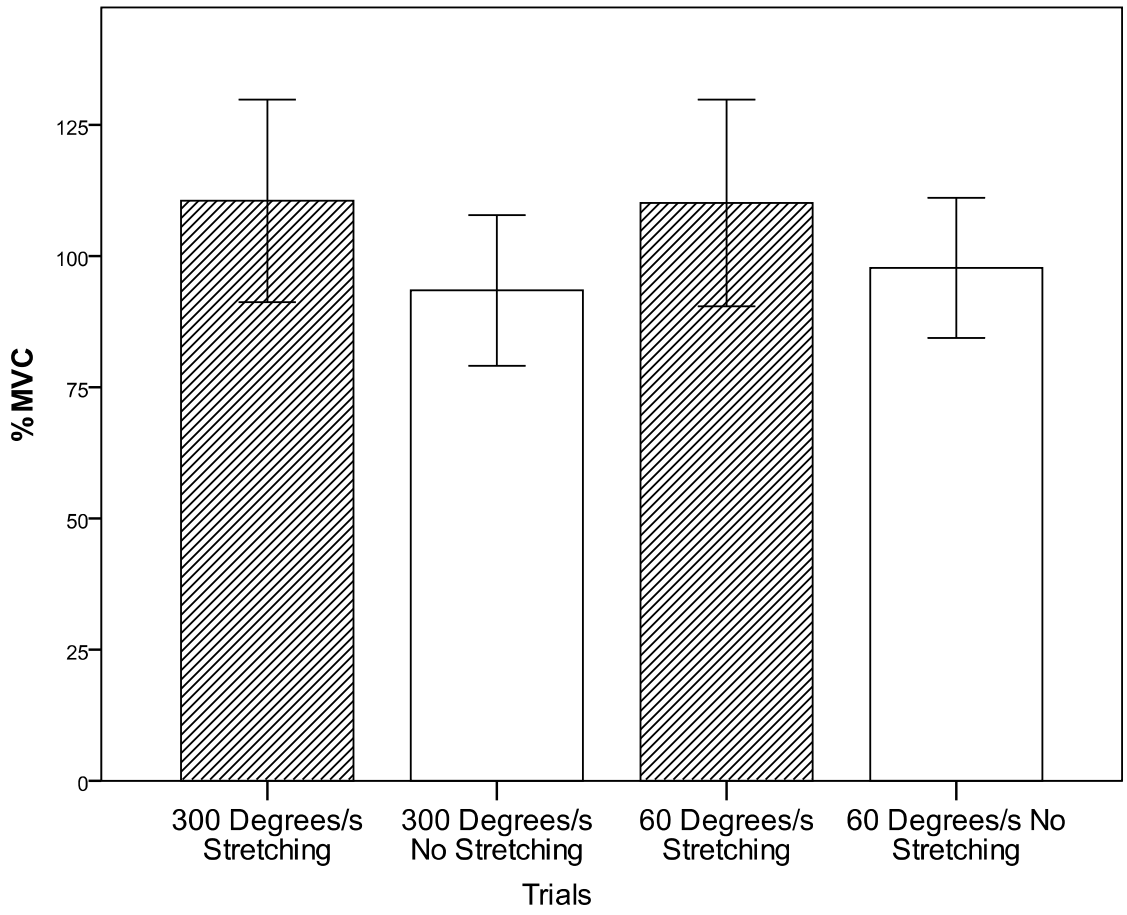

Figure 5. Mean percentage of MVC for all conditions for the vastus lateralis.

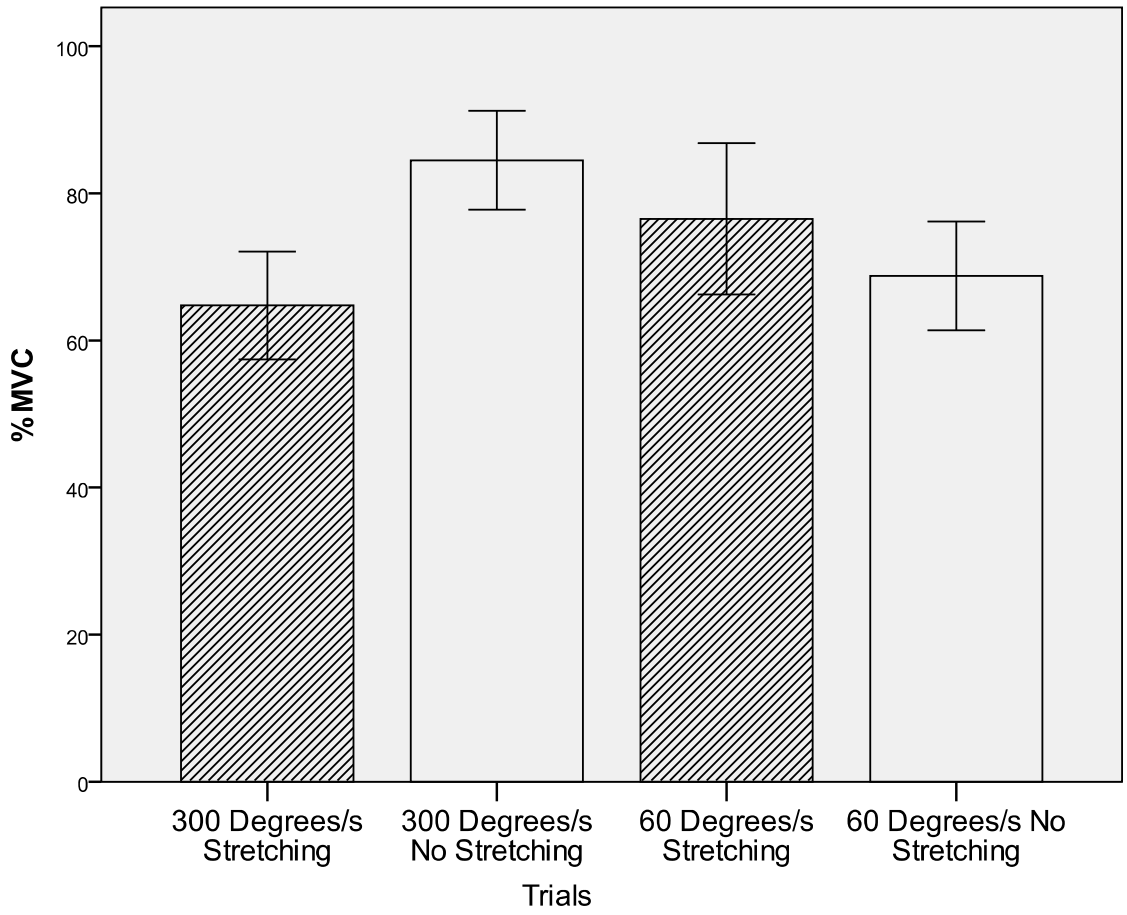

Figure 6. Mean percentage of MVC for all conditions for the biceps femoris.

CHAPTER V

DISCUSSION

Torque Production

The first key finding from the current study was that static stretching of the antagonist (i.e. hamstrings) significantly ($p < .05$) increased torque production for an isokinetic knee extension at 300˚/s. Antagonist stretching did not however produce a statistically significant enhancement in isokinetic knee extension torque at 60˚/s. These results indicate that the effects of antagonist stretching on torque production may be velocity specific.

Although no known research has investigated the effects of antagonist stretching on strength, a velocity specific effect has been found with agonist stretching. Nelson, Guillory,Cornwell, and Kokkonen (2001) found an opposite and negative effect for agonist stretching on peak knee extension torque. The quadriceps were stretched statically with 3 exercises prior to isokinetic knee extension tests. Each exercise was performed four times and each stretch was held for 30 s. Five different velocities were tested. Only the two slowest velocities (1.05 Rad/s and 1.57 Rad/s) showed a significant ($p < .05$) reduction in knee extension torque. At 1.05 Rad/s torque was reduced from M = 218 Nm, SD = 47 Nm to M = 199 Nm, SD = 49 Nm following agonist stretching. At 1.57 Rad/s torque was reduced for M = 204 Nm, SD = 48 Nm to M = 195 Nm, SD = 47 Nm. These changes are of similar magnitude to the current study. Torque decreased 7.2% and 4.5% at 1.05 Rad/s and 1.57 Rad/s, respectively, compared to the current study which had

a 12.2% increase in torque with antagonist stretching at 300˚/s. In the current study antagonist stretching exhibited the opposite effect to agonist stretching in the Nelson et al. (2001) study.

Siatras, Mittas, Mameletzi, and Vamvakoudis (2008) however, did not find a significant velocity specific effect for agonist stretching of the quadriceps. They investigated the effects of stretching duration on peak isokinetic knee extension torque at 60˚/s and 180˚/s. They looked at the effects of both 60 s and 30 s bouts of stretching. There were statistically significant ($p < .05$) decreases in torque for all agonist static stretching treatments. Peak knee extension torque was decreased by 5.5% and 11.6% at 60˚/s for 30 and 60 s, of stretching, respectively. At 180˚/s peak knee extension torque was reduced 5.8% and 10% for 30 and 60 s, respectively. According to this study agonist static stretching reduced torque production, independent of contraction velocity.

Other research investigating the effects of agonist stretching have also demonstrated reductions in torque production. Herda et al. (2008) explored the effects of acute static stretching of the hamstrings on isometric peak knee flexion torque and EMG and MMG activity of the biceps femoris. Torque decreased 15.94% at 101˚ and 7.2% at 81˚ compared to a 12.2% increase for KEF and an 8% increase for KES in the current study. Kokkonen et al. (1998) investigated the effects of agonists static stretching for the quadriceps and the hamstrings on one repetition maximal strength (1RM) using a variable resistance machine. They found a 7.3% decrease in 1RM for knee flexion and 8.1% decrease for knee extension.

Vertical Jump

The second key finding from the current study was that prior stretching of the hip flexors and dorsi flexors, the antagonists of the hip extensors and plantar flexors, significantly (p = .011) enhanced jump height and absolute jump power (p = .05). Church et al. (2001) examined the effects of performing proprioceptive neuromuscular facilitation (PNF) stretching for the hamstrings and quadriceps prior to performing a vertical jump. Participants performed a standard warm up with and without stretching on two different occasions. They found a significant (p = .01) decrease in vertical jump height. PNF stretching of the hamstrings and quadriceps resulted in a 1.47 cm decrease in jump height. In the current study static stretching of the hip flexors and dorsi flexors resulted in a 1.2 cm increase in jump height. Robbins and Scheuermann (2008) investigated the effects of 3 different volumes of static agonist stretching on vertical jump height. Ten collegiate athletes and 10 recreational athletes performed a VJ with and without 2 sets of 15 s, 4 sets of 15 s, or 6 sets of 15 s stretches for the hamstrings, plantar flexors, and quadriceps. The 6 sets of 15 s stretch group saw a significant ($p \leq .05$) decrease in jump height between the stretch (M = 58.199, SD = 2.23 cm) and non-stretch (M = 60.117, SD = 2.268 cm) condition. This is comparable and opposite to the increase in VJ following stretching of the hip flexors and dorsi flexors (M = 59.8cm, SD = 13.3 cm), compared with VJ performed with no stretching (M = 58.6, SD = 13.3 cm) in the current study. Cornwell et al. (2002) also found a significant ($p \leq .05$) decrease in VJ height following acute static stretching of the plantar flexors. They found a decrease of 7.4% compared to a 2.9%

increase in VJ in the current study.

Electromyography Implications

Two hypotheses have been proposed for agonist stretch induced force deficits (Bigland-Ritchie et al., 1992; Caldwell, 1995; Cornwell et al., 2002; Fowles et al., 2000; Herda et al., 2008; Knudson et al., 2001; Vujnovich, & Dawson, 1994; Walshe & Wilson, 1997; Walshe et al., 1996). One proposed mechanism for decreased strength and power following agonist, pre-activity, static stretching is mechanical adaptations, namely a reduction in stiffness and increase in length between resting sarcomeres that alters the length-tension relationship of the muscle. The second proposed mechanism is neural factors such as decreased recruitment and/or reflex sensitivity.

It was hypothesized in the current study that stretching the antagonist musculature would result in increased performance. This would occur by increasing the neural drive to the agonist, decreasing neural drive to the antagonist, reducing stiffness of the antagonist, thereby reducing braking forces to the agonist, or a combination of these factors. The current study found the KEF stretch condition to produced significantly ($p < .05$) more torque than the KEF non-stretch condition. A third key finding from the current study was that the EMG activity of the vastus lateralis for the KEF stretch condition was 9.7% higher than the KEF non-stretch condition; however, this did not reach statistical significance ($p > .05$). This would indicate that the difference in torque observed was not related to increased activation of the prime movers. The EMG activity of the antagonist biceps femoris was 16% lower in the KEF stretch vs. the KEF non-

stretch; again, however, this did not result in a statistically significant difference.

Herda et al. (2008) found no change in EMG following agonist static stretching. They hypothesized that decrements in force following stretching were related to mechanical factors. Cornwell et al. (2002) found a decrease in EMG activity and stiffness following agonist static stretching. However, these authors hypothesized that reductions in stiffness were insufficient to cause a decrease in force production. Fowles et al. (2000) found EMG activity was significantly decreased for the first 15 min following static stretching, and force decrements were greatest during this time. Electrical activity did, however, return to normal after 15 min while force decrements remained for 60 min. These authors theorized that neural factors played a bigger role in strength decreases early, but as time passed, the reduction in MVCs originated peripherally in the muscle. It is possible that improvements in knee extension torque following antagonist stretching at 300°/s in the current study was a result of a mechanically mediated response. If the length tension relationship of the hamstrings was disrupted, this could lead to a reduction in braking forces, which would allow the quadriceps to produce more torque. This is speculation because no measure of mechanical adaptation was taken.

Despite lack of statistically significant differences in biceps femoris EMG activity between KEF stretch and KEF non-stretch conditions, stretching the hamstrings did result in a moderate effect size ($d = 0.55$) for the reduction in biceps femoris EMG activity (Cohen, 1988). This indicates that stretching the hamstrings prior to KEF may have reduced electrical activity to these muscles. Decreased electrical activity to the antagonist biceps femoris could also lead to reduced braking forces and greater torque

production in the quadriceps. It should be noted however that Rhea (2004) proposed a different effect size scale for strength training research, which would classify 0.55 as a small effect versus Cohen's (1988) classification of 0.55 as a moderate effect size.

With each measured variable there was a large amount of subject to subject variability as shown by the relatively large standard deviations (Table 1). There appeared to be a very individual strength and power response among participants to antagonist stretching. This may have been due to initial levels of flexibility. One of the limitations of this study is that there was no initial flexibility assessment taken. There is evidence that tight or short antagonist musculature may result in decreased function of the agonist musculature (Sahrmann, 2002; White & Sahrmann, 1994; Winter el al., 2004). It is possible that individuals with lower initial levels of flexibility in antagonist musculature experienced a greater training effect with stretching than those with higher initial levels. To the investigator's knowledge no studies have investigated whether initial levels of flexibility affect the magnitude of treatment effect with agonist stretching. Future research should investigate if initial levels of flexibility affect the magnitude of treatment effect in antagonist stretching.

Practical Applications

It appears that antagonist stretching may improve strength at high velocities. Antagonist stretching of the hamstrings resulted in significantly greater torque during a high velocity, isokinetic knee extension (300˚/s). Stretching the antagonist to the hip extensors and plantar flexors during jumping resulted in significantly higher jump height

and absolute jump power. Jumping represents a high velocity movement.

The mechanisms for these improvements cannot yet be determined. There were no significant differences in EMG activity in the agonist vastus lateralis or the antagonist biceps femoris for any variable. There was however, a moderate effect size ($d = 0.55$) for reduced EMG activity of the biceps femoris during the KEF stretch condition.

Antagonist stretching may provide a method for acute enhancement for high velocity activities such as jumping. Strength and conditioning practitioners could use this information to implement antagonist stretching during training and competition to increase performance and/or overload of the neuromuscular system. Because responses to antagonist stretching appear to be very individual, practitioners should experiment with individual responses before applying this technique during competition.

Future research should investigate other muscle groups and movement patterns. It should also be determined if initial levels of flexibility affect responses to antagonist stretching prior to strength and power related performance. The effects of methods of antagonist stretching other than static (e.g., PNF, dynamic) should be investigated. Gender effects to antagonist stretching should also be investigated. Future research should also attempt to determine possible mechanisms, whether mechanical, neural, or a combination of both.

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APPENDICES

Appendix A.

Health Status Form

Name: _____

Age:

Read each question and circle yes or no. If you have any questions ask the investigator for clarification.

Do you have any cardiovascular disorder or disease (cardiovascular disease, coronary artery disease, etc.)?

Yes No

If yes explain

Do you have any current muscle injuries (strains, pulls, tears, etc.)?

Yes No

If yes explain

Do you have any current joint injuries (sprains, tears, etc.)?

Yes No

If yes explain

Do you have any current bone injuries (fractures, etc.)?

Yes No

If yes explain

Do you have any metabolic disorders or disease (diabetes, etc.)?

Yes No

If yes explain

Appendix B.

v6 2/3/2010

USU IRB Approval: 09/29./2010; Page 1 of 3
Approval Terminates: 09/28/2011
Protocol #2745
IRB Password Protected per IRB Administrator

Utah State University
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Full AAHRPP Accreditation

INFORMED CONSENT

Acute Effects of Antagonist Stretching on Jump Height, Agonist Peak Knee Extension Torque, and Electromyography of the Biceps Femoris and Vastus Lateralis Muscles

Introduction/ Purpose Professor Dale Wagner and John Sandberg, a graduate student in the Health, Physical Education, and Recreation Department at Utah State University are doing research to investigate the effects of stretching on jumping ability and strength in the knee joint. They will also be measuring the electrical activity of two leg muscles in the thigh to better understand how stretching influences muscle strength. There will be approximately 15 participants in this study.

Procedures If you agree to participate in this study, you will be expected to perform one strength training task and one jumping test on two different occasions. On one occasion these tasks will be performed without stretching, and the other trial will include stretches prior to the tasks. The order of the testing sessions (stretch or no-stretch) will be randomly determined. During stretching trials you will stretch the muscles of the hamstrings on the back of the leg, the hip flexors on the front of the hip, and the front of the lower leg. Each stretch will be held for 30 seconds and repeated 3 times. You will be asked to perform a vertical jump test. You will jump as high as possible, and will be asked to take several jumps in order to improve your score. On a separate day you will also be asked to perform 5 maximal knee extensions at two different speeds. During the knee extensions two electrodes will be placed on the front and back of your thigh. A small amount of hair may be removed from your legs and some marks from an indelible marker drawn on your thigh to allow for proper placement of the electrodes. All testing will take place in the Utah State University HPER building under the direction of the researcher. You will be required to come to the testing site on 5 separate occasions [once to familiarize yourself with the testing equipment, twice for the leg extension test (one session with stretching and one without), and twice for the vertical jump test (one session with stretching and one without)]. Testing will take place over approximately 10 days, and each session will take between 10 and 50 minutes. You may engage in normal daily activities but will be asked to refrain from any other strength training or stretching activities during this time. The following is an example of your time commitment:

1. Familiarization and practice with the isokinetic strength testing machine: 10 min.
2. Electrical activity of the leg muscles and maximal leg extension exercise (with or without stretching; trial 1): 50 min
3. Electrical activity of the leg muscles and maximal leg extension exercise (with or without stretching; trial 2): 50 min
4. Vertical jump test (with or without stretching; trial 1): 20 min
5. Vertical jump test (with or without stretching; trial 2): 20 min

Risks You will be asked to make short, explosive maximal efforts for lifting and jumping. Although the anticipated risk is small, there is some risk involved with this type of exercise. The most common effects of this type of testing are muscle aches and stiffness normally associated with strenuous exercise. More serious musculoskeletal injuries are possible, such as muscle strains or pulls. Every effort will be made

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Department of HPER
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Logan UT 84322-7000
Telephone: (435) 797-1497

INFORMED CONSENT

Acute Effects of Antagonist Stretching on Jump Height, Agonist Peak Knee Extension Torque, and Electromyography of the Biceps Femoris and Vastus Lateralis Muscles

to avoid injury through proper warm-up, technique instruction, and progression. You will be wearing electrodes (EMG); however, these electrodes do not emit electricity rather they are measuring the electrical activity that is present in your body during the exercise.

Benefits There may or may not be any direct benefit to you by participating in this research. However, this research may help you to better understand your initial levels of lower body strength. The study will also allow you to assess your current vertical jump, a measure of power important to sports movements and activities of daily living.

Explanation & offer to answer questions John Sandberg has explained this research study to you and answered your questions. If you have other questions related to this study, you may contact him at 847-454-5579 or email Johnsandberg03@hotmail.com. You may also contact Professor Dale Wagner at 435-797-8253 or email dale.wagner@usu.edu.

Payment/Compensation There is no cost to you for participating in this study. You will not be paid for participating in this study.

Voluntary nature of participation and right to withdraw without consequence You do not have to participate in the research project. Your participation is entirely voluntary. You may stop your participation at any time and any results obtained from your testing will be given and explained to you. Data collected prior to your withdrawal may still be used in the data analysis unless you request it not be used.

Confidentiality Your results will be kept in a locked file cabinet in a locked room at the HPER. Data will be coded so your name will not appear on any of the records with your personal information to protect your privacy. The code sheet will be destroyed after the data are compiled. Only the researchers will have access to the data.

IRB Approval Statement Utah State University (USU) has an Institutional Review Board (IRB) that is charged in making sure the studies performed at USU are worthwhile and safe. The IRB at USU has approved this study. If you have questions about your rights, concerns about how the research is being conducted, or have a research-related injury, you may contact the IRB administrator at 435-797-0567 or email irb@usu.edu.

Copy of consent You have been given 2 copies of this Informed Consent. Please make sure to sign both copies. One copy goes to the investigator, and you should keep the other copy.

v6 2/3/2010

USU IRB Approval: 09/29./2010; Page 3 of 3
Approval Terminates: 09/28/2011
Protocol #2745
IRB Password Protected per IRB Administrator

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Investigator Statement "I certify that the research study has been explained to the individual, by me or my research staff, and that the individual understands the nature and purpose, the possible risks and benefits associated with taking part in this research study. Any questions that have been raised have been answered."

Dale Wagner, Ph.D.,
Principal Investigator
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dale.wagner@usu.edu

John Sandberg
Student Researcher
847-454-5579
johnsandberg03@hotmail.com

Signature of Participant By signing below, I agree to participate.

Participant's signature _____ Date _____