THE EFFECTS OF HAMSTRING DELAYED ONSET MUSCLE SORENESS ON FUNCTIONAL KNEE JOINT STABILITY

By

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A THESIS PRESENTED TO THE GRADUATE SCHOOL
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Dynamic joint stability is an essential component of athletic performance. If deficits in dynamic stability exist, the athlete may be unable to perform at previous levels of competition and may also be prone to injury. Delayed onset muscle soreness (DOMS) is a response to unaccustomed bouts of strenuous exercise that results in certain physical and physiological markers being present in the local tissue. The purpose of this investigation was to determine the effects of hamstring DOMS on functional knee joint stability.

Thirty subjects (18 females, 12 males) with no previous knee injuries participated in this investigation. Subjects were assigned to one of two groups (DOMS, Control). Baseline measures of hamstring flexibility, pressure pain threshold (PPT), passive range of motion pain threshold (PROMPT), hamstring latency, and time to stabilization (TTS) were measured. Subjects in the experimental group then performed 6 sets of 10 eccentric

hamstring contractions using a prone leg curl machine. All subjects returned for reevaluation of all baseline measures 48 and 96 hours following the initial session.

Separate analyses of variance were performed for each dependent variable. Tukey HSD post hoc analyses were performed to determine where significance existed among the levels of each factor. A probability level of $P < 0.05$ was expected to designate statistical significance.

DOMS was adequately induced in the experimental group. Active hamstring range of motion in the DOMS subjects significantly decreased after 48 and 96 hours post exercise. PROMPT and PPT increased significantly 48 and 96 hours after the initial session for the DOMS group. Hamstring muscle latency in the medial hamstring had a slower response time to the external rotation perturbation. A significantly faster response time was also present at the 48h and 96h posttest sessions compared to the baseline measures. Time to stabilization for the vertical ground reaction force curve was faster during the 96h posttest session compared to the pretest and posttest 48h sessions, respectively.

Based on the results of the present study, it was concluded that DOMS has little to no effect on the functional outcome of dynamic joint stability as measured using a standing rotational perturbation or a jump landing procedure. Future research should investigate the effects of hamstring DOMS on other aspects of proprioception, including active and passive joint repositioning and also threshold to detection of passive movement. Additionally, research should be conducted to examine the effects of a functional fatigue protocol on the entire lower extremity to establish whether or not functional joint stability would be affected if the entire kinetic chain were involved.

CHAPTER 1
INTRODUCTION

Knee injuries, particularly those involving the anterior cruciate ligament (ACL), are a common occurrence in athletics. The ACL is the primary stabilizing ligament of the knee and athletes who participate in sports involving high-speed change of direction, pivoting, jumping and landing, and deceleration maneuvers are at an increased risk for ACL injury. Damage to the ACL results in decreased stability of the knee joint and is typically associated with a feeling of "giving way". The prevalence of injury in sports such as football, basketball, and soccer are relatively high because they involve some or all of the predisposing components stated above.[1] In many cases the injury occurs when there is no apparent contact involved. These non-contact injuries account for approximately 70% of all ACL injuries.[1, 2]

The incidence of ACL injury in sports is a continuing dilemma. In 2002, during three weeks of NCAA spring practices and one weekend of NFL mini camps, six players were diagnosed with having suffered season ending knee injuries, all involving the anterior cruciate ligament.[3, 4, 5] Of those six cases, four of the injuries involved no contact with an opposing player, which is in close agreement with the percentages reported earlier for non-contact ACL injuries. Because contact injuries cannot be controlled, non-contact incidences such as these have researchers probing for answers in an attempt to recognize predisposing factors related to ACL injury.

The hamstrings help to dynamically stabilize the knee during athletic movements that may predispose an athlete to ligament injury. DOMS is characterized by pain,

decreased strength, decreased range of motion, and swelling of the affected muscles. All of these characteristics can lead to altered responses from the hamstrings, whether it is a decrease in force generation or an increase in muscle reaction time. With a diminished response from these dynamic restraints, functional knee stability could be compromised, resulting in ACL disruption.

Statement of the Problem

Functional joint stability is a collection of anatomical and physiological components that are present in order to maintain a relatively homeostatic environment of the joint during active bodily movements.[6] These components are characterized as either static or dynamic in nature. Static components represent the structural aspect of the joint: ligament, capsule, cartilage, bony congruity, and friction, whereas the dynamic component corresponds to the functional aspect, primarily made up of the muscles that cross the joint. Biological control systems associated with dynamic stabilization are the feedforward or anticipatory and feedback or reactive mechanisms. Impulses from afferent stimuli through somatosensory, visual, and vestibular input allow these control mechanisms to function properly.[6, 7] The muscle's reactive response to a perturbation plays a major role in dynamic joint stability. The timing of this response is commonly referred to as muscle latency.

The hamstrings work synergistically with the ACL as stabilizers of the knee joint, providing resistance to anterior shear forces as well as rotational forces around the knee. The ACL provides a passive resistance, while the hamstrings actively stabilize the knee. An increased latency period of the hamstrings could increase the likelihood of a functionally unstable joint. This could further predispose an athlete to potential ligament injury due to the inhibition of the dynamic components of the sensorimotor system.

DOMS is a physiological response to unaccustomed physical activity that results in pain, decreased strength, decreased range of motion, and swelling of the affected muscles. There is limited research indicating an increase in muscle latency associated with muscular fatigue[8] and decreased strength,[9] with none having directly studied DOMS to determine if similar effects would be noted. Therefore, the purpose of this study was to assess functional knee joint stability after DOMS has been induced in the hamstring muscles. Functional joint stability will be assessed by measuring time to stabilization of the affected lower extremity and muscle latency of the hamstrings.

Research Hypotheses

1. Active hamstring range of motion will significantly decrease after the inducement of DOMS.
2. Passive range of motion pain threshold will significantly increase after the inducement of DOMS.
3. Pressure pain threshold measurement will significantly increase after the inducement of DOMS.
4. Hamstring muscle latency following knee perturbation will significantly increase after the inducement of DOMS.
5. Time to stabilization following a jump landing will significantly increase after the inducement of DOMS.

Definition of Terms

1. **Eccentric contraction** is the lengthening of a muscle when a force/load applied to that muscle is greater than the force production of the muscle.
2. **Electromyography** is the study of muscle function through the detection of electrical impulses emanating from the muscle itself.
3. **Goniometry** is a standardized technique of measuring joint motion.
4. **Homeostasis** is the maintenance of a stable internal environment of the body.
5. **Kinesthesia** is the ability to perceive extent, direction, or weight of movement.

6. **Muscle latency** is the time required for a muscle to respond following an induced perturbation.

7. **Perturbation** is a disruption in homeostasis.

8. **Proprioception** is the awareness of posture, movement, and changes in equilibrium and the knowledge of position, weight, and resistance of objects in relation to the body.

9. **Range of motion (ROM)** is the amount of movement of a particular joint measured in degrees by goniometry.

10. **Time to Stabilization (TTS)** is a measurement tool, calculated in milliseconds, used to assess the length of time needed for one to maintain dynamic stability following a standardized jump for distance and height to a single leg balance position.

Assumptions

Two assumptions were made for the purpose of this investigation

1. Subjects will be honest in filling out prescreening questionnaire.

2. Subjects will exert full effort when reacting to knee perturbations and performing time to stabilization techniques.

Limitations

Five limitations were identified for this investigation

1. A sufficient predetermined level of DOMS must be induced in the hamstring muscles; however, the level will vary across subjects.

2. Only hamstring flexibility and muscle soreness will be used as indicators of DOMS.

3. Only subjects who have not performed lower extremity resistance training during the previous six months will be used in the investigation.

4. Functional knee stability will only be assessed 48 and 96 hours after DOMS has been induced.

5. Only healthy subjects free from any acute knee injuries or lower extremity muscle strains will be used for this study.

Significance

Injuries to the anterior cruciate ligament continue to be a primary concern to all members of the sports medicine team. The exact cause of ligament failure is unknown, but many assumptions can be made. No study to date has assessed hamstring latency when DOMS has been induced. Determining whether DOMS affects the latency period of the hamstrings could identify a predisposing risk factor to ACL failure in athletics.

CHAPTER 2
REVIEW OF LITERATURE

Introduction

Dynamic joint stability is an essential component of athletic performance. This type of stability also helps to protect the joint from injury. If deficits in dynamic stability exist, the athlete may be unable to perform at previous levels of competition and may also be prone to injury. Likewise, athletes who have been previously injured may develop deficits in dynamic stability. The microtrauma that occurs during resistance and other types of exercise, generally referred to as DOMS, induces changes in the local tissue similar to those seen following macrotrauma. Thus, it is possible that this type of trauma would affect dynamic stability. However, at this time the effects of DOMS on dynamic stability have not been investigated. This review of literature will focus on the anatomy and biomechanics of the knee, mechanism of injury for the anterior cruciate ligament, delayed onset muscle soreness, and measures of dynamic stability.

Anatomy and Biomechanics of the Knee

Articulations

The knee joint is composed of two bony articulations, the tibio-femoral joint and the patellofemoral joint. The tibio-femoral (knee) joint, comprised of the femur and the weight-bearing tibia, is a modified hinge synovial joint that allows a great amount of ROM through movements of flexion and extension, but is limited with internal and external rotation.[10] The patellofemoral joint is made up of the patella and the femur. The patella, a large sesmoid type bone, is embedded within the patellar tendon and rests in the

trochlear groove of the femur.[11] The patella's primary function is as a fulcrum for the quadriceps muscles to increase the lever arm during active extension movements at the knee.[12]

Ligaments

Configured in and around the knee are four key ligaments that enable the joint to maintain passive stability during weight bearing activities. These ligaments can be divided into two groups, collaterals and cruciates, whose function is based on their arrangement at the knee. The collaterals are comprised of the lateral collateral ligament (LCL) and the medial collateral ligament (MCL), whereas the cruciates consist of the anterior cruciate ligament (ACL) and the posterior cruciate ligament (PCL). The orientation and biomechanics of the cruciate ligaments will be provided to allow a better understanding of their role in knee joint stability.

The ACL is an important stabilizing ligament in the knee joint and one that, if ruptured, typically requires surgery.[13] The ACL functions to reduce anterior shear force as well as control some varus, valgus and rotational forces of the tibia on the femur. Approximately 86% of the primary restraint of anterior tibial translation is provided by the ACL.[14] Damage to the ACL creates joint laxity and may lead to episodes of instability in the knee.[15] The ACL originates on the medial aspect of the lateral femoral condyle and inserts on the anterior tibial plateau.[16] Generally, the ACL is addressed as a single banded ligament that connects the tibia to the femur; however, that is not entirely the case. The ACL is divided into two separate bundles of fibers, an anteromedial and a posterolateral bundle, each offering passive resistance to different stresses placed on the tibio-femoral joint. When the knee is in a fully extended position (zero degrees), the anteromedial bundle is most taut, while the posterolateral bundle is taut in flexion.[16]

Movements through the range of motion are accommodated by a combination of the two bundles working synchronously.

The posterior cruciate ligament serves as the antagonist to the ACL, in that its orientation in the knee runs from the lateral wall of the medial femoral condyle to the posterior tibial shelf.[17, 18] The PCL serves to prevent straight posterior displacement of the tibia relative to the femur.[19, 20]

The PCL, like the ACL, consists of two so-called bands of fibers. During knee flexion movements, the anterolateral portion is tightened while the posteromedial portion is lax. Conversely, in knee extension, the anterolateral bundle is lax, whereas the posteromedial bundle is tight.[17, 18]

Muscles

An extensive knowledge of the musculature that crosses the knee joint is critical in order to understand the proper structure and function of the knee joint and how this relates to overall functional joint stability. There are 12 muscles that cross the knee joint. The primary muscles associated with knee joint movement are the four quadriceps and three hamstring muscles. The remaining muscles, which consist of the gastrocnemius, plantaris, and popliteus in the lower leg, and the sartorius and gracilis of the upper leg, are secondary type muscles that assist movement of the joint, but are not considered prime movers.

The quadriceps group is collectively made up of four muscles (Rectus Femoris, Vastus Intermedius, Vastus Lateralis, and Vastus Medialis) that act to extend the knee. Because the Rectus Femoris attaches on the pelvis, this allows it to also function as a hip flexor. The Vastus Medialis and Lateralis originate on the linea aspera on the posterior femur on their respective sides, and the Vastus Intermedius originates on the anterior and

lateral femoral shaft. All four muscles have a common insertion site into the tibial tuberosity via the patellar tendon.[21]

The hamstrings are the primary muscle group that makes up the posterior thigh, which consists of three separate muscles (Biceps Femoris, Semitendinosus, Semimembranosus) that's main functions are to flex the knee and extend the hip. The hamstrings have a common origin on the ischial tuberosity of the pelvis, though the biceps femoris has an additional origin on the linea aspera of the posterior femur. As a group, they travel down the posterior aspect of the upper leg with individually different insertions. The biceps femoris, semimembranosus, and semitendinosus insert into the head of the fibula, posterior medial tibial condyle, and anterior proximal tibial shaft, respectively.[21]

Besides providing joint motion, the hamstring muscles also function as joint stabilizers and secondary restraints to anterior tibial translation.[22,23] Some research suggests that there is an anterior cruciate ligament – hamstring reflex that allows this to happen. However, there has been much controversy over this neuromuscular response. In human and animal studies, researchers have investigated the relationship between anterior tibial displacement and the ACL – hamstring reflex with mixed results.[1, 13, 15, 24, 25, 26] Boden et al.[1] suggest that the hamstrings provide this protective effect when the hip is flexed because it allows the hamstrings to become tighter, allowing less anterior shear of the knee. This protective effect may be diminished with increased hamstring flexibility.[1] Their findings suggested that athletes who sustained an ACL injury had greater hamstring flexibility than a control group of subjects.

Mechanoreceptors

One of the most important components of motor control is our ability to utilize the numerous mechanoreceptors found throughout the human body. The importance lies in that mechanoreceptors provide afferent input to the central nervous system making them valuable contributors to proprioceptive feedback. The most commonly described mechanoreceptors, muscle spindles and Golgi tendon organs (GTO), are located in muscle and tendon tissue.[27, 28] Others that are equally important are those located in the ligaments and joint capsules, particularly Ruffini endings and Pacinian corpuscles.[6, 28] Muscle spindles are located in skeletal muscle and are responsible for detecting changes in length of the associated muscle, whereas Golgi tendon organs monitor active force and tension in the muscle through its location in the myotendinous junction. These two mechanoreceptors function via a monosynaptic reflex pathway and are considered to have a slow adaptation rate to their respective responses.[6, 7, 28] The Ruffini endings and Pacinian corpuscles respond through the same reflex pathway as mentioned earlier. Like the muscle spindles and Golgi tendon organs, Ruffini endings are slow adaptive and have a low threshold for stimulation. They are primarily located in the joint capsule and ligaments and are responsible for detecting changes in joint pressure. Pacinian corpuscles are also low threshold receptors, but adapt quickly to the stresses placed upon them. They function to sense high frequency vibrations within the joint capsule. This quick adaptive response classifies them as only dynamic receptors, meaning they are inactive when a constant stimulus is placed on the joint.[6, 7, 28] The mechanoreceptors described here have independent responsibilities for maintaining dynamic joint stability. However, in order for the joint to function properly, they must work in unison, dependent on each other for maximum proprioceptive feedback.

Mechanism of Injury for ACL Rupture

There are typically two types of mechanisms associated with ACL injuries, contact and non-contact. Contact injuries are those that take place when a direct blow is made to either side of the knee or to the anterior aspect of the knee, forcing it into a varus, valgus, or hyperextended position, respectively. There are numerous non-contact mechanisms that result in injury to the ACL, but the majority occurring in sports participation is while the athlete's foot is fixed or planted with either a rotational or varus/valgus force placed on the knee, such as cutting, planting, or pivoting to change direction.[8, 29] Injuries are also prevalent in athletic maneuvers that involve a sudden deceleration during or just before a change of direction or landing awkwardly from a jump.[1, 2, 23, 29] Research has provided evidence that non-contact ACL injuries are more common than contact mechanisms, accounting for approximately 70– 78% of all ACL injuries.[1, 2, 22, 23, 30]

Delayed Onset Muscle Soreness (DOMS)

When healthy people take part in unaccustomed bouts of strenuous exercise, the phenomenon of DOMS typically follows. Delayed onset muscle soreness is commonly noticed after exercise that is relatively intense, of long duration, and/or eccentric in nature. The onset of symptoms begins approximately 8 – 24 hours post exercise with intensity peaking at 24 – 48 hours,[31, 32] and symptoms lasting up to 7 days. Common physical and physiological markers associated with DOMS are soreness and pain,[31, 33 – 37] decreased strength,[33-37] increased plasma creatine kinase (CK) levels,[34 – 39] decreased range of motion,[34, 35, 40] and swelling.[31, 34, 35] Most recently, Nosaka et al.[35] noted a decrease in mean maximal isometric force after one bout of eccentric exercise of the biceps brachii muscle. Range of motion decreased immediately post to 3 days post

exercise. Along with range of motion decrease, there was a significant increase in circumference of the upper arm immediately post exercise, followed by a further increase in swelling at 2 days post exercise. Muscle soreness developed 1 day after exercise and peaked at 2-3 days after, then gradually attenuated. A significant increase was noted in CK after a single bout of exercise, peaking at 3-5 days post exercise.[35]

Exercise training can have a significant effect on the outcome and subsequent events associated with exercise induced muscle damage. It is widely accepted that a second bout of exercise done at the same intensity as the initial bout will provide somewhat of a protective effect on the muscle, where there may be no increases in muscle damage markers and the recovery time may be decreased.[34, 35, 39 – 41] This phenomenon is called the repeated bout effect. The time frame involved in the repeated bout effect is unclear. Some researchers have also investigated training prior to the first bout of eccentric exercise attempting to determine how it affects delayed onset muscle soreness and the markers involved.[36,38] Ebbeling and Clarkson[40] studied the effects of performing a second bout of exercise prior to full muscle recovery. They reported that symptoms were not exacerbated, significantly smaller changes in muscle damage indicators were found, and the recovery time was faster following a second bout of eccentric exercise of the elbow flexor muscles,[40] however, Nosaka and Clarkson[34] concluded that a second and third bout of high force eccentric exercise performed 3 and 6 days following the initial exercise session had neither increased the markers of damage to the tissue nor caused a change in recovery time.[34] When a bout of eccentric exercise was repeated 48 hours after an initial bout, there were no beneficial or detrimental effects on the time-course and/or intensity of DOMS, CK, or 1 repetition max strength.[39] Schwane,

et al.[36] suggested that progressive, short-term training could reduce the effects of delayed onset muscle soreness. They trained subjects with either uphill or downhill treadmill running for 1 and 2 weeks respectively. This was followed by the experimental test, which consisted of 45 minutes of intermittent downhill running at 80%$_{VO2Max}$. The researchers reported that subjects who performed the downhill running training for 2 weeks displayed less DOMS markers than those subjects who trained downhill for 1 week and also showed less DOMS symptoms/markers than the control and uphill training groups.[36] In trained individuals, a smaller metabolic response was reported after performing one bout of high intensity eccentric exercise. Significantly higher CK levels were seen in untrained subjects compared to subjects who took part in a regular training regimen.[38] A training effect can be seen from up to 6 weeks to as long as 6 months, but there has been no effect noticed after 6 months.[35,41]

Electromyography (EMG)

Elecromyography techniques are used to determine the electrical activity associated with muscular contraction and nerve function. It can be used to define the onset and intensity of muscle activation. From this, latency periods in muscle can be determined from EMG readings. Muscle latency is used to assess the time from joint movement to the initial onset of muscle activity.

Co-contraction of the muscles surrounding the knee joint is thought to provide a protective mechanism against ligamentous injury. Colby et al.[22] assessed the activation times of muscular contraction while subjects performed four types of athletic movements (sidestep cutting, cross-cutting, stopping, and landing) typically associated with ACL injuries. They reported that in all trials the quadriceps were activated at higher intensities than the hamstrings leading up to, at foot contact, and at the propulsion phase of the

maneuver the subject was performing.[22] These findings might suggest that because the quadriceps are creating more force, more often that this would produce an undesirable anterior shear force of the tibia resulting in increased stress on the ACL.

Beard et al.[25] and Jennings et al.[24] compared reflex hamstring contraction latency in ACL deficient knees to normal knees using a specially designed weight-bearing apparatus. Patients were positioned standing inside the rig with their thigh to be tested resting against a pad. The placement of the pad did not allow any movement of the femur anteriorly. An accelerometer was then placed on the tibial tuberosity. Finally there was a compressed air piston mechanism pressed against the posterior tibia. The perturbation was initiated when the piston was released and an anterior shear force was applied to the tibia. This study was designed to investigate function and instability of the knee joint.[24,25] Some skepticism arises when function is mentioned with this maneuver, when there is no functional task addressed.

Measures of Dynamic Stability

A more accurate assessment of dynamic joint stability would be to use a maneuver that incorporates a functional task. Recent research has addressed this issue. Colby et al.[42] and Ross et al.[43] integrated this for measurement of time to stabilization. Colby and colleagues[42] required subjects to perform a 1-legged step down onto a force plate and a 1-legged hop onto the force plate. The step down measure was from a set height of 19 cm and the hop test was performed at a distance equal to the subject's leg length.[42] The design used by Ross et al.[43] was similar in theory. Subjects were asked to jump from a two-foot stance a distance of 70cm landing on one foot on a force plate. Included in this design was a standardized protocol for measuring jump height needed for the measure to be consistent. Subjects needed to achieve 50% of their maximum vertical jump height

while covering the 70cm distance. Upon landing, subjects had to remain in the single leg stance position for 20 seconds. A majority of areas typically used to assess dynamic joint stability are represented with these procedures.[43]

A more functional way to assess hamstring latency was designed by Schultz et al.[44] The researchers used a weight-bearing perturbation device designed to induce a forward moment with either an internal or external rotation of the trunk and femur relative to the tibia. Similar to the concept used by Colby[22], this device was designed to simulate a typical mechanism associated with ACL injury. Unlike the previous research, Schultz's design allowed the presence of a silent period of the hamstrings before the perturbation was initiated. This maneuver was thus deemed a valid and reliable measure when assessing hamstring latency.

Using time to stabilization and hamstring latency as measures of dynamic stability will allow this research to evaluate DOMS as a potential factor affecting neuromuscular control. Speculation can be made that when the signs and symptoms associated with DOMS are exacerbated, an athlete's neuromuscular control could be diminished, further leading to injury. Lephart et al.[45] have assessed a functional stability paradigm where proprioceptive deficits can lead to decreased neuromuscular control, which in turn can lead to functional instability, finally leading to ligament injury. This paradigm is considered cyclical, meaning that if any of the previously mentioned steps are present, the progression of functional instability will continue.

Athletes with diminished time to stabilization could have difficulty achieving the proper balance needed while performing athletic maneuvers such as landing from a jump. When those muscles are negatively affected by DOMS, this could predispose the athlete

to episodes of instability where the only means for joint protection is the passive restraints of that joint (ligaments, cartilage, joint capsule, etc). Similar consideration can also be taken when assessing hamstring latency. The diminished ability of the muscles to fire at the proper time and rate could lead to instability, resulting in joint injury. Therefore, without adequate input from the dynamic control system, the athlete can become vulnerable to joint damage.

Summary

The exact cause for anterior cruciate ligament injury is unclear. However, a number of predisposing factors are commonly noted in the literature. At this time there is no research to confirm or refute delayed onset muscle soreness as one of those predisposing factors. The microtrauma associated with DOMS presents with specific physiological markers that may affect dynamic joint stability. Strength deficits and an increase in pain perception could cause changes in joint mechanics and muscle firing patterns. A lower force production of the hamstrings during athletic maneuvers could increase the percent of quadriceps to hamstring muscle activation, as noted by Colby et al.,[22] resulting in increased anterior shear forces of the tibia. This would diminish the effects the hamstrings provide in protecting the knee during dynamic movements. This overall weaker hamstring response could reduce functional knee stability, ultimately leading to a predisposition to ligament damage. Thus, determining joint stability using functional tasks is the goal of this research.

CHAPTER 3
METHODS

Subjects

Thirty healthy, college age subjects were recruited from the University of Florida student population. The first twenty subjects participated in both measures, while the final 10 exclusively took part in the TTS procedure. Prior to participation in the study, all subjects read and signed an informed consent form approved by the university institutional review board (Appendix A). Subjects were evaluated for previous knee injuries using a history questionnaire (Appendix B) and were excluded if prior knee injuries were present. Subjects were also excluded if they were suffering from a previous hamstring injury or any other injury or condition that might have affected dynamic stability or balance. Finally, subjects who have performed lower extremity weightlifting exercises within the previous six months were excluded from this study to eliminate a training effect.

Instrumentation

Perturbation Device

The perturbations were performed with a lower extremity perturbation device (LEPD) (Figure 3-1) designed similarly to those used in previous research.[44, 46, 47] The LEPD produces an unexpected forward perturbation with either an internal or external rotation of the trunk and femur in relation to the fixated foot and tibia. Subjects were wearing a waist harness with hooks attached to each side while being restrained at the hips using cables attached to a release mechanism. To standardize the procedures, a load

cell (Transducer Techniques, Inc, Temecula, CA) (Figure 3-2) was attached in the middle of each cable. The height adjustable release mechanism was mounted to the wall and consisted of a .64-cm universal push-to-connect coupling system (Porter-Cable Corporation, Jackson, TN) and a trigger switch to detect when the perturbation was initiated (Figure 3-3).

Electromyography

A 16 channel Myopac EMG system (Run Technologies, Laguna Hills, CA) interfaced with a laptop-type personal computer was used to record raw EMG signals for the onset times of the lower extremity musculature following a weight-bearing perturbation procedure. The specifications for the electromyography unit included an amplifier gain of 1-mV/V, frequency bandwidth of 10 – 1000 HZ, CMRR 110 dB, input resistance of 1 MΩ, and a sampling rate of 2000 Hz. Upon completion of data sampling, an analog to digital conversion of the EMG data was performed and stored on the PC using DATAPAC 2000 (Run Technologies, Laguna Hills, CA) software.

Triaxial Force Platform

Performance of the TTS procedure was assessed using a triaxial force platform (Bertec Corporation, Columbus, OH) (Figure 3-4). The raw signal was acquired at a frequency of 2000 Hz and stored on the same laptop-type computer using the DATAPAC 2000 (Run Technologies, Laguna Hills, CA) software.

Inclinometer

Hamstring flexibility was assessed using an inclinometer (Figure 3-5). The inclinometer resembles a flat goniometer with 360° marked in single degree increments on the circumference. A freely rotating arm fixed at the center of the inclinometer is used to determine the angular position as it aligns with the degree markings on the

circumference. Because the arm can move without restriction, gravity maintains it in the downward position. Thus, during limb movement the arm remains in the downward position, indicating limb position.

Measurements

Hamstring Flexibility

Subjects were positioned supine with their involved hip actively flexed to 90º and the contralateral lower extremity flat on the table (Figure 3-6). A specially designed apparatus made of PVC pipe was used to ensure the subjects' hip remained at this angle through the entire measurement (Figure 3-7). The subject was then instructed to actively extend their knee as far as possible and hold that position for 3 seconds. A Velcro™ strap was used to secure the inclinometer just above the ankle. Three trials were performed and the average ROM obtained from the three trials was used as the measure of hamstring flexibility (Appendix C).

Pain Measurement

Subjects were assessed for the level of perceived pain as pressure and passive stretch were applied separately to the hamstring muscles. Both tests were performed with the subjects in the same supine position used for flexibility testing. To assess the perceived pain during a passive stretch, the knee was passively extended to the end range as the examiner applied 6.0-kg of force. A Nicholas Manual Muscle Tester (MMT) (Model 01160, Lafayette Instrument, Lafayette, IN) (Figure 3-8) was used to control the amount of pressure applied. When 6.0-kg of force had been achieved, the subjects were instructed to mark on a visual analogue pain scale (Appendix D) the amount of pain they felt at that moment. They were asked to make a vertical slash across a 10-cm long line between the limits of no pain felt (left end of line) and unbearable pain (right end of line).

The subjects were then assessed for pressure-pain threshold using an algometer (Ametek, Chatillon, NY) (Figure 3-9). The subjects were again passively stretched to end range while an examiner applied 9-kg of pressure with the algometer directly to the belly of the medial hamstrings. The subjects were again asked to mark a vertical slash representing the amount of perceived pain on a separate visual analogue pain scale. The distance of the mark in millimeters from the left end of the line was used as the measure of perceived pain for each visual analogue scale.

Muscle Latency

Muscle latency following knee perturbation was assessed using EMG. To prepare the subjects for this, the skin overlying the medial and lateral hamstring muscles (MH and LH), and the vastus medialis and lateralis (VM and VL) was shaven and cleaned with isopropyl alcohol to reduce skin impedance. Bipolar 1-mm x 10-mm Ag/AgCl surface electrodes were then placed over the muscles with an interdetection surface distance of 1.5-cm between electrodes. Manual muscle testing was performed to confirm correct electrode placement using real time oscilloscope displays. The waist harness was fitted snugly to the subject and the release mechanism was adjusted to a height level with the subject's anterior superior iliac spine (ASIS) while the subject was standing on the force platform in the flexed knee position. Having the subject focus on the computer screen directly in front of them aided to control visual feedback. The position of the subject was standardized prior to the perturbation using the load cells and a predetermined voltage formula. The voltage formula consisted of a y-intercept equation in which the unknown variable was determined by inserting 5% of the subject's body weight. The product of the formula was then multiplied by 0.10 and this was the final number inserted into the computer. The subjects were instructed to lean into the cables until the voltage reached

the determined level. Three trials of internal and external rotation perturbations were provided for the subjects to become accustomed with the device followed by ten random perturbations (5 IR, 5 ER) while allowing subjects 30s rest time between trials.

The acquired raw signals were digitally processed using a symmetric root mean square (RMS) algorithm with a 10-msec time constant. Muscle latency was measured as the time between the initiation of perturbation and the onset of muscle activity, which was determined by calculating a threshold voltage (V_o) for each muscle. The V_o required for muscle onset was determined when the EMG activity exceeded 30% of the muscles peak amplitude for that trial, which was calculated from the following equation: V_o = Max * 0.30.[48] The onset of muscle activity was determined by comparing discrete data points in a point-by-point fashion to the V_o. The muscle was considered active (or a reflex event to have occurred) when the V_o was exceeded for a minimum of 10-msec. The average MRT of the five trials was calculated and used for statistical comparison.

Time to Stabilization

Subjects were evaluated for TTS in the medial/lateral, anterior/posterior and vertical planes following a single leg landing from a jump height equivalent to 50% of their maximum vertical jump height, which was assessed prior to the TTS measure. Subjects were positioned under the Vertec™ vertical jump device (Figure 3-10) and while standing on their toes, their reach height was determined. Subjects were then asked to jump as high as possible from a stationary stance, touching as many vanes as possible. Maximum vertical jump height was determined based on the number of vanes touched. This procedure was performed a total of three times, with the highest score being used. TTS was then assessed as the subjects jumped from a two-footed stationary stance to a one footed stabilization position onto a force platform 70-cm away (Figure 3-11a-c).

After landing on the platform, subjects were instructed to balance on a single leg for 5-sec. Subjects were allowed three practice trials to become accustomed to the task and then needed to perform this procedure for a total of 3 successful trials. TTS was determined as the time in msec necessary for the sequential average of the data points to fall within 0.25 standard deviations of the mean of the first 3-sec following landing.

Procedures

After successfully meeting the criteria for inclusion into this study, subjects were asked to report to the Athletic Training/Sports Medicine Research Laboratory for measures of hamstring flexibility, pressure-pain threshold (PPT), passive range of motion pain threshold (PROMPT), time to stabilization (TTS), and hamstring muscle latency. Baseline measures of hamstring flexibility for each subject were taken prior to experimentation. Perturbations and stabilization procedures along with the lower extremity to be tested were randomly assigned as to eliminate any potential threats to validity. Following the baseline measures of muscle latency and TTS, subjects were randomly assigned to either an experimental or control group. Subjects assigned to the experimental group performed 6 sets of 10 eccentric contractions of the hamstrings using a prone lying leg curl machine. Subjects were assessed for single leg 1 repetition max strength concentrically, with 100% of that 1RM being used as the exercise intensity for the subject. Subjects were instructed to lower the weight from a fully flexed knee to a fully extended knee. This movement was standardized using a metronome and lasted for 10 seconds and subjects were given a 10 second rest between each repetition. Subjects were given 1.5 minutes to rest between sets. Participants were asked to return for reevaluation of TTS and muscle latency 48 and 96 hours after the initial testing date. Upon return, subjects had their hamstring flexibility reevaluated, as well as each measure

of pain threshold using the visual analog pain scale used to assess the level of DOMS achieved. Hamstring flexibility, pain threshold, TTS, and perturbation procedures were performed identical to the pretest trials.

Statistical Analysis

The design of this study was a pretest – posttest design. Statistical analysis for the DOMS measure was done using separate two-way mixed design analyses of variance for each dependent measure (AROM, PROMPT, PPT). To determine statistical significance for the muscle latency measure, separate three-way mixed design analyses of variance were used with the independent variables consisting of muscle (MH, LH), time (pretest, 48-hr post, 96-hr post), and physiological state of the hamstrings (DOMS induced, control). Three separate two-way analyses of variance with repeated measures on the factors of time and group were used for the TTS procedure (Fz, Mx, My). If statistical significance was noted, a Tukey HSD post hoc analysis was performed to establish where the significance lies. A probability level of $P < .05$ was expected to designate statistical significance.

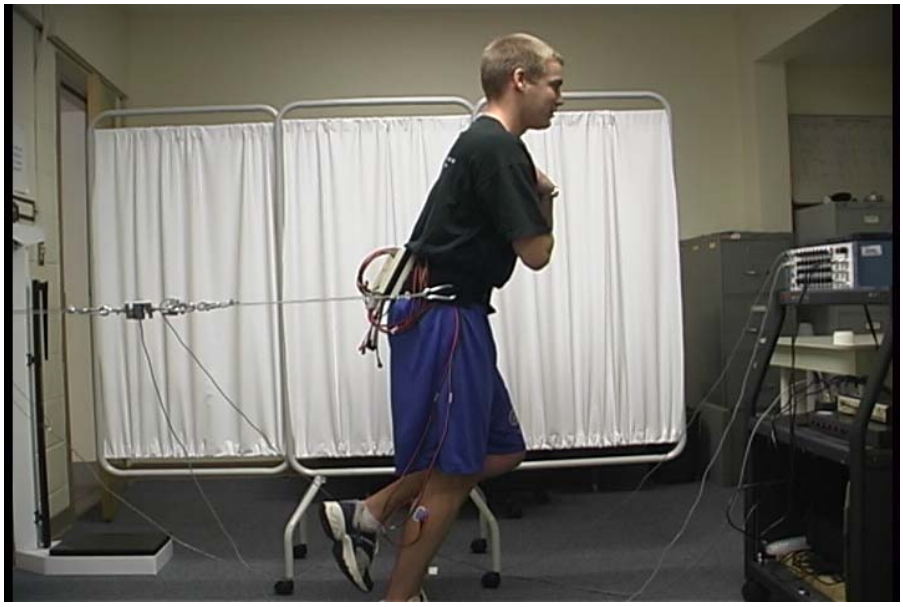


Figure 3-1. Lower extremity perturbation device

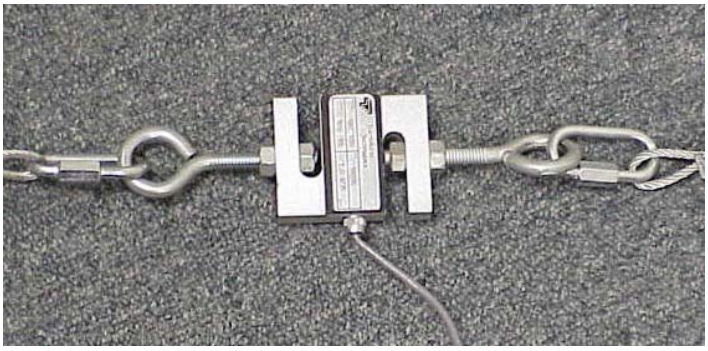


Figure 3-2. Load cell

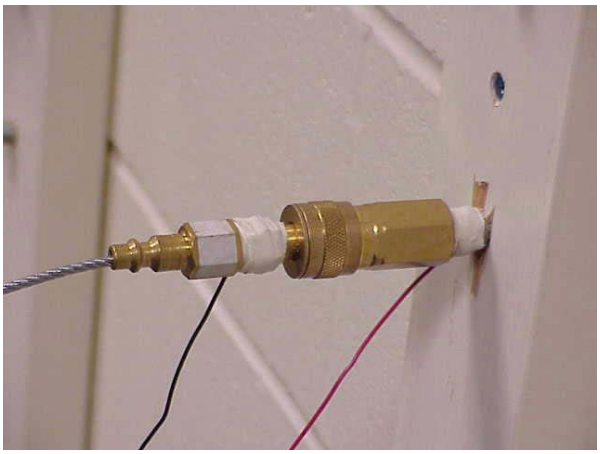
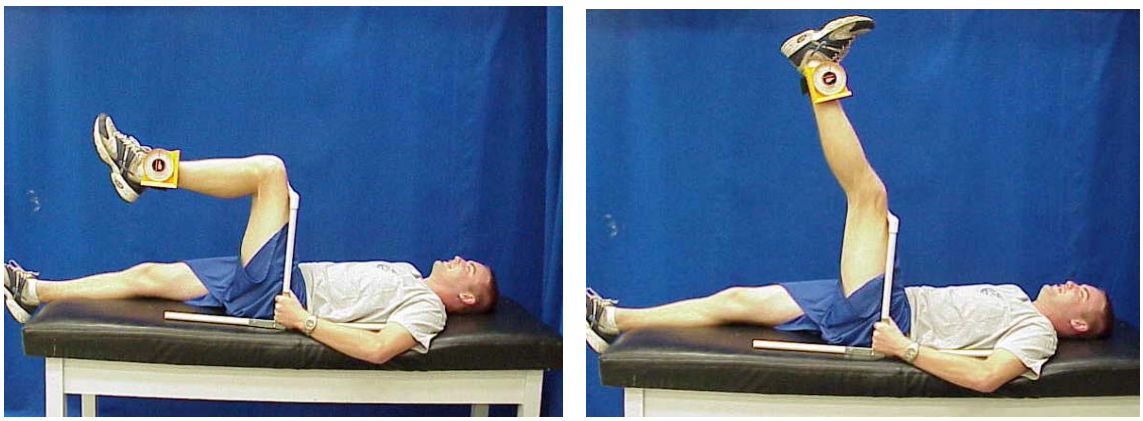


Figure 3-3. Height adjustable release mechanism and trigger switch

Figure 3-4. Bertec triaxial force platform

Figure 3-5. Inclinometer



A B

Figure 3-6. Subject positioning for ROM measure. A) start and B) end

Figure 3-7. Specially designed PVC device used for measuring hamstring range of motion

Figure 3-8. Nicholas Manual Muscle Tester. This device is a handheld dynamometer used to measure force output.

Figure 3-9. Algometer used for measuring the amount of pressure applied to the muscle.

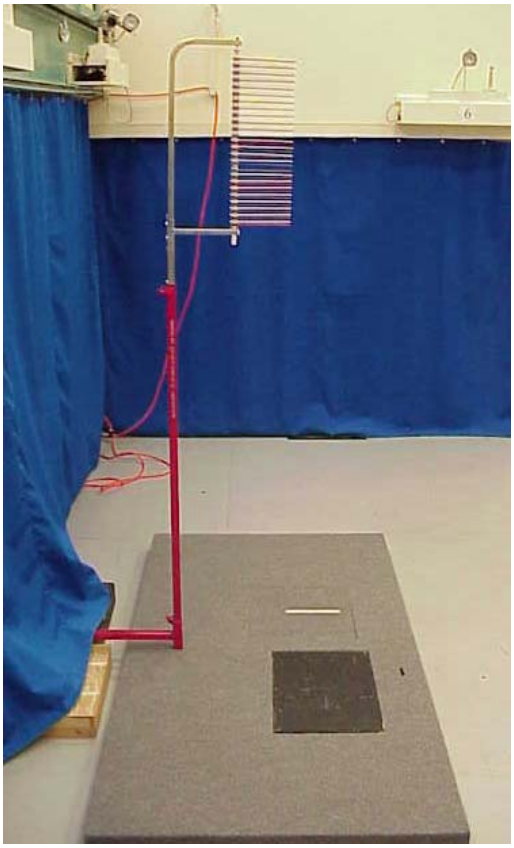
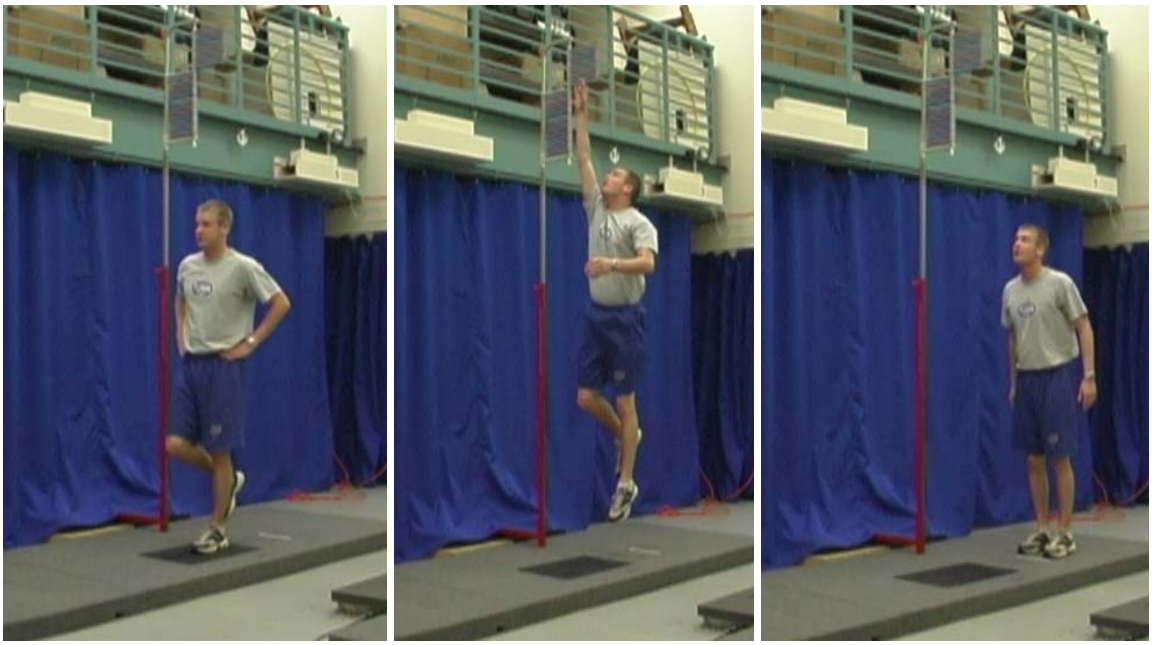


Figure 3-10. Vertec vertical jump device.



C B A

Figure 3-11. Time to stabilization jump landing sequence. A) starting position, B) mid-flight, and C) landing phase.

CHAPTER 4
RESULTS

Statistical analysis for the DOMS measures was conducted using separate 2-way mixed design ANOVA for each dependent variable (active range of motion, passive range of motion pain threshold, and pressure pain threshold). Two 3-way mixed design ANOVA were used to analyze the muscle latency times for internal and external rotation perturbations. Finally, three separate 2-way mixed design ANOVA were performed for the three TTS measures (based on Fz, Mx, and My) analyzed during the jump landing procedure. Tukey's HSD post hoc analysis was conducted when significance was established. The alpha level was set at 0.05 for all statistical tests.

DOMS Measures

Measurement of Active Range of Motion

Significant main effects for time ($F_{2, 56} = 19.08$, $P < 0.001$) and group ($F_{1, 28} = 11.22$, $P = 0.002$) were observed for the active range of motion measures. Subjects showed significant deficits in hamstring AROM at 48h and 96h posttreatment. A significant recovery of AROM was noticed between the second and third test sessions. Overall, hamstring flexibility was significantly less in the DOMS subjects compared to the control subjects. A significant time x group interaction ($F_{2, 56} = 19.98$, $P < 0.001$) was also noted. Data presented in Table 4.1 indicate that subjects in the DOMS group displayed marked decreases in hamstring flexibility over the 48h and 96h test periods when compared to the pretest measures. A significant decrease from baseline to 48h post exercise was observed. Subjects began to regain significant flexibility between the

second and third test session, although significant decreases at 96h posttest were still present compared to baseline. No differences were noted among the three trials for the control groups, but significant decreases were seen comparing between control and DOMS groups at 48h posttest and 96h posttest, respectively.

Table 4-1. Active hamstring range of motion (º ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	65 ± 14	43 ± 24*†	51 ± 18*†‡	53 ± 21†
Control	71 ± 13	71 ± 13	72 ± 12	71 ± 13
Total	68 ± 14	57 ± 24*	62 ± 19*‡	

* Significantly less than pretest group ($P < 0.05$)
† Significantly less than control group at same posttest time ($P < 0.05$)
‡ Significantly greater than posttest 48h ($P < 0.05$)

Passive Range of Motion Pain Threshold

Significant main effects for time ($F_{2, 56} = 11.97$, $P < 0.001$) and group ($F_{1, 28} = 4.58$, $P = 0.041$) were present for the PROMPT measure. Data presented in Table 4.2 indicate that there was a significant increase in pain perception due to passive stretch of the hamstring muscles in the DOMS group. Overall, subjects also noted significantly higher levels of pain 48 and 96h after exercise. Muscle soreness peaked by the third session, but was only slightly higher than the 48h session. Additionally, a significant time x group interaction ($F_{2, 56} = 10.58$, $P < 0.001$) was identified. A significant increase in pain perception was noted when comparing each posttest DOMS measure to the pretest DOMS measure. No significant changes were present among the control groups, although significant differences were seen when comparing the 48h control group to the 48h posttest DOMS group and the 96h posttest control group to the 96h posttest DOMS group.

Table 4-2. Pain quotient as measured on a visual analog pain scale for passive range of motion pain threshold (PQ ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	18.1 ± 20.6	46.6 ± 27.6*†	47.1 ± 28.8*†	37.3 ± 28.8†
Control	19.3 ± 21.0	20.7 ± 24.1	19.8 ± 25.4	19.9 ± 23.1
Total	18.7 ± 20.4	33.6 ± 28.7*	33.4 ± 30.1*	

* Significantly greater than pretest group ($P < 0.05$)
† Significantly greater than control group at same posttest time ($P < 0.05$)

Pressure Pain Threshold

A significant main effect for time ($F_{2, 56} = 7.23$, $P = 0.002$) was identified for the PPT measure (Table 4.3). Pain perception peaked at 48h but began to diminish by the 96h post exercise session. A significant group x time interaction ($F_{2, 56} = 6.91$, $P = 0.002$) was also present. Subjects in the DOMS group reported significantly greater levels of perceived pain at the 48h and 96h post exercise sessions compared to the first session. No significant changes were seen across the trials for the control subjects, but a significant difference was present between the posttest 48h control group and the posttest 48h DOMS group. No significant group main effect ($F_{1, 28} = 1.09$, $P = 0.304$) was present for this measure.

Table 4-3. Pain quotient as measured on a visual analog pain scale for Pressure Pain Threshold (PQ ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	43.9 ± 17.8	63.1 ± 21.8*†	58.5 ± 24.2*	55.2 ± 22.5
Control	45.6 ± 27.7	45.0 ± 28.6	47.6 ± 29.8	46.1 ± 28.1
Total	44.7 ± 22.9	54.1 ± 26.6*	53.1 ± 27.2*	

* Significantly greater than pretest group ($P < 0.05$)
† Significantly greater than control group at posttest 48h ($P < 0.05$)

Muscle latency

Internal Rotation Perturbation

No significant main effects were detected for muscle ($F_{1, 18} = 0.043$, $P = 0.838$) or group ($F_{1, 18} = 2.01$, $P = 0.173$); however, a trend was observed for time ($F_{2, 36} = 2.77$, $P = 0.076$) main effect with the internal rotation lower extremity perturbation (Table 4.4). No significant 2-way interactions were detected for time x group ($F_{2, 36} = 0.35$, $P = 0.705$), muscle x group ($F_{1, 18} = 0.408$, $P = 0.531$), or time x muscle ($F_{2, 36} = 1.03$, $P = 0.368$). Comparison of the 3-way interaction for time x muscle x group also detected no significant results ($F_{2, 36} = 1.80$, $P = 0.180$).

Table 4-4. Internal rotation muscle latency (msec ± SD)

Muscle	Group	Pretest	Post 48h	Post 96h	Total
MH	DOMS	97 ± 24	72 ± 23	81 ± 23	92 ± 26
	Control	106 ± 15	99 ± 23	96 ± 33	
LH	DOMS	101 ± 33	106 ± 122	64 ± 23	94 ± 58
	Control	120 ± 34	83 ± 29	87 ± 34	
	Total	106 ± 28	90 ± 64	82 ± 30	

External Rotation Perturbation

Significant main effects were observed for time ($F_{2, 36} = 8.60$, $P = 0.001$) and muscle ($F_{1,18} = 4.97$, $P = 0.039$) for the latent muscle reaction times of the hamstrings (Table 4.5). Subjects recorded significantly quicker response times during the 48h (19 msec) and 96h (16 msec) posttest trials as compared to the pretest measure. Medial hamstring activation times (86 ± 25 msec) were significantly faster than lateral hamstring response times (99 ± 31 msec). No significant interactions were detected, however a trend was noted for the time by group interaction ($F_{2, 36} = 3.05$, $P = 0.060$). No

significant main effect was observed between the DOMS and control groups ($F_{1, 18}$ = 545.50, P = 0.668)

Table 4-5. External rotation muscle latency (msec ± SD)

Muscle	Group	Pretest	Post 48h	Post 96h	Total
MH	DOMS	104 ± 27	81 ± 25	79 ± 18	86 ± 25†
	Control	85 ± 20	84 ± 31	84 ± 25	
LH	DOMS	114 ± 26	79 ± 23	86 ± 26	99 ± 31
	Control	113 ± 43	96 ± 32	103 ± 18	
	Total	104 ± 31	85 ± 28*	88 ± 23*	

* Significantly less than pretest group ($P < 0.05$)
† Significantly faster response than lateral hamstring ($P < 0.05$)

Time to Stabilization

Vertical Ground Reaction Force (Fz)

A significant time main effect ($F_{2, 54}$ = 5.04, P = 0.010) for TTS based on Fz was present. Data presented in Table 4.6 indicate that subjects displayed significant improvement of TTS during the 96h posttest session (1515 ± 572 msec) compared to the baseline (1868 ± 514 msec) and posttest 48h session (1839 ± 605 msec). Neither the group main effect ($F_{1, 27}$ = 1.44, P = 0.240) nor the time x group interaction ($F_{2, 54}$ = 0.71, P = 0.497) were observed to be significant.

Table 4-6. Vertical TTS based on Fz (msec ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	1905 ± 404	1911 ± 606	1696 ± 453	1830 ± 643
Control	1834 ± 612	1772 ± 617	1346 ± 634	1635 ± 749
Total	1868 ± 514*	1839 ± 605*	1515 ± 572	

* Significantly greater than posttest 96h group ($P < 0.05$)

Medial/Lateral Ground Reaction Moment (Mx)

No significant time ($F_{2, 54}$ = 1.49, P = 0.234) or group ($F_{1, 27}$ = 1.14, P = 0.294) main effects for TTS based on Mx were present. Similarly, no significant changes were

observed for the time x group interactions ($F_{2, 54} = 0.82$, $P = 0.448$). Data are presented in Table 4.7.

Table 4-7. Medial/Lateral TTS based on Mx (msec ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	1789 ± 343	1640 ± 203	1582 ± 419	1670 ± 529
Control	1579 ± 290	1641 ± 425	1506 ± 377	1575 ± 564
Total	1680 ± 329	16401 ± 331	1542 ± 392	

Anterior/Posterior Ground Reaction Moment (My)

Analysis of TTS based on My revealed no significant main effects for time ($F_{2, 54} = 0.22$, $P = 0.806$) or group ($F_{1, 27} = 2.86$, $P = 0.102$). Additionally, no significant differences were detected for the time x group interaction ($F_{2, 54} = 0.72$, $P = 0.492$). Data appear in Table 4.8.

Table 4-8. Anterior/Posterior TTS based on My (msec ± SD)

Group	Pretest	Post 48h	Post 96h	Total
DOMS	1606 ± 288	1618 ± 334	1657 ± 346	1627 ± 538
Control	1599 ± 268	1494 ± 319	1453 ± 315	1516 ± 558
Total	1603 ± 273	1554 ± 327	1552 ± 341	

CHAPTER 5
DISCUSSION AND CONCLUSIONS

Discussion

Neuromuscular control testing continues to be a thoroughly studied topic among researchers in the sports medicine field. The majority of research in this area has focused on the dynamic measurement of joint stability and postural control. Current research uses the TTS measure defined in the present study. The most recent research has investigated whether ground reaction forces differ between a static single limb stance and a dynamic single limb stance. Results indicate that greater GRFs exist in the A/P and M/L planes for dynamic single limb stance compared to static single limb stance. It was concluded that the static measure might be a better indicator of stable posture.[49]

Shultz and colleagues continue to incorporate the use of the LEPD in the examination of hamstring muscle activation. In fact, she is currently investigating how the menstrual cycle of healthy females affects latent muscle reaction times of the lower extremity. Bell et al.[50] have investigated the effects of trunk position on muscle reaction times of the lower extremity using the LEPD. They concluded that trunk position does not affect muscle reflex onset based on where center of pressure is in relation to the foot, but that reflex amplitude is affected.

Several quantitative physiological markers of DOMS have been used to confirm the presence of this condition. Although these physiological measures were not used in this study, successful completion of the work hinged on the ability to effectively induce

DOMS. Alternatively, subjective (PROMPT and PPT) and objective (AROM) outcome measures associated with DOMS were utilized.

The purpose of this investigation was to determine if hamstring DOMS had any deleterious effects on functional joint stability at the knee. More specifically, it was hypothesized that measures of DOMS would be significantly changed after the exercise protocol for the experimental group. It was further hypothesized that subjects in the experimental group would present with significantly slower reaction times in relation to both latent muscle response times of the hamstrings and TTS following a jump landing procedure.

DOMS Measurements

Based on the results presented, I am confident that DOMS was adequately induced. Certain markers that were discussed in Chapter 2, such as range of motion and perceived pain, were significantly different after the exercise protocol. Active range of motion in the experimental group showed the greatest decrease approximately 48h after the exercise session (Figure 5.1). Subjects began to regain motion at 96h post exercise, but were still not fully recovered from the muscle tightness. Peak levels of perceived pain were noted at 48 – 96h post exercise for the PROMPT measure (Figure 5.2). Similar results were observed with the PPT measures, noting that levels of perceived pain peaked at 48 – 96h post exercise (Figure 5.3). All measures are similar to previous literature related to the time course and intensity of DOMS.[31, 32, 34, 51] Typically, symptoms are noticed approximately 8 – 24h after exercise, with symptoms reported peaking at 48 – 72h. Symptoms begin to diminish after this time and will subside within 5 – 7 days.

Previous research has used other means of determining DOMS

markers,[32, 34, 35 - 37, 51, 52, 53] however, our markers were deemed more relevant with respect to this study. This may create limitations when assessing the level of DOMS that was induced when making comparisons to previous research. For this study, only three measures associated with assessment of DOMS markers were used, however these measures were all confirmed to have significant changes. Other measures such as muscular strength, plasma creatine kinase levels, and muscular swelling were not included. We chose to use the AROM, PROMPT, and PPT as the measures of DOMS inducement due to their ease of measurement as well as to eliminate any invasive measures such as with the CK tests. No correlation analyses have been performed specifically to determine whether differences exist among the multiple DOMS markers. All the procedures are independently considered valid and reliable measures to assess DOMS.[36, 38, 39, 51]

Muscle Latency

When assessing joint stability with EMG, multiple models have been used[8, 24, 25, 44, 54 - 57] A review of the relevant research revealed no data published to assess temporal patterns of a dynamic protocol. To avoid learning effects in previous studies, researchers allowed multiple practice trials before the actual recording of the data. This was also done in the present study, but because of the number of test trials over time, it seems that there may be a greater training effect associated with the LEPD. Following two sessions, pretest and posttest 48h, it appears that subjects may have begun to develop a learning pattern relative to the perturbation. This was evidenced by their reduced latency times. Although only significantly different during the EROT perturbation, there was a similar trend observed with the IROT perturbation. It is difficult to explain this faster response time. Measurements from pretest to both posttests were performed

identically. Subjects may have become accustomed to the device and learned to anticipate the rotational perturbation, which would allow them to respond sooner. On the other hand, when the subjects were leaning forward, they may have been required to utilize predominantly their quadriceps to maintain stability in the upright posture. Therefore, when the perturbation was initiated, some level of reciprocal inhibition of the hamstrings may have existed, causing them to be recruited far slower than in previous studies.

Previous research assessed muscle latency in the hamstrings using both weight-bearing[15, 24 - 26, 44, 46, 47] and non weight-bearing perturbations.[54] The assessment of non weight-bearing perturbations cannot be directly related to this research because the proprioceptive feedback is more likely to come from joint receptors being stimulated as opposed to the muscle spindles that would be activated with a weight-bearing task. When non-weight bearing, the muscles are not loaded and would require a larger amount of joint movement to stimulate the muscle spindle. The more obvious receptors that would be activated during this type of perturbation would be the receptors located in the joint as well as the capsule and ligaments.[54] Weight-bearing perturbations require somewhat of a preactivation of the muscle, which allows the muscle spindles to respond to changes in muscle length earlier than other receptors.

Shultz et al.[44] described the same activation patterns of the medial and lateral hamstrings in response to both IROT and EROT as were seen in the present study. It was noted that the medial hamstrings respond faster to both perturbations. This makes sense for EROT, because the afferent response from the muscle spindle would cause a reflex contraction of the medial hamstring to prevent the trunk and femur from further

externally rotating. For the muscles to respond to the IROT perturbation similarly is not as easily understandable when the same muscle spindle theory is applied. In this instance, the lateral hamstring should respond quicker due to the stretch reflex associated with that muscle. Shultz et al.[44] stated that the possibility for this faster response from the medial hamstrings could be due to the innervation of the muscle. The semitendinosus and semimembranosus are supplied by the tibial nerve, whereas the biceps femoris is supplied by both the tibial (long head) and common peroneal (short head) nerves.[21] Based on this reasoning, a second theory was identified. Therefore, this theory implies that if the tibial nerve and common peroneal nerve are not stimulated by the perturbation, or that the recording area of the biceps femoris was over the peroneal nerve, differing times could be recorded. The researchers further identified factors in their study to support this theory. When the medial hamstrings were compared to the medial and lateral gastrocnemius muscles, which are all innervated by the tibial nerve, there were no significant differences.[44]

Although the results of Shultz et al.[44] are consistent with our findings based on the firing patterns of the hamstrings, we noticed a much longer latency when compared to their findings. A long latency response time for the medial hamstring ranged from 58 – 60 msec and 70 – 77 msec for the lateral hamstring. Our results ranged from approximately 86 – 92 msec for medial hamstring and 94 – 99 msec for the lateral hamstrings. It is difficult to make a direct assessment relative to other research because the methods of this study are not entirely the same as others. Shultz et al.[44] used a similar perturbation device that required their subjects to maintain their center of mass over the midfoot. In this investigation, subjects were asked to lean into the cables using load cells

as the standardization protocol. Although the subjects' position was consistent across the trials, center of mass location could not be assessed. A possible flaw with this setup is that it does not mimic the typical injury model for most ligament injuries in sports. Most frequently a deceleration or sudden change of direction results in ligament damage. With a deceleration or change of direction, the athletes center of mass should remain within or posterior to the base of support. Upon further assessment of this model, it would appear that the center of mass of the subject would lie outside the base of support. Bell et al.[50] concluded no change in hamstring response time relative to where the center of pressure lies, whether it is over the heel, midfoot, or toes. Their study, however, could not assess the possibility of the subjects' center of mass falling outside that base of support. Subjects may have also been able to incorporate the use of their hips to become more adept at responding to the perturbations. Because the hip is a triaxial joint, able to move in all three of the cardinal planes, there is the possibility that the subjects were responding to the perturbation by utilizing their ability to rotate the pelvic girdle relative to the femur and limit the internal and external rotation at the knee joint.

Time to Stabilization

Static and dynamic procedures have been utilized to investigate stability of the lower extremity. Various static measurements for the lower extremity have been used in previous research. These static measures either incorporate the use of a single leg balance test,[8, 58] center of pressure velocity (COPV),[59, 60] or postural sway.[61] To date, very few studies have used a functional task to assess joint stability. Colby et al.[42] and Ross et al.[43] have attempted to study stability of the lower extremity using a functional task. A potential limitation with these studies is that they only made comparisons between injured populations to uninjured populations. No research has been performed

to validate whether a jump landing is different in healthy subjects with an intervention protocol or whether there is a change in results over time. Colby et al.[42] concluded that subjects with ACL reconstructed knees showed significantly slower stabilization times based on the vertical force component compared to healthy subjects during a step down maneuver. Our results also noted differences in the vertical component, however these results showed faster stabilization times over multiple sessions while performing a jump landing technique. Figure 5.1 indicates that a learning effect may have taken place based on the Fz for the TTS measure. Subjects appeared to improve their ability to "stick" the landing after two sessions. This may have occurred because the subjects were more familiar with the task after multiple trials during multiple sessions. The jump landing task takes a great deal of coordination to complete successfully. Subjects were asked to focus on three main criteria in order for the jump landing to be considered successful (reach 50% of their max vertical jump height, cover a distance of 70cm, and land in the center of the force plate on one foot). Subjects who could not focus on all three criteria simultaneously seemed to be unable to complete the task consistently. This inconsistency was evident in the fact that, although all subjects completed the trials successfully, there was variability among subjects based on the number of total trials attempted to complete three successfully. It appears that subjects need to be adequately familiarized with the jump landing procedure in order to remain consistent over time.

It is difficult to compare the results of the present study with those of previous research. Different methods were used to assess TTS and different subject populations were used. Collection frequency could be a limitation of previous research as well. The current investigation used a sampling frequency of 2000Hz to collect data, while other

researchers have been collecting at much lower sampling rates, some as low as 180Hz for the TTS procedure.[62] In previous studies using the jump landing procedure with low sampling frequencies, researchers would have included fewer data points in the analysis, which could have resulted in altered outcomes. Based on the results of this procedure, it is likely that DOMS has little or no effect on a subject's ability to stabilize after a functional task, such as a jump landing maneuver. Because of the dynamic nature of the task, subjects would have to incorporate the use of not only the hamstring muscles, but would also need to rely on the entire kinetic chain of the lower extremity to stabilize themselves. It would appear that function of the lower extremity as a whole to control dynamic posture is too great to be significantly affected by limiting one muscle group.

Conclusions

The exercise procedure conducted to induce DOMS can be considered a valid protocol for this type of research. Subjects presented with marked changes across time for each measure. The change in the associated markers is consistent with past literature. The results of the present study suggest that functional joint stability, as measured by the combination of hamstring muscle latency and TTS after a jump landing, is not affected by DOMS. When subjects return to activity while affected by DOMS, the physiological and physical markers of muscle damage are still present. It is difficult to speculate why no effect is present. Apparently these markers are not influential enough to change the performance of the affected muscles. The protective effect the hamstrings provide at the knee joint to assist with dynamic stability does not appear to be influenced by DOMS.

Suggestions for Future Research

Future research should continue to build upon the present work as well as previous literature utilizing the jump landing procedure. An area that needs to be addressed is the

reliability of testing subjects over multiple days to determine if a learning response exists. Additionally, the development of a standardized TTS protocol to establish baseline criteria for functional task procedures is also necessary. The task suggested here is an effective model, but something more appropriate would be to develop criteria to standardize both jump height and jump distance. The incorporation of a standardized jump distance based on a percentage of the standing broad jump for each subject would produce a consistent trajectory from take off to landing. This would create a reliable pattern among subjects that to date has not been identified.

Utilizing a functional task to determine if hamstring DOMS affected knee joint stability was the goal of this research. Further research should examine the effects of hamstring DOMS on other aspects of proprioception. Some areas that should be investigated include active and passive joint repositioning in addition to threshold to detection of passive movement. Because the hamstrings play a major role in joint movement at both the hip and knee, it would seem sensible to study each independently.

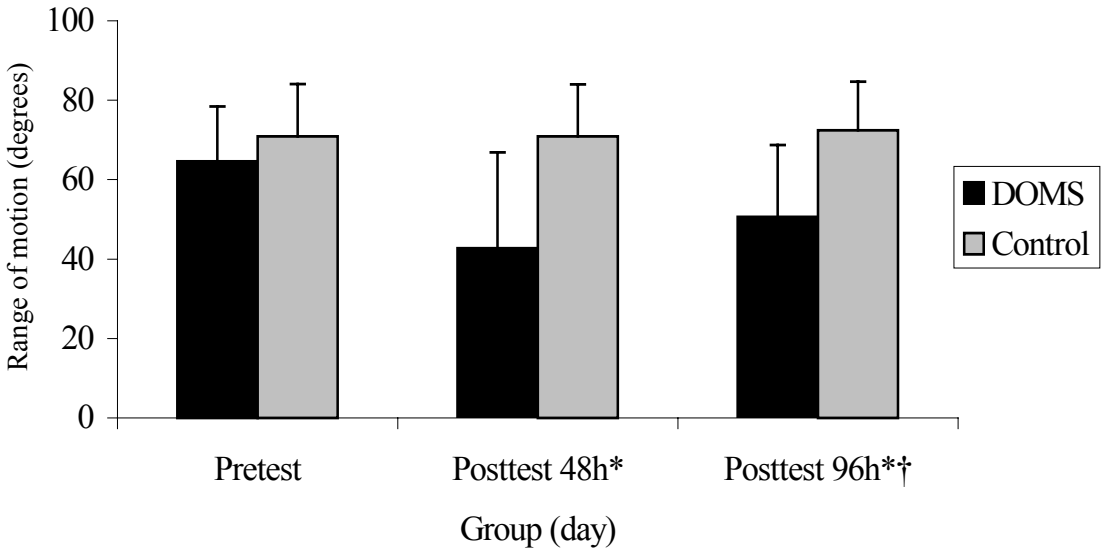


Figure 5-1. Active hamstring range of motion

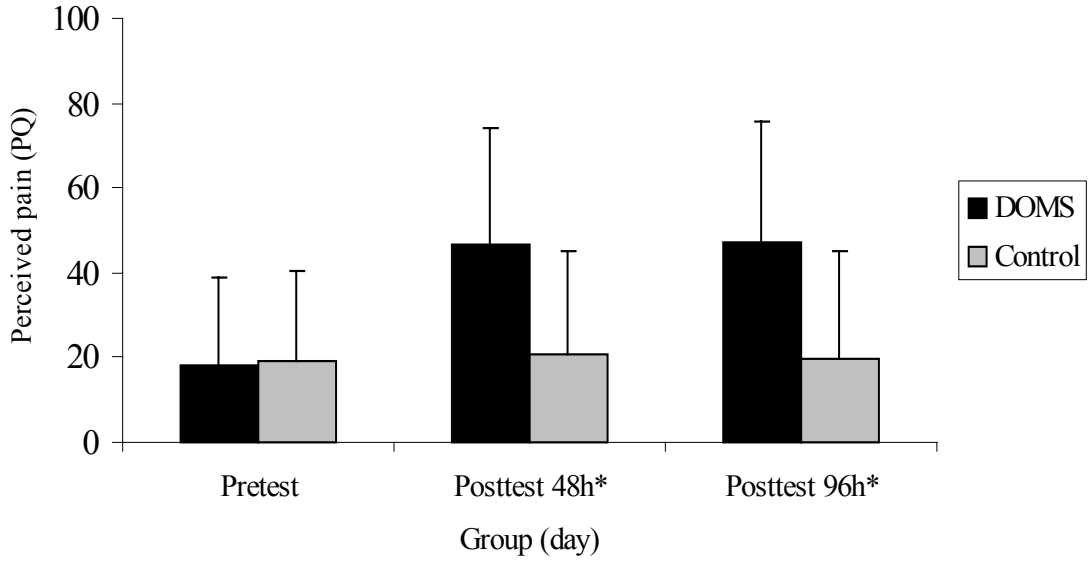


Figure 5-2. Passive range of motion pain threshold

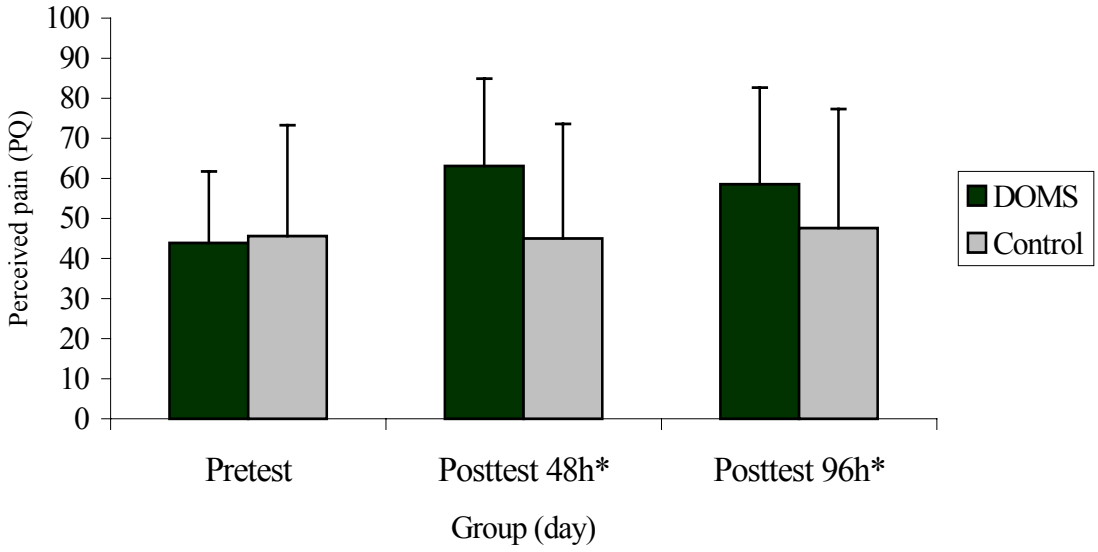


Figure 5-3. Pressure pain threshold

APPENDIX A
LETTER OF INFORMED CONSENT

Informed Consent Agreement

Project Title: The effects of hamstring delayed onset muscle soreness on functional knee joint stability.

Investigators: Kyle A Smink, ATC, Graduate Student, Department of Exercise and Sport Sciences & Michael E. Powers, PhD., ATC, CSCS, Assisstant Professor, Department of Exercise and Sport Sciences.

Purpose of the study:
The purpose of this study is to determine if the occurrence of delayed onset muscle soreness in the hamstring group can affect functional knee joint stability. Delayed onset muscle soreness is a physiological response that occurs when individuals take part in unaccustomed bouts of rigorous exercise and is typically noticed 24 – 48 hours after the initial activity.

At this time, no study has been published investigating the occurrence of delayed onset muscle soreness and its effects on functional joint stability. We are performing this research in order to help gain knowledge and further understand this subject as it relates to the sports medicine field.

Please read this consent carefully before you decide to participate in this study.

What will you do in this study?

You will be excluded from participating in this study if you have had any leg injuries, either muscular or ligament, that required a doctor visit within the past six months. You will also be excluded from this study if you have taken part in any rigorous weight training for your leg muscles in the past six months.

Upon arrival to the Athletic Training/Sports Medicine Research Lab (FLG 105D), you will be asked to complete a medical history questionnaire to determine if you are eligible to participate in this study. If eligible, we will measure your hamstring (muscles in the back of your thigh) flexibility, pressure-pain threshold, and passive (relaxed) range of motion pain threshold. We will ask you to lie on your back with your non-dominant (the leg you would not kick a ball with) leg flexed at the hip. The opposite leg will remain flat on the table. A specially designed device made of PVC pipe will be used to make sure your hip remains at this angle through the entire measurement. You will then be asked to straighten your knee as far as possible. As you do this we will measure how far in degrees you can straighten your knee. While remaining is this position, an

examiner will stretch your hamstring muscles (by straightening your knee) to the maximum range of motion. You will be asked to make a pencil mark on a visual analog pain scale representing the amount of pain you feel during the stretch. This scale consists of a 10-centimeter line with the left end representing no pain at all and the right end representing the most unbearable pain imaginable. After you make the pencil mark, your hamstrings will be stretched again. This time, pressure will be applied to the hamstring muscles using an algometer (a device about the size of a pencil with a rubber tip used to apply a standard amount of pressure). You will again be asked to make a pencil mark in a visual analogue scale representing the amount of pain you feel while the pressure is applied.

Following the baseline measures for hamstring flexibility and pain threshold, you will be measured for muscle latency and time to stabilization. First, small areas of your skin will be shaven and cleaned with isopropyl alcohol. Self-adhesive surface electrodes will then be placed on the skin overlying the medial and lateral hamstrings (rear thigh), medial and lateral quadriceps (front thigh), and medial and lateral gastrocnemius (calf) muscles. These electrodes will detect electrical impulses of the muscle, however, you will not feel these impulses and no electrical current will enter the body. A device called a goniometer (a device that measures joint angles) will be placed over the outside of your knee to assess how far your knee is bent. You will then be asked to perform the knee perturbation and time to stabilization measures in a random order determined by a random numbers chart.

For the knee perturbation, you will be fitted with a harness applied snugly around your waist. Two cables connected to two separate release mechanisms affixed to a wall will be attached to the harness. You will be asked to stand on a force plate and assume a single leg stance on the test leg. You will then be asked to lean forward so that your knee is flexed to approximately 30º and your weight is supported by the cables attached to the wall. You will be able to view a computer screen, which will allow you to monitor the position of your weight. You will be wearing headphones to avoid hearing sounds that may allow anticipation reflexes to occur. At random times one of the two cables will be released. This will cause your hips and upper body to move forward and rotate causing the knee to naturally rotate and flex. Three trials of each cable release (left and right side) will be performed so you may become accustomed with the device. Immediately following, ten random perturbations (5 left and 5 right) will be performed while allowing a 30 second rest time between trials. When all 10 trials are successfully completed, we will perform the same procedures for the opposite leg.

For the time to stabilization measurement, your maximum vertical leap (how high you can jump) will be determined. To do this, we will first measure how high you can reach while standing on your toes. You will then be asked to jump as high as possible and touch markers supported on a stand. Based on the number of markers you touch, the height of your jump is determined. We will have you repeat this two more times to ensure that we get an accurate measure. We will then measure how long it takes you to balance after jumping onto a platform. You will be asked to jump so that you reach a height equivalent to half of your maximum jump height and land on a platform about 27" away. We will ask that you land on the test leg only and balance yourself while your hands remain on your hips for a period of 5 seconds. After the 5-second period you will be asked to return to the starting position and repeat the measurement. This will be done

two more times for a total of three trials for each leg.

Following the baseline measures of muscle latency and time to stabilization, you will perform 6 sets of 10 negative (muscle lengthens while it contracts, otherwise known as a eccentric) contractions of the hamstrings using a leg curl machine. First, we will determine your 1 repetition maximum strength, which will be used as your exercise intensity. When the three sets are completed the session will be over.

You will be asked to return for reevaluation of time to stabilization and muscle latency 48 and 96 hours after the initial testing date. Upon return, you will have your hamstring flexibility and pain threshold reevaluated which will be used to assess the level of DOMS achieved. All evaluation procedures will be performed identical to the pretest trials, However no resistance exercise (leg curls) will be performed.

Time required:
Three sessions requiring approximately 90 minutes each.

Risks:
Discomfort and soreness in the hamstring muscles will be experienced following the bout of eccentric exercise. You may also experience some discomfort with the pressure threshold measure, but this discomfort will only last a few seconds while the measure is being taken. As with any type of resistance exercise, there is a slight risk of musculoskeletal injury. A certified athletic trainer will be present to evaluate and treat any such injuries that may occur. If you are still suffering from soreness in the hamstring muscles after the 96-hour posttest measure, the certified athletic trainer will instruct you on ways to decrease the soreness. No stretching may take place prior to this time.

Benefits/Compensation:
There are no direct benefits to you for participating.

Confidentiality:
Data will be kept confidential to the extent provided by the law. Your information will be assigned a code number. The list connecting your name to this number will be kept in a locked file. When the study is completed and the data have been analyzed, the list will be destroyed. Your name will not be used in any report.

Voluntary Participation:
Your participation is completely voluntary. There is no penalty for not participating.

Right to withdraw from the study:
You have the right to withdraw from the study at anytime without penalty.

Who to contact if you have questions about the study:

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148 Florida Gym
PO Box 118205
Gainesville, FL 32611-8205
(352) 392-0584, ext. 1332
Fax: (352) 392-5262
E-mail: mpowers@hhp.ufl.edu

Who to contact about your rights in the study:

UFIRB Office
Box 112250, University of Florida
Gainesville FL 32611-2250
(352) 392-0433.

Agreement:
I have read the procedure described above. I voluntarily agree to participate in the procedure and I have received a copy of this description.

Participant:_____ **Date:**_____

Principal Investigator:_____ **Date:**_____

APPENDIX B
INCLUSION QUESTIONNAIRE

History questionnaire

Subject #:

1. Have you visited a physician for any knee injuries in the past 6 months?

   YES NO

2. Have you had any "giving way" episodes with your knee in the past 6 months?

   YES NO

3. Have you had any "locking" or "clicking" episodes with your knee in the past 6 months?

   YES NO

4. Have you had any knee pain walking up or down stairs in the past 6 months?

   YES NO

5. Have you visited a physician for any hamstring muscle injuries in the past six months?

   YES NO

6. Have you participated in any strenuous lower extremity weight training within the past six months?

   YES NO

APPENDIX C
DECRIPTIVE INFORMATION AND HAMSTRING FLEXIBILITY

Descriptive Information

Subject #:
Gender:
Age:
Height:
Weight:

Standing max reach:

Vertical jump height:

Hamstring Flexibility

Pretest:	Right	Left
Trial 1:		
Trial 2:		
Trial 3:		
Post-test 48h:	Right	Left
Trial 1:		
Trial 2:		
Trial 3:		
Post-test 96h:	Right	Left
Trial 1:		
Trial 2:		
Trial 3:		

APPENDIX D
VISUAL ANALOGUE PAIN SCALE

Subject # Pre / Post Date: Session #

Range of Motion

No Pain _____Unbearable Pain

Pressure

Medial Hamstring

No Pain _____Unbearable Pain

APPENDIX E
RAW DATA

Table E-1. Subject demographic raw data

Subject	Age (y)	Height (cm)	Mass (kg)
ks001	22	170	60.0
ks002	21	163	60.0
ks003	23	170	64.0
ks004	21	157	60.0
ks005	22	177	76.0
ks006	21	170	55.0
ks007	20	163	57.0
ks008	21	170	71.0
ks009	21	170	63.5
ks010	23	170	67.5
ks011	22	160	70.0
ks012	24	183	76.5
ks013	23	170	63.5
ks014	21	173	68.5
ks015	20	154	57.5
ks016	27	180	65.5
ks017	24	178	77.0
ks018	21	173	84.5
ks019	20	188	74.0
ks020	22	170	59.5
ks021	22	166	62.0
ks022	22	178	74.0
ks023	20	165	59.0
ks024	20	160	47.5
ks025	21	170	84.5
ks026	22	188	85.0
ks027	22	178	83.0
ks028	22	170	68.5
ks029	22	185	70.5
ks030	21	175	59.0

Table E-2. Active range of motion (AROM) raw data (degrees)

Subject	Pretest	Post-test 48h	Post-test 96h	Group
ks001	78	65	71	DOMS
ks003	62	24	33	DOMS
ks005	67	48	67	DOMS
ks006	68	28	48	DOMS
ks008	85	53	75	DOMS
ks009	86	72	67	DOMS
ks011	70	70	63	DOMS
ks017	82	85	77	DOMS
ks018	62	23	27	DOMS
ks019	43	19	30	DOMS
ks002	78	83	80	Control
ks004	80	84	85	Control
ks007	85	84	87	Control
ks010	90	90	90	Control
ks012	80	71	78	Control
ks013	73	73	72	Control
ks014	78	76	78	Control
ks015	66	64	64	Control
ks016	61	64	52	Control
ks020	76	82	84	Control

Table E-3. Passive range of motion pain threshold (PROMPT) raw data (PQ = 0 – 100)

Subject	Pretest	Post-test 48h	Post-test 96h	Group
ks001	72.5	61	77	DOMS
ks003	5	22	16.5	DOMS
ks005	7	23	6	DOMS
ks006	10	55.5	68	DOMS
ks008	0	64.5	42	DOMS
ks009	36	73.5	98.5	DOMS
ks011	32	54	62	DOMS
ks017	0	8	59.5	DOMS
ks018	2	83.5	71	DOMS
ks019	0	14.5	0	DOMS
ks022	4	84	60.5	DOMS
ks024	16.5	45	32	DOMS
ks026	22	27	20.5	DOMS
ks027	21.5	7	28	DOMS
ks028	43	76.5	65	DOMS
ks002	0	0	0	Control
ks004	39	48	52	Control
ks007	0	0	0	Control
ks010	49	49	57	Control
ks012	0	0	0	Control
ks013	14.5	12.5	15.5	Control
ks014	54	47	63	Control
ks015	7	9	8.5	Control
ks016	40.5	40	18	Control
ks020	15	12	11	Control
ks021	52	74.5	65	Control
ks023	7	1	0	Control
ks025	2	1	1.5	Control
ks029	8	9.5	3	Control
ks030	2	7	2	Control

Table E-4. Pressure pain threshold (PPT) raw data (PQ = 0 – 100)

Subject	Pretest	Post-test 48h	Post-test 96h	Group
ks001	58	64	55.5	DOMS
ks003	39	53	43	DOMS
ks005	37	36	18.5	DOMS
ks006	37	74.5	73	DOMS
ks008	64.5	79	85	DOMS
ks009	47	92.5	100	DOMS
ks011	43	71	56.5	DOMS
ks017	22	41	68.5	DOMS
ks018	43	100	82	DOMS
ks019	0	21.5	13.5	DOMS
ks022	64	43	53	DOMS
ks024	46.5	72	62	DOMS
ks026	70	82	75.5	DOMS
ks027	34	53.5	32.5	DOMS
ks028	53.5	63.5	59	DOMS
ks002	5	6	0	Control
ks004	54	61	67	Control
ks007	63.5	38	47	Control
ks010	46.5	59	56	Control
ks012	13.5	4	22	Control
ks013	61	72	75	Control
ks014	88	85	90	Control
ks015	11	14	13.5	Control
ks016	74	66.5	70	Control
ks020	0	12	10.5	Control
ks021	63	59	77	Control
ks023	22.5	8.5	5	Control
ks025	62.5	62.5	63	Control
ks029	56	77.5	70.5	Control
ks030	63	50	47.5	Control

Table E-5. IROT hamstring muscle latency raw data (msec)

Subject	Pre MH	Post48 MH	Post 96 MH	Pre LH	Post 48 LH	Post 96 LH	Group
ks001	91	30	87	139	30	95	DOMS
ks003	90	62	30	108		30	DOMS
ks005	135	94	95	117	52	65	DOMS
ks006	76	82	64	100	47	33	DOMS
ks008	92	86	91	71	68	80	DOMS
ks009	99	85	88	115	76	88	DOMS
ks011	60	60	70	56	76	72	DOMS
ks017	107	87	119	151	107	78	DOMS
ks018	138	96	84	101	75	66	DOMS
ks019	89	38	82	55	81	37	DOMS
ks002	101	77	65	131	63	38	Control
ks004	123	99	58	89	83	62	Control
ks007	124	69	96	167	30	44	Control
ks010	98	98	90	157	107	133	Control
ks012	123	88	101	123	94	109	Control
ks013	108	128	87	89	88	73	Control
ks014	97	90	105	94	108	109	Control
ks015	83	144	102	83	46	73	Control
ks016	117	110	180	165	121	107	Control
ks020	87	87	80	99	94	125	Control

Table E-6. EROT hamstring muscle latency raw data (msec)

Subject	Pre MH	Post48 MH	Post 96 MH	Pre LH	Post 48 LH	Post 96 LH	Group
ks001	93	30	65	124	30	99	DOMS
ks003	102	72	70	81	64	66	DOMS
ks005	127	99	64	133	77	77	DOMS
ks006	70	77	78	84	82	60	DOMS
ks008	73	94	58	116	101	113	DOMS
ks009	90	92	93	128	98	106	DOMS
ks011	97	46	77	91	87	88	DOMS
ks017	110	101	123	148	102	100	DOMS
ks018	116	99	81	93	93	37	DOMS
ks019	161	103	79	147	60	115	DOMS
ks002	77	35	46	103	89	90	Control
ks004	76	76	77	73	74	76	Control
ks007	60	68	55	188	144	96	Control
ks010	114	95	100	95	58	88	Control
ks012	104	116	103	109	111	100	Control
ks013	84	94	87	161	131	124	Control
ks014	76	100	99	113	108	132	Control
ks015	59	35	59	34	41	95	Control
ks016	114	97	123	138	107	110	Control
ks020	86	127	88	111	95	120	Control

Table E-7. Time to stabilization based on vertical ground reaction force (Fz) raw data (msec)

Subject	Pretest	Post48	Post96	Group
ks03	2175	1908	1295	DOMS
ks05	2645	1714	1848	DOMS
ks06	1979	2622	1674	DOMS
ks08	1158	1342	1426	DOMS
ks09	2290	2120	1640	DOMS
ks11	1839	2550	2322	DOMS
ks17	1847	2631	2130	DOMS
ks18	2155	2587	1943	DOMS
ks19	1184	1024	847	DOMS
ks22	2128	1355	968	DOMS
ks24	1897	1699	1422	DOMS
ks26	2059	2571	1990	DOMS
ks27	1696	1638	2161	DOMS
ks28	1617	997	2070	DOMS
ks02	1085	2633	2322	Control
ks04	2717	1372	2206	Control
ks07	2187	1206	979	Control
ks10	2359	1500	1203	Control
ks12	953	1636	2004	Control
ks13	1125	1732	809	Control
ks14	838	1098	544	Control
ks15	1721	1599	1054	Control
ks16	2169	2833	839	Control
ks20	2135	2723	1461	Control
ks21	1645	1023	912	Control
ks23	2543	1960	1182	Control
ks25	2387	2387	2536	Control
ks29	2112	1827	1441	Control
ks30	1539	1053	704	Control

Table E-8. Time to stabilization based on medial/lateral ground reaction force (Mx) raw data (msec)

Subject	Pretest	Post48	Post96	Group
ks03	1373	1541	1771	DOMS
ks05	2016	1667	2139	DOMS
ks06	1966	1973	1747	DOMS
ks08	1484	1689	1435	DOMS
ks09	1868	1795	2387	DOMS
ks11	1575	1299	1246	DOMS
ks17	1996	1216	968	DOMS
ks18	2214	1711	1466	DOMS
ks19	1851	1609	1444	DOMS
ks22	2125	1620	2166	DOMS
ks24	2218	1611	1179	DOMS
ks26	1851	1910	1586	DOMS
ks27	1141	1726	1132	DOMS
ks28	1366	1600	1470	DOMS
ks02	1869	1616	1320	Control
ks04	1318	2043	1727	Control
ks07	1516	964	898	Control
ks10	1561	1256	1240	Control
ks12	1511	1323	2107	Control
ks13	1099	1672	1658	Control
ks14	1469	1198	1387	Control
ks15	1801	2279	1540	Control
ks16	1834	2375	1552	Control
ks20	1404	1157	1724	Control
ks21	2134	1573	859	Control
ks23	1513	1928	1419	Control
ks25	1875	1606	1652	Control
ks29	1675	2074	2222	Control
ks30	1111	1555	1283	Control

Table E-9. Time to stabilization based on anterior/posterior ground reaction force (My) raw data (msec)

Subject	Pretest	Post48	Post96	Group
ks03	1891	1232	1513	DOMS
ks05	1108	1113	1900	DOMS
ks06	1945	1733	1348	DOMS
ks08	1768	1379	1932	DOMS
ks09	1641	1875	1787	DOMS
ks11	1662	1483	1498	DOMS
ks17	1465	1017	1453	DOMS
ks18	1669	1820	1289	DOMS
ks19	1541	2012	2057	DOMS
ks22	2103	1856	1634	DOMS
ks24	1288	1705	1798	DOMS
ks26	1324	1811	1533	DOMS
ks27	1277	2090	1067	DOMS
ks28	1807	1530	2393	DOMS
ks02	1633	1640	1129	Control
ks04	1622	1624	1404	Control
ks07	1442	812	1370	Control
ks10	1783	1142	1894	Control
ks12	1805	1296	1257	Control
ks13	1920	1671	1479	Control
ks14	1390	1139	1347	Control
ks15	2025	1498	1254	Control
ks16	1329	1324	1572	Control
ks20	1246	1478	863	Control
ks21	1251	1852	1629	Control
ks23	1799	1792	1319	Control
ks25	1571	1982	1319	Control
ks29	1258	1811	1941	Control
ks30	1914	1351	2021	Control

APPENDIX F
ANOVA SUMMARY TABLES

Table F-1. Active range of motion (ANOVA)

Source	SS	DF	MS	F	Significance
Group	7902.346	1	7902.346	11.222	0.002
Error	19717.827	28	704.208		
Time	1823.262	2	911.631	19.082	0.000
Time x Group	1909.306	2	954.653	19.983	0.000
Error	2675.358	56	47.774		

Table F-2. Passive range of motion pain threshold (ANOVA)

Source	SS	DF	MS	F	Significance
Group	6760.000	1	6760.000	4.583	0.041
Error	41303.433	28	1475.123		
Time	4396.317	2	2198.158	11.967	0.000
Time x Group	3885.817	2	1942.908	10.577	0.000
Error	10286.533	56	183.688		

Table F-3. Pressure pain threshold (ANOVA)

Source	SS	DF	MS	F	Significance
Group	1867.778	1	1867.778	1.095	0.304
Error	47775.278	28	1706.260		
Time	1569.672	2	784.836	7.228	0.002
Time x Group	1501.206	2	750.603	6.913	0.002
Error	6080.456	56	108.580		

Table F-4. Internal rotation hamstring muscle latency (ANOVA)

Source	SS	DF	MS	F	Significance
Group	4039.120	1	4039.120	2.014	0.173
Error	36093.463	18	2005.192		
Time	11917.994	2	5958.997	2.767	0.076
Time x Group	1552.550	2	761.275	0.353	0.705
Error (Time)	77537.708	36	2153.825		
Muscle	88.714	1	88.714	0.043	0.838
Muscle x Group	847.063	1	847.063	0.408	0.531
Error (Muscle)	37341.163	18	2074.509		
Time x Muscle	3244.221	2	1622.111	1.027	0.368
Time x Muscle x Group	5682.575	2	2841.288	1.799	0.180
Error (Time x Muscle)	56859.445	36	1579.429		

Table F-5. External rotation hamstring muscle latency (ANOVA)

Source	SS	DF	MS	F	Significance
Group	355.782	1	355.782	0.190	0.668
Error	33749.259	18	1874.959		
Time	82299.626	2	4114.83	8.601	0.001
Time x Group	2918.814	2	1459.407	3.051	0.060
Error (Time)	17222.554	36	478.404		
Muscle	4666.586	1	4666.586	4.969	0.039
Muscle x Group	1507.157	1	1507.157	1.605	0.221
Error (Muscle)	16905.978	18	939.221		
Time x Muscle	1019.349	2	509.675	1.833	0.174
Time x Muscle x Group	31.005	2	15.503	0.056	0.946
Error (Time x Muscle)	10007.553	36	277.988		

Table F-6. Fz (ANOVA)

Source	SS	DF	MS	F	Significance
Group	754142.407	1	754142.407	1.442	0.240
Error	14118967.75	27	522924.731		
Time	2174292.874	2	1087146.437	5.044	0.010
Time x Group	305274.461	2	152637.231	0.708	0.497
Error	11638461.88	54	215527.072		

Table F-7. Mx (ANOVA)

Source	SS	DF	MS	F	Significance
Group	194639.022	1	194639.022	1.143	0.294
Error	4597459.284	27	170276.270		
Time	300364.828	2	150182.414	1.491	0.234
Time x Group	164279.966	2	82139.983	0.816	0.448
Error	5437986.165	54	100703.447		

Table F-8. My (ANOVA)

Source	SS	DF	MS	F	Significance
Group	270936.707	1	270936.707	2.858	0.102
Error	2559315.561	27	94789.465		
Time	42795.786	2	21397.893	.216	0.806
Time x Group	142122.692	2	71061.346	0.718	0.492
Error	5345552.934	54	98991.721		

LIST OF REFERENCES

1. Boden BP, Dean GS, Feagin JA Jr, Garrett WE Jr. Mechanisms of anterior cruciate ligament injury. *Orthopedics*. 2000 Jun;23(6):573-8.

2. McNair PJ, Marshall RN, Matheson JA. Important features associated with acute anterior cruciate ligament injury. *NZ Med J.* 1990; 14: 537-539.

3. Sheridan, Phil. Buckhalter likely lost for year. *The Philadelphia Enquirer* 27 Apr. 2002: C01.

4. Chargers draftee Charles tears ACL. *The Associated Press News Service* 27 Apr. 2002. 28 Apr. 2002 <http://www.nfl.com/teams/story/SD/5284673>.

5. Maisel, Ivan. "Walking wounded; College teams fight to keep players healthy." *Sports Illustrated on the Web* 05 May 2002. 07 May 2002 <http://sportsillustrated.cnn.com/inside_game/ivan_maisel/news/2002/05/03/maisel_insider/>.

6. Riemann BL, Lephart SM. The sensorimotor system, Part 1: The physiologic basis of functional joint stability. *J Ath Train* 2002; 37(1): 71-79.

7. Lephart SM, Pincivero DM, Rozzi SL. Proprioception of the ankle and knee. *Sports Med.* 1998; 25(3): 149-155.

8. Rozzi SL, Lephart SM, Fu FH. Effects of muscular fatigue on knee joint laxity and neuromuscular characteristics of male and female athletes. *J Ath Train*. 1999; 34(2): 106-114.

9. Emerson RJ. Basketball knee injuries and the anterior cruciate ligament. *Clin Sports Med*. 1993 Apr; 12(2): 317-28.

10. Hoppenfeld Stanley. *Physical examination of the spine and extremities*. Norwalk, CT: Appleton & Lange, 1976.

11. Starkey, C, Ryan J. *Evaluation of orthpedic and athletic injuries.* 2nd ed. Philadelphia: FA Davis Company, 2002.

12. Hungerford DS, Barry M. Biomechanics of the Patellofemoral Joint. *Clinical Orthopaedics and Related Research*. 1979; 144: 9-15.

13. Fujita I, Nishikawa T, Kambic HE, Andrish JT, Grabiner MD. Characterization of hamstring reflexes during anterior cruciate ligament disruption: In vivo results from a goat model. *J Orthop Res*. 2000; 18(2): 183-189.

14. Seto JL, Orofino AS, Morrisey MC, Medeiros JM, Mason WJ. Assessment of quadriceps/ hamstring strength, knee ligament stability, functional and sports activity levels five years after anterior cruciate ligament reconstruction. *Am J Sports Med*. 1988; 16: 170-180.

15. Di Fabio RP, Graf B, Badke MB, Breunig A, Jensen K. Effect of knee joint laxity on long-loop postural reflexes: evidence for a human capsular-hamstring reflex. *Exp Brain Res*. 1992; 90(1): 189-200.

16. Smith BA, Livesay GA, Woo SL. Biology and biomechanics of the anterior cruciate ligament. *Clinics in Sports Med*. 1993; 12(4): 637-670.

17. Covey DC, Sapega AA. Anatomy and function of the posterior cruciate ligament. *Clinics in Sports Med*. 1994; 13(3): 509-518.

18. Girgis FG, Marshall JL, Al Monajem ARS. The cruciate ligaments of the knee joint. Anatomical, functional and experimental analysis. *Clin Orthop*. 1975; 106: 216-231.

19. Butler DL, Noyes FR, Grood ES. Ligamentous restraints to anterior-posterior drawer in the human knee. A biomechanical study. *J Bone Joint Surg*. 1980; 62: 259-270.

20. Gollehon DL, Torzilli PA, Warren RF. The role of the posterolateral and cruciate ligaments in the stability of the human knee. A biomechanical study. *J Bone Joint Surg*. 1987; 69: 233-242.

21. Sieg KW, Adams SP. *Illustrated Essentials of Musculoskeletal Anatomy*, 2nd ed. Gainesville, FL: Megabooks; 1985.

22. Colby S, Francisco A, Yu B, Kirkendall D, Finch M, Garrett W. Electromyographic and kinematic analysis of cutting maneuvers: implications for anterior cruciate ligament injury. *Am J Sports Med*. 2000; 28(2): 234-240.

23. Arendt EA, Agel J, Dick R. Anterior cruciate ligament injury patterns among collegiate men and women. *J Athl Train*. 1999; 34(2); 86-92.

24. Jennings AG, Seedhom BB: Proprioception in the knee and reflex hamstring contraction latency. *J Bone Joint Surg*. 1994 ; 768: 491-494.

25. Beard DJ, Kyberd PJ, Fergusson CM, Dodd CA. Proprioception after rupture of the anterior cruciate ligament. An objective indication of the need for surgery? *J Bone Joint Surg Br* 1993;75(2):311-5

26. Beard DJ, Kyberd PJ, O'Connor JJ, et al: Reflex hamstring contraction latency in anterior cruciate ligament deficiency. *J Orthop Res* . 1994; 12(2): 219-228.

27. Powers, Scott K., and Edward T. Howley. Exercise Physiology. 3rd ed. Boston: McGraw-Hill, 1997.

28. Lephart SM, Pincivero DM, Giraldo JL, Fu FH. The role of proprioception in the management and rehabilitation of athletic injuries. *Am J Sports Med.* 1997; 25(1): 130-137.

29. Feagin JA, Lambert KL. Mechanism of injury and pathology of anterior cruciate ligament injuries. *Orthop Clin North Am*. 1985; 16: 41-45.

30. Noyes F, Mooar PA, Matthews DS, Butler DL. The symptomatic anterior cruciate-deficient knee, I: the long term functional disability in athletically active individuals. *J Bone Joint Surg Am.* 1983; 65: 154-162.

31. Bobbert MF, Hollander PA, Huijing PA. Factors in delayed onset muscle soreness of man. *Med Sci Sports Exerc.* 1986; 18(1): 75-81.

32. Isabell WK, Durrant E, Myrer W, Anderson S. The effects of ice massage, ice massage with exercise, and exercise in treatment of delayed onset muscle soreness. *J Ath Train*. 1992; 27(3): 208-217.

33. Friden J, Sjostrom M, Eklblom B. Myofibrilar damage following intense eccentric exercise in man. *Int J Sports Med.* 1983; 4: 170-176

34. Nosaka K, Clarkson PM. Muscle damage following repeated bouts of high force eccentric exercise. *Med Sci Sport Exerc*. 1995; 27(9): 1263-1269.

35. Nosaka K, Sakamoto K, Newton M, Sacco P. How long does the protective effect on eccentric exercise-induced muscle damage last? *Med Sci Sports Exerc*. 2001; 33: 1490-1495.

36. Schwane JA, Williams JS, Sloan JH. Effects of training on delayed muscle soreness and serum creatine kinase activity after running. *Med Sci Sports Exerc.* 1987; 19(6): 584-590.

37. Tiidus PM, Ianuzzo CD. Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Med Sci Sports Exerc*. 1983; 15: 461-465.

38. Evans WJ, Meredith CN, Cannon JG, Dinarello CA, Frontera WR, Hughes VA, Jones BH, and Knuttgen HG. Metabolic changes following eccentric exercise in trained and untrained men. *J Appl Physiol*. 1986; 61(5): 1864-1868.

39. Smith LL. Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Med Sci Sports Exerc.* 1991; 23(5): 542-551.

40. Ebbeling CB, Clarkson PM. Muscle adaptation prior to recovery following eccentric exercise. *Eur J Appl Physiol*. 1990; 60: 26-31.

41. Byrnes WC, Clarkson PM, Spencer-white J, Hseih SS, Frykman PB, Maughan RJ. Delayed onset muscle soreness following repeated bouts of downhill running. *J Appl Physiol*. 1985; 59: 710-715.

42. Colby S, Hintermeister R, Torry M, Steadman R. Lower limb stability with ACL impairment. *J Orthopaedic Sports Physical Therapy* 1999; 29(2): 444-451.

43. Ross S, Guskiewicz K, Yu B. Comparison of time to stabilization measures in functionally unstable versus stable ankles. *J Ath Train* 2001; 36(2); S-76.

44. Shultz SJ, Perrin DH, Adams JM, Arnold BL, Gansneder BM, Granata KP. Assessment of neuromuscular response characteristics at the knee following a functional perturbation. *J Electromyogr Kinesiol* 2000;10 (3):159-170.

45. Lephart SM, Henry TJ. The physiological basis for open and closed kinetic chain rehabilitation for the upper extremity. *J Sport Rehab* 1996; 5(1): 71-87.

46. Rose HM, Shultz SJ, Arnold BL, Gansneder BM, Perrin DH. Acute orthotic intervention does not affect muscular response times and activation patterns at the knee. *J Ath Train.* 2002; 37(2): 133-140.

47. Shultz SJ, Perrin DH, Carcia CR, Gansneder BM. Lower extremity alignment affects muscle activation patterns at the knee following a weight-bearing perturbation. *J Ath Train* 2002; 37(2): S-28.

48. Cauraugh, JH, Sangbum K. Two coupled motor recovery protocols are better than one. *Stroke* 2002; 33: 1589-1594.

49. Ross SE, Guskiewicz KM. Differences between ground reaction force measures of a static single limb stance and a single limb stance following a jump landing. *J Ath Train* 2003 38(2)S: S-24.

50. Bell DR, Sander TC, Gansneder BM, Shultz SJ. Posterior trunk position increases reflex amplitude at the knee in response to a perturbation. *J Ath Train* 2003; 38(2)S: S-103.

51. Nosaka K, Clarkson PM. Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Med Sci Sports Exerc* 1996; 28(8): 953-961.

52. Nosaka K, Sakamoto K, Newton M, Sacco P. The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. *Eur J Appl Physiol* 2001; 85: 34-40.

53. Smith LL, Fulmer MG, Holbert D, McCammon MR, Houmard JA, Frazer DD, Nsien E, Israel RG. The impact of a repeated bout of eccentric exercise on muscular strength, muscle soreness and creatine kinase. *Br J Sp Med* 1994; 28(4): 267-271.

54. Solomonow M, Baratta M, Zhou BH, Shoji H, Bose W, Beck C, D'Ambrosia R. The synergistic action of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *Am J Sports Med* 1987; 15: 207-213.

55. Ebig M, Lephart SM, Burdett RG, Miller MC, Pincivero DM. The effect of suddent inversion stress on EMG activity of peroneal and tibialis anterior muscles in the chronically unstable ankle. *J Orthop Sports Phys Therapy* 1997; 26(2): 73-77.

56. Isakov E, Mizrahi J, Solzi P, Susak Z, Lotem M. Response of the peroneal muscles to sudden inversion of the ankle during standing. *International J Sport Biomech* 1986; 2: 100-109.

57. Johnson MB, Johnson CL. Electromyographic response of peroneal muscles in surgical and nonsurgical injured ankles during sudden inversion. *J Orthop Sports Phys Therapy* 1993; 18(3): 497-501.

58. Hoffman M, Schrader J, Koceja D. An investigation of postural control in postoperative anterior cruciate ligament reconstruction patients. *J Ath Train* 1999; 34(2): 130-136.

59. Hertel J, Gay MR, Denegar CR. Differences in potural control during single-leg stance among healthy individuals with different foot types. *J Ath Train* 2002*;* 37(2): 129-132.

60. Hertel J, Denegar CR, Buckley WE, Sharkey NA, Stokes, WL. Effect of rear-foot orthtics on postural control in healthy subjects. *J Sport Rehab* 2001; 10: 36-47.

61. Ochsendorf DT, Mattacola CG, Arnold BL. Effect of orthotics on postural sway after fatigue of the plantar flexors and dorsiflexors. *J Ath Train* 2000; 35(1): 26-30.

62. Ross SE, Guskiewicz KM. Time to stabilization: A method for analyzing dynamic postural control. *Ath Therapy Today* 2003; 8(3): 37-39.

BIOGRAPHICAL SKETCH

I was born on June 11, 1975, to David James and Eve Ann Smink (Rodman) in Shamokin, PA. Shamokin is a small coal-mining town in east central Pennsylvania. This is where I spent my entire youth growing up with my older brother Keith. When I was in fourth grade, I experienced the first tragedy of my young life. My father was taken from us in an automobile accident. This was a difficult time in our lives, but we had friends and family to help us through.

My mother has worked hard throughout her life to provide for us and I love her immensely for that. She has been the most supportive person in my life, sacrificing herself to ensure I would not falter on my own. She has taught me much about respect, honesty, and the importance of family. While growing up a stubborn young boy, I did not always adhere to her sound advice, ignorant to the fact she had many similar life experiences to draw from. It was not until she met my stepfather that I truly began to understand what it would take to become a man. He has taught me many life lessons, such as hard work, dedication, and patience. Without this man, I truly do not think I would be where I am today.

My education began in the Shamokin area public school system where I graduated from high school in 1993. Since I had not chosen a career goal, I chose to work for a year before attending college. It was during this time that I had an accident while playing basketball that changed my life forever. During the summer of 1993, I tore the anterior cruciate ligament in my left knee, which required surgery to repair. Following the

surgery and rehabilitation process, I realized that my career goal was to become a physical therapist so I could help others who were in similar situations. The next two and a half years, I attended the University of Pittsburgh at Bradford studying sports medicine. It was at that point that I realized I no longer had the desire to work in a physical therapy clinic and that I wanted to work as an athletic trainer. I immediately began researching schools in the area with accredited undergraduate programs and came across Lock Haven University. Upon being accepted, I then applied for acceptance into the athletic training education program, which I was denied after my first year. Unaffected, I continued to work hard and study. The following year I reapplied and was accepted to begin classes in the curriculum. The next two years were an exciting time for me. I was assigned to work with the university football, volleyball, and track & field teams as well as with a local high school. While working closely with my program director, I once again had a change of goals I needed to achieve. I wanted to learn more about the research process, but in order to achieve this goal, I would need to continue my education towards a master's degree.

I applied to several universities and was thrilled to accept an assistantship position from the University of Florida. I began my graduate education in the fall of 2001. During my first year, I was assigned to work with a local private school, Oak Hall. I thoroughly enjoyed working there and met many wonderful people. The summer between my first and second year in Gainesville is when I realized that I wanted to give back to the students. I assisted teaching a lab section for the undergraduate students and it was at that point I made a decision that I wanted to become an instructor in the athletic training field. Throughout my second year, in which I worked at Gainesville High

School, I continued to teach numerous lab sessions in the AT department. Based on this newfound desire to teach, I decided to apply to several universities in an attempt to continue my education towards my PhD.

I am eager to graduate from UF so I may pursue my next aspiration. I will be enrolling at the University of Delaware in the fall of 2003 in the Biomechanics and Movement Science department pursuing my doctoral candidacy. My life has taken many twists and turns over the years, but now I feel I am on track to attain my final goal.