REACTIVE KNEE STIFFNESS STRATEGIES BETWEEN VARIOUS CONDITIONING HISTORIES

by

David Craig Oates II

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789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 - 1346

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Approved: _____

Charles Buz Swanik, Ph.D.
Professor in charge of thesis on behalf of the Advisory Committee

Approved: _____

William B. Farquhar, Ph.D.
Chair of the Department of Kinesiology and Applied Physiology

Approved: _____

Kathleen S. Matt, Ph.D.
Dean of the College of Health Sciences

Approved: _____

James G. Richards, Ph.D.
Vice Provost for Graduate and Professional Education

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ABSTRACT

Context: Throughout functional performance, optimizing joint stiffness through appropriate muscular activation is crucial for maintaining stability and preventing injury such as ACL rupture. Various conditioning techniques may serve to affect joint stability through neurophysiologic mechanisms that may increase joint stiffness, or improve the ability to absorb loads. Understanding the strategies by which individuals with varying conditioning histories regulate knee stiffness would be crucial for designing training programs to optimize joint stability and prevent injury. **Objective**: To assess if knee stiffness strategies differ in participants with various conditioning histories. **Design**: Post-test only with repeated measures. **Setting**: University laboratory. **Patients or Other Participants**: 15 endurance athletes (19.8±1.1 yrs, 65.9±8.8 kg, 176.6±8.6 cm), 12 power athletes (20.3±1.3 yrs, 74.5±10.3 kg, 175.5±4.9 cm), and 15 control subjects (20.1±1.6 yrs, 79.0±12.8 kg, 177.0±4.3 cm) with no previous knee injury participated in this study. **Interventions**: Participants were seated on a custom stiffness device that generated a rapid 40° flexion perturbation to the knee (30° to 70° flexion arc) and instructed to either maintain muscle contraction or relaxation, resist the perturbation, or reactively relax. Muscle activation was recorded through electromyography from the quadriceps and hamstring muscles. **Main Outcome Measures**: Stiffness values (Nm/deg/kg) at short range (0-4°) and long range (0-40°), and muscle activation amplitude (%MVIC) and timing (ms) were compared across groups and conditions using analyses of variance. **Results**: Stiffness results revealed that passive short-range stiffness was greater in the

endurance athletes (0.057±0.012 nm/°/kg) than the control group (0.047±0.008 nm/°/kg, p=0.021); while passive long-range stiffness was greater in the power athletes (0.0020±0.001 nm/°/kg) than the endurance athletes (0.0016±0.001 nm/°/kg, p=0.016). Active-reactive long-range stiffness was greater in the endurance athletes (0.051±0.017 nm/°/kg) than both the control (0.033±0.011 nm/°/kg, p=0.001) and the power (0.037±0.015 nm/°/kg, p=0.044) groups. Endurance athletes also displayed greater peak EMG in their quadriceps during the passive condition ($p < 0.050$) compared to power and controls. The endurance group all had increased area EMG area under the curve (AUC) for their quadriceps than both power and control groups during the PRE time period ($p < 0.050$) for the passive reactive condition. No other significant EMG and stiffness differences were found. **Conclusions**: This study's findings indicate that a power-based training history may help increase passive joint stiffness, while endurance-based training can improve reactive muscular characteristics after knee perturbation. Both training groups displayed potential alterations in preparatory and reactive muscle activation and stiffness regulation, with either regimen possibly improving knee joint stability and preventing injuries. However, in cases of patients with increased laxity, endurance training may be beneficial to muscle tone and subsequent joint stiffness; conversely, power training may potentially facilitate the deactivation of certain muscles that can negative affect joint stability.

Key Words: anterior cruciate ligament, athlete, power, endurance

Chapter 1

INTRODUCTION

Coordinated movement is dependent on an individual's ability to detect joint loads and generate an appropriate muscle contraction such that injury may be avoided. This *neuromuscular control* is dependent on afferent feedback of joint force and position from peripheral mechanoreceptors (*proprioception*) and integration in the central nervous system contributing to an efferent muscular response and subsequent change in joint stiffness.[1,2,3] Disruptions in neuromuscular control affecting joint stiffness have been observed among subsets of injured and functionally unstable joints[4,5,6], but limited research exists regarding training and conditioning techniques that potentially enhance this process and may contribute to the prevention of injuries such as ruptures of the anterior cruciate ligament. Additionally, while higher stiffness was originally thought to contribute to greater joint stability, evidence has emerged that absorption of load, or joint compliance may be more beneficial for preventing injury.[5,7] By understanding how athletes with varying conditioning histories emphasizing fast- or slow- twitch fiber dominance differ in their ability to sense joint loads and react to joint perturbations clinicians, may be able enhance prevention and rehabilitation efforts.

Joint stiffness, the resistance of a joint to changes in displacement[8], is a key factor in optimizing performance and the prevention of injury. Three important components contribute to this property at each joint. Static stabilizers formed by capsuloligamentous structures provide a degree of innate joint stiffness[1,9,10], although

these structures alone are inadequate to absorb higher levels of forces associated with vigorous physical activity.[9] Therefore, dynamic restraints will provide stability through both reflexive muscular responses, and volitional muscle contraction, which are capable of modifying joint stiffness in preparation for, and in response to, potentially injurious perturbations.[10] While muscle contraction will yield a proportional increase in joint stiffness, it remains unclear if this is most beneficial for optimizing joint stability and preventing joint injury. Recent evidence has suggested that while increased muscular contraction may improve the resistance to injurious loads[11,12], compliance may allow the same forces to be absorbed, allowing for the dissipation of energy through muscle lengthening.[5] Several researchers have suggested that stiffness optimization may be *task dependent* so it remains unclear whether greater stiffness or compliance is best for injury prevention.[13,14,15]

A key factor controlling joint stiffness is the ability of the nervous system to continuously integrate sensory information and develop motor responses. This neuromuscular control depends on two distinct components: preparatory (feed-forward) and reactive (feedback) control.[3,16] The combination of these mechanisms form a synergistic neuromechanical relationship that continuously regulates joint stiffness in preparation of loading (feedforward)[3,17,18], and in response to unanticipated perturbations (feedback).[19] Neuromuscular control thereby represents cyclic activity whereby current levels of muscular output are sensed and modified based on previous and future events. Therefore a key component to its regulation comes from the ability of the joint mechanoreceptors to transmit afferent information regarding joint position, movement, and force (proprioception) to the central nervous system.[1,10] For example, a rapid stretch of the joint will generate an afferent signal from the muscle spindle,

which would subsequently promote a muscular contraction through the monosynaptic stretch reflex.[17,20,21] Alternately, increased load on the joint would activate the Golgi tendon organ (GTO), which through polysynaptic reflexes will cause inhibition in the antagonistic muscle.[17,22,23] Aside from these spinal reflexes, afferent activity will ascend to the central nervous system, and facilitate the formation of appropriate volitional responses to changes in joint motion. Deficits in neuromuscular control are therefore often attributed to altered proprioceptive acuity of these sensory organs, and consequently, proprioceptive tasks such as joint angle reproduction or kinesthetic awareness have been employed by researchers to understand changes across injured populations. During any functional task such as running or cutting, a load is placed on the joint that the neuromuscular control system must adequately predict and match using muscles along the kinetic chain. These forces must be accurately detected in order for controlled columnar buckling[7] to occur. If the forces are not appropriately detected, an individual may attempt to contract with too much force leading to an error in coordination, or experience an uncontrolled buckling, such that abnormal forces in the frontal and coronal planes are placed on the joint. Currently it is unclear how this concept of columnar buckling and appropriate force detection relates to conditioning history, joint stiffness and unanticipated perturbations under a controlled laboratory setting.

As an individual's ability to regulate joint stiffness and neuromuscular control during unanticipated loading may be influenced by various factors, one important and potentially modifiable variable is conditioning history. The type of training an individual performs may predict whether more fast- or slow-twitch muscle fibers are present, and may have important implications in the prevention and treatment of joint

injury. Slow twitch (Type I) muscle fibers' growth is facilitated mainly through focus on high repetitions and low loads commonly seen in endurance athletes.[24] Fast twitch (Type II) muscle fiber adaptations are promoted through training focused on low repetitions and high loads, commonly seen in power athletes. Structural and performance differences exist in fiber type, with Type II fibers showing a higher intrinsic speed of contraction, larger peak power, and quicker fatigue than Type I fibers.[25,26] Previous research examining stiffness differences between power and endurance athletes have been inconclusive, as power athletes appear to have increased stiffness compared to endurance athletes, but the source of these changes is unclear.[27,28,29,30] Much confusion in this topic originates from the methodological tasks participants underwent, which were largely functional and did not control for variables that may influence the results.

Altered joint stiffness and sensorimotor function has been associated with an increased risk of injury, but little is known regarding how conditioning may affect these factors, as well as the individual's ability to detect and replicate force. While limited research has examined differences in joint stiffness between power and endurance athletes, these studies have not provided the necessary experimental control to understand how these differences occur, and how they may be implemented in injury prevention and treatment protocols. Therefore the specific aims of this study were:

1. To determine the effects of four **knee perturbation conditions** (passive, active, reactive, deactive) on joint stiffness and muscle activation.

H1: Each knee perturbation condition and requisite task goal would differ in the level of stiffness and muscle activation strategies used to optimize stiffness

qualities, such as increased EMG pre-activation and a greater stiffness during reactive conditions.

2. To investigate if **group differences** existed in knee stiffness regulation and muscle activation strategies (timing and amplitudes of muscle EMG) between power athletes, endurance athletes, and non-athlete controls during knee perturbations.

H 2: Athletes would have increased knee stiffness values when compared to control subjects, specifically in that:

H2.1: Athletes would have differing muscle activation strategies compared to control subjects as demonstrated by increased short-range stiffness and both greater and faster muscular activation.

H2.2: Power athletes would have a greater ability to activate/deactivate muscles quickly as observed through a faster deactivation of quadriceps EMG activity. Endurance athletes would have a greater peak EMG and overall increased muscle amplitude.

H2.3: Power athletes would also have increased knee stiffness values compared to endurance athletes.

3. To determine the **interaction effect** of **group** conditioning history on the ability to prepare and react to each type of **perturbation condition**

H 3: Each group would differ in their muscle activation strategies used to optimize stiffness qualities based on the knee stiffness perturbation condition and task goals

H3.1: Power athletes would have greater responses to testing conditions that were biased towards either reactive movements or quick deactivation of the knee musculature when compared to control subjects and endurance athletes.

H3.2: Endurance athletes would have greater responses to passive testing conditions and those conditions that require preparatory activation for movements when compared to control subjects and power athletes.

Chapter 2

METHODS

2.1 Participants

Based on an *a priori* power analysis ($\alpha < .05$, $\beta = 0.8$) using previous knee stiffness research[31], a conservative estimate of 13 subjects per group was used for this study. 42 subjects were collected and used for this study. Subjects were male, healthy participants between 18-25 years age range and recruited from the University of Delaware population. Power athletes were recruited as division I collegiate track sprinters. Endurance athletes were recruited as division I collegiate cross country runners. Controls were recruited as healthy and active subjects, with active being defined as exercising at least twenty minutes a day three to four times a week. Participants in the study were recruited on a volunteer basis. Methods of recruitment involved word of mouth as well as visitations to the sports teams and classes within the Department of Kinesiology and Applied Physiology at the University of Delaware. Participants were excluded from the study if any of these conditions apply: (1) any fractures to the test leg within the last year, (2) history of knee ligament reconstruction surgery, (3) other knee injuries requiring surgery, (4) any current (within the last month) bone, muscular, or joint injuries to the hip, knee, and ankle, and (5) any cardiovascular, metabolic or neurological problems that would limit moderate physical activity.

2.2 Instrumentation

2.2.1 Stiffness and Proprioception Assessment Device

Testing was performed using the custom-build stiffness and proprioception assessment device (SPAD) (Appendix A). The SPAD is a brushless Danaher/Kollmorgen servomotor (B-404-B-B4) fitted into a gearbox (UT018-050, 50:1) that is connected to an amplifier/controller (Copley Xenus driver XSL-12-36-R). The amplifier (input: 230 VAC, 3PH, output: 18 FLA, 50/60 Hz) is mounted ~4ft above the ground and connected to the motor with a 12-foot long feedback cable (Model # CEF-RO- 006-900, Pacific Scientific, Rockford, IL) and also connected to a personal computer through a Kvasar CAN cable. The controller receives a three-phase, 240-volt, 30-amp enclosed I-T-E switch power supply through a power cable (Model # CEP-A6-006-904, Pacific Scientific, Rockford, IL). The mated servomotor and gearbox are mounted in a cast aluminum pedestal that is offset from the subject's chair. An adaptor arm and torque reaction sensor (Model # T5400, Futek Advanced Sensor Technology, Irvine, CA) with a 565 N capacity and 1.43 X 10^5 ft-lb/rad torsional stiffness was coupled to the gearbox. Signals from the torque reaction sensor passed through a conditioner (Model # D502, Futek Advanced Sensor Technology, Irvine, CA) at 60 Hz and have a 0 to 10 Vdc analog output range. The signal conditioner digitally displayed torque values and also sends an analog torque signal through a BNC box so that is can be recorded and displayed in LabVIEW software.

For safety purposes, internal motor settings could not exceed preset speeds and there were three emergency stop switches that were able to disable the motor during testing. The operator and test participant each held an emergency stop button. When depressed, the motor was disabled. Also, if the adaptor arm moved

within the last 5% of the participant's end range of motion a proximity sensor (#S4602896, Turck Inc. USA, Minneapolis, MN) disabled the motor. When the motor was disabled, the operator had to re-start the power supply to continue tests. The LabVIEW motor control software also had "soft limits" which disabled the motor amplifier if motion exceeded individualized motion limits. The SPAD was also fitted with mechanical stops to limit motion through an adjustable range and brass screws in the adapter arm flange connected to the gear box which would fail under excessive torque. The SPAD device was operated using a personal computer with a customized LabVIEW virtual instrument and motor control software program.

2.2.2 Electromyography

Surface electromyography (EMG) was collected from the vastus medialis, vastus lateralis, medial hamstrings (semitendinosis), and lateral hamstrings (biceps femoris) to determine stiffness regulation strategies by analyzing the amplitude and timing of muscular contractions.[2] Self-adhesive Ag/AgCl bipolar surface electrodes (Phillips Medical Systems, Andover, MA) were used with a wired telemetered EMG unit (Bortec AMT-8, Bortec Biomedical, Calgary, Alberta, Canada) to record EMG with a real-time visual display on the monitor. Electrode placement was identified by bony landmarks and through palpation of the mid-belly of the contractile component of the muscle during an isometric contraction.[32] The reference electrode was placed on the patella. Each electrode is 10mm in diameter and was placed 25mm apart. The electrode placement site was shaven, abraded, and cleansed with an alcohol swab (70% ethanol solution) to decrease the impedance from the skin. The signal was converted from analog to digital data with an A/D card, and then passed to a computer where the raw EMG data was synchronized with position and

torque and sampled at 2,400 Hz and further analyzed with LABVIEW software (National Instruments, Austin, Tx). The EMG signal was bandpass filtered at 20-400Hz, rectified, and low-pass filtered at 5Hz to create a linear envelope. EMG was normalized to maximum voluntary isometric contractions (MVICs) for the muscles of the quadriceps and hamstrings.[2] All EMG data equipment was used to determine the timing, sequence, amplitude (peak & area), and pattern of the quadriceps/hamstrings muscle groups during preparatory and reactive phases of the stiffness testing.

2.3 Procedures

Participants were asked to report to the Human Performance Lab for testing. They completed the University of Delaware IRB [156420-5] approved consent form as well as the Physical Activity Readiness Questionnaire to determine participant eligibility for the study (Appendix B). Participants answered questions pertaining to the exclusionary criteria. If the participant answered "Yes" to any questions pertaining to pains in the heart or chest, faintness or dizziness, bone or joint problems, or low back problems, he was unable to participate in the study. The accepted participants rode a stationary bike for a 5-minute warm-up followed by 5 minutes of stretching of their quadriceps and hamstrings, provided with a handout and verbal instructions (Appendix C). Both stretches were held for 30 seconds, repeated 3 times. The same stretching sequence was completed once the testing is completed.

2.3.1 Knee Stiffness Testing

Knee stiffness was tested next by applying a flexion perturbation to the knee joint. The perturbations for each testing condition remained constant, with a quick acceleration to a velocity of 100^{o}/sec and a flexion arc of 40^{o}. The testing conditions were defined based on the amount of muscle activation *prior* to the perturbation, and

the instructions on how to react to the perturbation. The conditions included relaxed non-reactive (PS), relaxed reactive (PRS), 85% quadriceps MVIC non-reactive (AS), 85% quadriceps MVIC reactive (ARS), and 85% quadriceps MVIC deactivation (DS). The testing conditions and explanations are listed in Table 1. During the PS trials, the subject were instructed to completely relax their quadriceps and hamstrings, and instructed to remain completely relaxed throughout the entire perturbation. During the PRS stiffening trial, the subject were instructed to contract as hard and as fast as they can once they began to detect movement of the leg. During the AS trial, the subject was instructed to maintain a constant extension contraction at 85% of their MVIC, and instructed not to intervene with the perturbation while attempting to maintain the 85% of their MVIC. For the ARS trial, the subject was again instructed to maintain the 85% MVIC contraction, except that during this trial the subject was instructed to react and contract as hard and as fast as they could once they sensed the perturbation. For the DS trial, the subject was instructed to maintain the 85% MVIC contraction, except that during this trial the subject was instructed to relax and deactivate their quadriceps muscles when the perturbation occurs as quickly as they could. All perturbations during stiffness testing were applied randomly during a 10-second window to avoid subject anticipation. Pre-activation levels were monitored in real-time through EMG and a digital torque sensor. If a subject failed to relax, the trial was repeated. Three trials were collected for each condition, with short rest periods of 1 minute between repetitions to avoid fatigue. Analog signals for torque, position, and EMG were collected, along with stiffness calculations, by a custom software program using LabView 2010 (National Instruments, Austin, TX).

2.3.2 Data Reduction and Analysis

All signals and data were analyzed with a separate LabView 2010 program. Stiffness values were calculated according to the position data at 4° and 40° of the flexion perturbation. EMG was averaged over the three trials for each of the five conditions (PS, PRS, AS, ARS, DS). Stiffness values were normalized to the subject's mass to allow for between-subject comparisons. EMG was analyzed for a window of 150-ms prior to the perturbation and 500-ms after the initiation of the perturbation. EMG was normalized to the maximum voluntary isometric contractions (MVICs) of the hamstrings and quadriceps.[32] The EMG dependent variables collected and analyzed were electromechanical delay (EMD), peak EMG torque, time to peak (TTP) torque, pre-perturbation area [PRE] (150ms prior to perturbation), reflexive area [POST-1] (0-250ms post-perturbation), and further reflexive area [POST-2] (250-500ms post-perturbation).

2.3.3 Statistical Analysis

Data was analyzed using custom LabVIEW software programs as well as SPSS statistical software. A three-way repeated-measures ANOVA with one between-subject factor (Group, 3 levels), and two within-subject factor (Condition, 5 levels; Range, 3 levels) was used to determine if differences exist in all stiffness and EMG dependent variables Pairwise comparisons were used for post-hoc analysis when appropriate. The alpha level was set *a priori* at p=0.05.

Chapter 3

RESULTS

3.1 Demographics and Maximum Voluntary Isometric Contractions

Demographic data for age, height, mass, and peak MVIC torque are displayed in table 2. Endurance athletes were significantly lighter than controls, while no other demographics were different across groups ($p < 0.050$).

3.2 Stiffness

Results for stiffness testing are shown in table 3. A significant group by condition by range interaction effect was observed ($F_{8,156}=2.52, p=0.013$). Pairwise comparisons revealed short-range stiffness was greater than long-range stiffness across all conditions ($p < 0.050$). For the different kinds of stiffness testing conditions, all groups had greater short-range stiffness in the pre-activated conditions (AS, ARS, and DS) than in the conditions that were relaxed to start (PS & PRS) ($p <0 .050$). The short-range stiffness was greater in the endurance group than the control group ($p=0.021$) under the passive condition. For long-range stiffness similar patterns were observed across all groups, with PRS having the highest long-range stiffness, followed sequentially by ARS, AS, PS, and lastly DS ($p < 0.050$). Long-range stiffness was higher in the power group compared to the endurance group ($p=0.016$) in the passive condition. For the active-reactive condition, the endurance group had higher long-range stiffness than the control group ($p=0.001$) and power ($p=0.044$) groups.

3.3 EMG

3.3.1 Peak Muscle Activity

Peak EMG means are found in table 4. A significant muscle, by condition, by group interaction effect was observed ($F_{24, 468} = 2.219$, $p = 0.001$). The endurance group had greater peak EMG in the VM during ARS when compared to the control group ($p = 0.010$), and also displayed greater peak EMG in the VL compared to power and control groups during PS ($p < 0.050$). The control group had greater peak LH than the power in the AS, PRS, and ARS conditions ($p < 0.050$), while also exhibiting greater peak LH than the endurance group during AS ($p = 0.033$). The endurance group displayed greater peak EMG for both VM and VL compared to MH and LH across all conditions ($p < 0.050$). For the power group, they displayed greater peak EMG for both quadriceps compared to hamstrings for all conditions except PS ($p< 0.050$). For the control group the quadriceps muscles had greater peak EMG than the hamstrings for AS, PRS, ARS, and DS conditions ($p < 0.050$). They also had greater peak EMG for LH compared to MH for AS, PRS, ARS, and DS conditions ($p < 0.050$). Controls also displayed greater LH peak EMG compared to VM for PS ($p = 0.021$).

3.3.2 Time to Peak

Time-to-peak means are found in table 5. There was a significant muscle by condition interaction effect ($F_{12, 420} = 2.988$, $p = 0.001$). In VM and the VL muscles, time-to-peak was shortest in DS ($p < 0.050$). For the MH and LH, time-to-peak was longest in PS ($p < 0.050$) except for the LH in the PRS condition. In PS, the MH had longer time-to-peak than the VM ($p = 0.008$). In the PRS condition, both quadriceps muscles (VM and VL) had longer time-to-peak than the LH ($p = 0.038$, $p = 0.044$).

During ARS, the VM had a longer time-to-peak than both MH and LH ($p < 0.050$). For DS, both the MH and LH had longer time to peaks than the VM and VL ($p < 0.050$).

3.3.3 EMG Area Under the Curve (AUC)

3.3.3.1 Passive Stiffness Condition

Area under the curve (AUC) values are located in table 6. For PS, there was only an effect of time ($F_{2, 78} = 93.045$, $p < 0.001$), with POST-1 being greater than the PRE and POST-2 ($p < 0.001$) and the POST-2 being greater than the Pre ($p < 0.001$). There was no effect of group ($F_{2,39} = 1.566$, $p = 0.222$).

3.3.3.2 Active Stiffness Condition

AS values are located in table 6. For the active condition there was a time by muscle interaction effect ($F_{6,234} = 12.281$, $p < 0.001$). Both quadriceps and hamstring groups had greater muscle activation during POST-1 then both the PRE and POST-2 periods ($p < 0.050$), while each muscle's POST-2 period was greater than the PRE period ($p < 0.050$). For all the time periods, there was a trend of greater muscle activity for the VM and VL compared to the MH and LH groups ($p < 0.001$), while also having great activity in the LH than the MH ($p < 0.001$).

3.3.3.3 Passive Reactive Condition

Values of PRS muscle activity are located in Table 6. There was a significant interaction effect of muscle by group by time ($F_{12,234} = 1.902$, $p = 0.035$). During PRE the endurance group had greater VL activity than both the power and control groups ($p < 0.050$). During POST-1, the endurance group had greater VM muscle activity than the control group ($p = 0.037$) and the control group had greater LH activity than the power group ($p = 0.050$) during the POST-2 period. In both the VM and VL muscles,

all groups increased from PRE to POST-1, and from POST-1 to POST-2 ($p < 0.050$). For the MH, all groups had greater muscle activation in the POST-1 and POST-2 periods than during PRE ($p < 0.050$); however in the LH the endurance and power groups had greater activation during POST-1 and POST-2 than PRE ($p < 0.050$) while the control group had POST-2 greater than POST-1, which was greater than PRE ($p < 0.050$). During PRE the endurance group had VL activation greater than VM ($p = 0.045$) while the control group had the greater LH activation than the VM ($p = 0.022$). In POST-1 & POST-2 the endurance and power groups had greater quadriceps activation than the hamstrings ($p < 0.050$); however VL & MH was equal at POST-2 for controls.

3.3.3.4 Active Reactive Condition

Values of active reactive muscle activity are located in table 6. There was a significant time by muscle by group interaction effect ($F_{12,234} = 3.275$, $p < 0.001$). For all muscles, activity was greatest POST-2 and POST-1 than PRE ($p < 0.050$), although endurance athletes had VL activity increasing from POST-1 to POST-2 ($p = 0.038$), while power and control groups decreased LH activity from POST-1 to POST-2 ($p < 0.050$). For all 3 time periods quadriceps had greater muscle activation than the hamstrings ($p<0.050$), except for the control group in the PO..ST-1 period, in which VM was not greater than LH ($p < 0.050$). During PRE, the control group had greater LH activity than MH ($p < 0.001$). In both POST-1 and POST-2 the endurance and control groups have greater LH activation than MH ($p < 0.050$). During PRE, the control group had greater LH activation than the endurance group ($p = 0.043$). During POST-1 both endurance and power had increased VM activation compared to controls ($p < 0.050$), while the controls had increased LH activation compared to endurance

power groups ($p < 0.050$). During POST-2 the endurance group that increased VM activation compared to controls ($p = 0.017$), while controls had greater LH activation than the power group ($p = 0.040$).

3.3.3.5 Deactive Condition

Values are located in Table 6. There was an effect of time ($F_{2, 78} = 183.185$, $p < 0.001$), while also having an interaction effect of muscle by group ($F_{6, 117} = 2.862$, $p = 0.012$). For time, POST-1 was greater than PRE and PRE was greater than POST-2 ($p < 0.001$). For the muscle by group, the endurance group had a greater VL activation than the power group ($p = 0.040$). For both the endurance and power groups, their quadriceps muscles had greater activation than the hamstring muscles ($p < 0.050$), with the power group having greater VM activation than the VL ($p = .047$). The control group had greater VM and VL activation than MH ($p < 0.001$), but also greater LH activation than MH ($p < 0.001$).

3.3.4 EMD

All EMD values are located in table 7. There is an effect of muscle on EMD ($F_{3, 108} = 14.889$, $p < 0.001$), where both hamstrings had greater EMD than the quadriceps ($p < 0.050$).

Chapter 4

DISCUSSION

The present study aimed to determine if conditioning history affects joint stiffness and muscle activation strategies during a knee perturbation. The ability to properly regulate joint stiffness through appropriate activation of stabilizing muscles is crucial for functional movements and injury prevention, such as non-contact anterior cruciate ligament ruptures.[33] Little evidence exists regarding the role of conditioning histories on precise, neuromechanical measures of joint stiffness regulation.[27,29] Understanding this relationship will lend valuable insight into potential rehabilitation and conditioning techniques that might optimize function and joint stability. Our primary findings indicated that a power-based training history increased passive joint stiffness, while endurance-based training improved reactive muscular characteristics after knee perturbation.

4.1 Stiffness Regulation

The ability to appropriately regulate the resistance of a joint to external load is vital to functional stability. This joint stiffness is dependent on the biomechanical stiffness properties of the individual structures surrounding the joint including capsuloligamentous, cutaneous, and musculotendinous structures.[1,9,10] Of these, stiffness of the musculotendinous unit may be most crucial as it is highly modifiable through both voluntary and involuntary contraction, and physiologic changes to the muscle.[10] Conditioning history has been found to affect multiple variables related to performance and joint stability among athletes. Training using higher loads and low

reps (power training) contributes to increased formation of Type II muscle fibers and increased muscular power[25,26]; while training involving increased repetitions of lower loads (endurance training) is associated with proliferation of Type I muscle fibers and improved running economy.[24,34]

In our study, we attempted to understand the role of conditioning history on joint stiffness through precise measurements of the resistance to a knee flexion perturbation, across various ranges (short-range versus long-range) and muscular reaction conditions. As expected, we observed that short-range stiffness was consistently higher than long-range stiffness[35]; and the test conditions requiring muscular pre-activation (AS, ARS, DS) displayed higher short-range and long-range stiffness compared to passive conditions.[36] These findings are consistent with previous research.[31,37] Short-range stiffness is regulated by the joint's static restraints, the elastic components of the muscle, and notably, the reverse-pivot of bound actin-myosin cross-bridges[35,36], while long-range stiffness relies on the cycling of cross-bridges throughout an eccentric contraction.[36] Accordingly, it would then be expected that heightened muscular activation would increase the number of bound actin-myosin cross-bridges, thereby increasing short-range stiffness. Interestingly, long-range stiffness was highest in the passive-reactive condition where subjects were asked to react maximally from a relaxed state. Stiffness has previously been seen as highest in the active-reactive condition[38]; however, we utilized a higher level of pre-contraction, potentially diminishing the ability of reflexes to assist with agonist recruitment.

Group differences were most notable in the passive condition, as short-range stiffness was highest among endurance-trained athletes; while long-range stiffness was highest in power-trained athletes. During the passive condition, resting

muscle tone may have an immediate effect on stiffness through the initial range of motion. Power-trained athletes have previously displayed higher stiffness at the knee[27,28] and ankle[27,28,30] joints during both perturbations and functional movement.[27,28,29,30] However, the previous studies quantified stiffness indirectly by calculating the dampening effect of the muscle from subjects' hopping frequencies[27]; or by using kinematic and kinetic data to estimate joint load as position changes.[28,29,30] The SPAD utilized in our study allows for a more precise, albeit less functional perturbation, which provides valuable information as to the source of stiffness differences.[31,37] Higher short-range stiffness in endurance athletes may be indicative of changes to the static joint restraints, as well as heightened fusimotor regulation of muscle spindles and the resulting muscle tone. No studies have attempted to definitively quantify changes in muscle tone among these individuals. Muscle tone has been described as a "state of readiness" of the muscle that would result in an increased amount of linked actin-myosin cross-bridges at rest.[6,39] Alternately, the increased passive stiffness in power athletes may be secondary to either a heightened reflexive response, or greater passive stiffness characteristics in the series and parallel elastic components of the muscle. The protein titin has been theorized to contribute to this greater stiffness as well, but research has yet to confirm its direct contribution.[40] One additional explanation of these differences may come from the nature of these sports. Cross-country runners recruited for the endurance-trained group would typically use a smaller range-of-motion, consisting of largely eccentric movement during activity, whereas sprinting athletes used in the power-trained group would utilize a greater range of motion, largely concentric, to propel themselves forward.[41,42] Utilizing

smaller perturbations might be ideal for determining whether differences originate in muscle tone, range-of-motion, or both.

Stiffness was also observed to be significantly different between groups in the active-reactive trial, with endurance athletes displaying higher long-range stiffness than power athletes and controls. Additionally, while stiffness was lower in the active-reactive trials than passive-reactive trials for power and control subjects, endurance athletes displayed similar stiffness values. This difference might be explained by the manner in which pre-activation was achieved (85% of maximum contraction). In the presence of volitional muscular pre-activation and response, long-range stiffness is almost entirely regulated by the cycling of cross-bridges through eccentric loading.[36] However, with a high degree of pre-contraction, it is possible that power athletes and controls had a more difficult time recruiting additional motor units throughout a perturbation, and may have experienced a degree of fatigue. It would be expected that fatigue would be less in endurance trained athletes, secondary to a higher percentage of Type I muscle fibers.[43] Therefore, under our controlled laboratory setting, the endurance group had a greater ability to recruit additional motor units in response to a perturbation. This hypothesis may be confirmed through analysis of electrical muscle activity.

4.2 Muscular Activation

As previously mentioned, joint stiffness is regulated by resistance from cutaneous, capsuloligamentous, and musculotendinous tissue.[1,9,10] While each of these components possesses a degree of innate static restraint, modification of joint stiffness related to a task is achieved through activation or deactivation of the surrounding musculature.[10] Magnitude of muscle activation was highest in active-reactive

condition and lowest in the passive condition. Also, as expected, quadriceps muscles displayed greater activity than hamstring muscles in passive-reactive, active, and active-reactive conditions, due to their role as the primary restraint against a knee flexion perturbation.

Several differences in muscle activation were observed across groups, and these changes were notably task-dependent. Under the passive condition, subjects were asked to remain relaxed throughout the perturbation. However, the knee flexion perturbation was large and rapid enough to induce a reflexive response in many subjects. Although no differences were observed in AUC over time, endurance-trained subjects demonstrated the greatest peak vastus lateralis activation in response to the perturbation. Interestingly, although this group demonstrated higher short-range stiffness in this condition, the muscular activation would not be rapid enough to affect this value.[2,36,44] Rather it would affect long-range stiffness, which was higher in the power group. This supports the hypothesis that endurance athletes may have greater fusimotor gain and muscle tone, and therefore were more sensitive (reflexively) to a perturbation.

Previous research has examined neuromuscular differences in power and endurance athletes, and found that presynaptic inhibition is observed to be higher in endurance athletes limiting muscle tone; however, this is just one factor related to muscle tone. Fusimotor gain may also be mediated at the cortical levels.[45,46] However, it remains unclear why this did not translate into an increase in long-range stiffness, and may be explained by generally greater amounts of hamstring activation compared to power athletes (although not statistically significant), that could have decreased flexion stiffness throughout the perturbation.

Although the passive perturbation represents valuable insight into the role fusimotor regulation plays in knee joint stiffness, rarely during physical activity would muscle activation be absent. To address the effect of muscular pre-contraction without a reaction, subjects were asked to hold 85 percent of their maximum voluntary isometric contraction throughout a perturbation. Although this produced no differences between endurance and power-trained athletes, increased peak hamstring activation was observed in control subjects. Although quadriceps activation and knee stiffness remained equivocal to other groups throughout this condition, it appeared that healthy controls required co-contraction of the quadriceps and hamstrings to achieve similar joint stiffness. It is unclear whether this hamstring stiffness is beneficial or detrimental to the stiffness of control subjects. However, the effect of lower hamstring activity in trained athletes has been previously documented, as desensitization of antagonistic Golgi tendon organs is reported as a key adaptation to resistance training.[23,47,48] Although power training has been described as most effective to achieve this desensitization, we did not detect any differences in this condition between groups.

Passive-reactive trials were used to test the ability of these individuals to initiate a muscular contraction, in response to a sudden perturbation, from a resting state. No group differences were detected in peak and time-to-peak EMG, but endurance athletes demonstrated greater pre-activation in the vastus medialis than power athletes and controls. These findings provide further support towards the hypothesis of increased quadriceps muscle tone among endurance athletes. While instructions indicated that subjects should remain relaxed until they felt their knee move, endurance athletes slightly increased their muscle activation to facilitate a better

reactive response.[49,50] This is in agreement with previous literature, which found greater EMG activation and increased muscle pre-activation in endurance athletes compared to a power group during hopping.[27]

Some very notable differences were observed when subjects were asked to pre-contract their muscle to 85 percent of their MVIC and then react maximally to the perturbation. As previously stated, it was thought that this would represent a functional response to a knee perturbation, as some level of pre-activation would be present, with a reactive response superimposed after the perturbation. While previous studies have utilized a smaller degree of pre-contraction (30 percent of MVIC)[36,38], we opted for a higher degree of muscle pre-activation; this increased muscle activity was thought to more closely mimic high level athletic maneuvers that the subject's would typically perform.[9] In this condition, endurance athletes displayed greater VM activity at POST-1 (0-250ms post-perturbation) and POST-2 (250-500ms post-perturbation) than controls, and power athletes had greater VM activity at POST-1 than controls. This finding is consistent with our stiffness data where endurance athletes were able to produce the highest long-range stiffness throughout this perturbation. Although it was thought that the ability to react would be greatest among power-trained athletes, it is possible that the high level of pre-contraction caused a degree of fatigue that limited the ability to raise their quadriceps activity throughout the entire perturbation. However, it is important to note that both groups of athletes were capable of equally increasing their muscular activity at POST-1 compared to control, suggesting that either training may be beneficial in improving some degree of knee stability during the time-range when injury is most likely to occur (within 200ms).[2,36,44] Previous research has found that various forms of training (balance,

perturbation, plyometric) that are components of these forms of training may be beneficial to decreased deficits and potential reduced injury risk.[48,51,52,53]

Another notable difference that was observed throughout the active-reactive condition was increased LH activation among healthy controls. This group demonstrated the highest LH activity throughout the perturbation, as well as greater pre-activation in this muscle. Conversely, power athletes demonstrated the least LH activity at POST-2 during this condition. This supports findings observed across other conditions where controls appeared to require quadriceps-hamstrings co-activation to attempt to maximize their stiffness, while power trained athletes appeared best at de-activating this antagonistic muscle. The ability of the power athletes to better inhibit antagonistic muscles is consistent throughout the literature and is associated with desensitization of Golgi tendon organs.[23,47,48] However, it is unclear whether the co-contraction observed in control subjects is beneficial or detrimental. Prior investigations have indicated that quadriceps-hamstrings co-activation may be ideal for maintaining knee joint stability.[4,54] However, certain functional situation may call for optimization of joint stiffness that requires a deactivation of muscles instead of an absolute increase in contraction.

The final condition tested in our study was to ask subjects to reactively deactivate their muscle in response to a perturbation. Anecdotal evidence suggests that, in some situations, a controlled de-activation may be equally effective in dissipating injurious forces, rather than assuming greater stiffness is always better.[7] This "controlled columnar buckling" would require athletes to deactivate their muscle allowing the knee to absorb loads throughout the sagittal plane, as opposed to allowing loads to transmit to the frontal or coronal planes.[7] Although we hypothesized that a

desensitization of antagonistic mechanoreceptors would facilitate deactivation among power-trained athletes, our data only partially supported this finding. No differences in peak muscular activation were observed; however, greater VL activation was observed in endurance athletes when compared to power athletes throughout the perturbation. While lowest values were observed in power-trained athletes, they were not significantly different than controls. This data lends support to the hypothesis that this deactivation strategy may not be optimal among endurance-trained athletes. However, the clinical implications of this are unclear as all groups displayed equal stiffness values. Further research should explore if training type might alter which strategy (activation or deactivation) is best for optimizing joint stability.

4.3 Limitations

Several limitations may affect the interpretation of our findings. Primarily, our study only used athletes from one institution, and all subjects were males that competed in track or cross-country. It is unclear if inclusion of other sports that utilize more frontal plane motion would alter our findings. Further research may investigate power- and endurance-trained athletes across a variety of sports, including those that present with higher rates of knee joint injuries. Similarly, including only males makes it unclear if the same statistical trends may be observed for females. Multiple factors including quadriceps angle and menstrual cycle changes have been associated with altered knee joint stiffness and neuromuscular control among females[55,56,57,58] and it was our goal to exclude these as contributing factors; however, incorporation of this group would improve the strength of our findings. Finally, our purpose was to investigate the role of conditioning histories on stiffness to determine the advantages of power versus endurance-based training techniques for optimizing

joint stability. However, this study design does not account for innate differences that may have existed, leading athletes to pursue their respective sport or events. Future research should study how power-based and endurance-based training may change joint stiffness in groups of untrained controls.

4.4 Conclusions

4.4.1 Clinical Significance

The results from this study indicate that an individual's conditioning history can play a potential role in stiffness regulation strategies. Both conditioning groups displayed potential alterations in preparatory and reactive muscle activation and stiffness regulation that may be beneficial for dynamic restraint and joint stability. Therefore, either training regimen may be effective in improving knee joint stability and preventing injuries. However, in situations where a clinician may be rehabilitating a patient with increased laxity, endurance training may potentially have a beneficial effect on muscle tone and subsequent joint stiffness. Conversely, power training may potentially facilitate the deactivation of certain muscles that can negatively affect joint stability; although further research would be required to accurately determine the potential training effects.

4.4.2 Future Research

This study was the first to precisely measure joint stiffness and neuromuscular activation across various reaction conditions in power and endurance trained athletes. Directions for future research might investigate how these factors change across a wider range of athletes including alternate sports as well as females. Furthermore, while differences in these factors were observed across groups of trained versus untrained athletes, studies might aim to determine if training incorporating low

load, high repetition or high load, low repetition is able to alter joint stiffness and neuromuscular control among untrained controls; as well as following injuries such as rupture of the anterior cruciate ligament.

Chapter 5

LITERATURE REVIEW

Lower extremity injuries occur frequently in sport and exercise, with the knee being one of the most commonly injured joints.[59] The Centers for Disease Control (CDC) developed a report in 2006 that indicated that the number of arthroscopic procedures on the knee increased 49% from 1996 to 2006 and that approximately 984,607 arthroscopic procedures were performed on the knee in 2006.[60] The report also found the total number of orthopedic procedures performed in ambulatory surgical centers that were knee arthroscopies increased from 15% to 51%. One particular injury that has a high incident rate, and is commonly discussed, is an anterior cruciate ligament (ACL) sprain. ACL injuries occur at a rate of approximately 200,000 per year in the United States[61], with an average annual cost of approximately 2 billion dollars in surgery, rehabilitation, and time lost from work.[62] In addition to the financial cost, ACL tears, in concordance with other associated injuries, can lead to future debilitating conditions such as early onset osteoarthritis.[63] Seventy percent of ACL injuries occur in a non-contact manner, generally during a jump-landing, pivoting, or cutting maneuver.[64] Research into ACL injuries and prevention has had a large focus in identifying biomechanical risk factors that may contribute to an individual's likelihood for lower extremity injury.

Risk factors for lower extremity injury have been categorized into extrinsic and intrinsic factors. Extrinsic factors are defined as causes external to the body, including sport played[65,66], footwear[67], playing surface[68,69], prophylactic bracing[70,71,72], and

conditioning history.[24] Intrinsic factors, defined as being internal to the body, include hormonal[73], anatomical[33], biomechanical, and neuromuscular factors.[33] The biomechanical and neuromuscular factors are ones of particular interest, due to the potential of modification through therapeutic exercise and conditioning programs.

Joint Stability

Joint stability is important during performance of daily activities and rigorous sport competition. Maintaining this functional joint stability is predicated on two primary components: static and dynamic restraints. The primary responsibilities of static restraints (ligaments, bones, and joint capsules) are mechanical in nature, which include the guiding of normal joint kinematics.[9] Static restraints also provide sensory information about joint position and motion, which allows for mediation of dynamic stability.[1,10] Despite their mechanical properties, static stabilizers alone cannot provide sufficient stability to adequately absorb the physiologic loads associated with functional activities.[9] In order to properly absorb the high forces associated with sport and exercise, musculotendinous structures must work in synergy with the static restraints to maximize stability.[9] This supportive role of muscles and tendons is termed dynamic restraint, which acts by absorbing loads and compressing the joint.[10] Due to their ability to manage these higher loads, muscles and the dynamic restraint mechanism are the primary stabilizers for joints, such as the knee, during functional movements.

This dynamic stabilization is influenced by multiple mechanoreceptors located within the capsuloligamentous and musculotendinous tissue. They detect stress and strain within the tissue and relay afferent, sensory signals to the central nervous system that provide information on joint position, forces, and motion.[1,74,75] There are

two primary mechanoreceptors that have been identified in the musculotendinous unit; muscle spindles and Golgi tendon organs (GTOs).[76] Muscle spindles, which are positioned in parallel with sarcomeres, act as detectors of changes and rate of change in muscle length.[22,77] They are comprised of various specialized nerve endings that are intermixed within a capsular connective tissue structure. The afferent signals to the spindles are innervated by Ia and II nerves. The spindles are comprised of intrafusal fibers, and contain efferent nerves (gamma and beta motor neurons). The extrafusal fibers, which have contractile properties, are innervated by alpha and beta motor (efferent) nerves.[1,17,78] The gamma motor neurons control activity by regulating the sensitivity to length and changes in length, thus allowing for muscle shortening while still transmitting afferent signals.[17,78] The activity of the gamma motor nerves, and thus the sensitivity of the muscle spindles, are influenced by descending signals from the cerebral cortex, and also reflexively by afferent signals from muscles, skins, and other joint mechanoreceptors.[79] When a muscle is stretched, the spindles detect the change in length, transmit an afferent signal to the spinal cord, thus triggering the monosynaptic stretch reflex, which causes an agonist muscle contraction to resist changes in length. These afferent signals from the muscle spindles are also sent along ascending tracts to supraspinal centers that help with the perception of joint motion and position, and also with the formulation and modification of motor control strategies.[17,20,21]

The second type of mechanoreceptor, Golgi Tendon Organs (GTOs), are located at the musculotendinous junction and are responsible for detection of tension or load in the muscle.[22] GTOs protect muscles from excessive loads by transmitting signals to the spinal cord and reflexively inhibiting agonist muscles and exciting

antagonist muscles.[17,22,23] Another function of GTOs is to provide feedback along with supplemental signals, such as cutaneous, articular, and other muscle receptors, to help generate a perception of load and weight. This perception, termed "force sense", is a combination of a peripherally driven sense of force and a centrally incorporated "sense of effort".[80,81,82] The perception is created when a motor command is generated, and then a copy of the command (termed corollary discharge) is sent to the somatosensory centers of the brain; it is the size of this discharge that is interpreted as effort.[83] The combination of these two components, sense of force and effort, allow for an individual to be able to determine differences between loads.[80,81,82]

The testing of force sense is a relatively new measure in sports medicine research. Recent studies have examined force sense in various pathologies, such as individuals with ankle instability, and found that individuals with perceived ankle instability along with increased reports of giving away sensations had larger errors in force sense testing.[76,84] Other articles have theorized about the effects of chronic adaptations to the GTOs, and concluded that there are increases in short and long term latency stretch reflex responses that may lead to increase force production and quicker reaction times.[23] Damage to the GTOs leads to desensitization of the organs, which may cause either an inappropriate grading of the joint load, or an over-perception of the force generated by the muscle in response to the load. In either case, the muscular response is unequal to the applied load.[23,85] The mismatch in actual versus perceived load can lead to either an inadequate force production, which cannot properly match the given load, or an overestimated force production that may cause an excessively high force response. Both of these scenarios could potentially lead to further injury of tenomuscular or capsuloligamentous structures.[76] While there is little experimental

evidence concluding any chronic, physiological changes to the GTOs over time, it is theorized that they may be providing composite information of motor unit activation levels, which would deliver feedback for control of muscle activation.[23] These adaptations may also lead to possible changes to the stretch reflex response, which could benefit rehabilitation programs tailored to improve stretch reflex in individuals with delayed responses.[23] The ability of the GTOs to relay this information and possibly adapt over time, in situations such as different conditioning histories, may affect this information. Unfortunately this has not been measured and is of particular interest to clinicians and researchers.

Force sense contributes to the regulation of muscle stiffness through the accurate prediction and detection of joint loading. This afferent information is used in the regulation of muscle stiffness. Any inappropriate estimation of this force sense can lead to improper increase in motor unit recruitment, and thus unnecessary increase in muscle stiffness.[86] Therefore, while proprioception is important for maintaining joint stability, protecting the joint is ultimately dependent on the muscle tension produced and regulation of joint stiffness.[87] Mechanical stiffness is characterized by objects that deform under influence from an external force, generate an opposing force, and can store elastic energy.[8] Joint stiffness is defined as the ratio of the change in force about a joint to the change in displacement or joint angle, and is altered primarily by the level of stiffness in muscle, which is in turn regulated by the amount of activation.[8] An increase in muscle activation can lead to 10-fold gain in knee joint stiffness.[1,88,89] These muscle forces help provide joint stability due to the interactions of the actin-myosin complex within the sarcomere and the amount of pre-contraction in a muscle.[8,87] The pre-contraction leads to an increase in crossbridge formation

between actin and myosin heads, allowing for an increase in force production and absorption, and thus an increase in active joint stiffness.[35,90] The amount of force generated by muscle fibers is also affected by length-tension and force-velocity relationships, as well as the viscoelastic properties of the tissue. Muscles are able to generate the most force, and be most effective in joint stabilization, when they are at an optimal sarcomere length due to the maximal number of cross-bridge formations at this position.[87] The force-velocity relationship affects stabilization due to the velocity of a muscle contraction; it is directly proportional to the load imposed on it. At higher velocities the force produced is smaller, whereas at lower velocities the muscles forces are increased.[87] All of these components demonstrate that an increase in stiffness in muscles can lead to an increased resistance to perturbations and provide greater dynamic restraint to joint displacement via the origin and insertion locations of musculotendinous structures.[10] While an increase in stiffness provides additional dynamic restraint, this is not always ideal for coordinated movement. Conditions such as multiple sclerosis, in which patients display an increase in spasticity in muscles around a joint, create abnormalities in muscle stiffness levels that can disrupt normal movement; in these cases excessive stiffness does not allow for proper movement and disrupts coordination.[91]

In dynamic movements, the ability of the muscles, tendons, and ligaments to properly transfer energy and dissipate loads is essential for movement efficiency, and is influenced by muscle stiffness in order to increase performance or prevent injury. Sinkjaer et al[36] examined the mechanical response of a stretch to the ankle dorsiflexors at different levels of voluntary contraction. In the study, Sinkjaer described the actin-myosin interactions as the intrinsic component of active stiffness, while also exploring

the reflexive component that occurs during a rapid stretch of the muscle. Sinkjaer's total mechanical response of the active muscle was defined as the sum of the intrinsic responses (contractile apparatus plus mechanical behavior of passive tissues) plus the reflex mediated response.[36] While examining the total stiffness, the intrinsic component linearly increased with an increase in muscle pre-activation, while the contribution of the reflexive portion peaked between 30-80% MVIC and increased the overall stiffness 40-100% beyond just the intrinsic stiffness, with a 75% increase at 30% MVIC.[36] This study demonstrates that the more motor units that are recruited in the muscle, the overall stiffness increases and the muscle becomes less responsive to brief fluctuations.[36] Sinkjaer demonstrated that the reflexive contribution is essential to movement, as that contributes a significant amount of stiffness to dynamic stability.

While increases in muscle stiffness have been shown to contribute to joint stability, task-dependent situations may exist that require an optimal level, and that level may not necessarily coincide with an absolute increase in stiffness. Examples arise where excessive muscle stiffness might be detrimental to performance or injurious to muscle, tendon and joint structures. Other task-dependent situations may arise where an insufficient level of muscle stiffness might not be able to provide the appropriate deceleration of joint rotations.[3] Gender disparities may also exist, with conflicting data suggesting females have greater stiffness[38], while others suggest females have lower levels[55,56,57,58], or that there is no difference in stiffness between genders.[92,93] This disparity in the literature has been argued to help explain the increased lower extremity injury rates in females compared to males[59]; however, there remains a large number of males who also sustain ACL injuries via non-contact

mechanisms, which also occur through a failure in appropriate stiffness regulation and stability.

In order to optimize performance, an optimal stiffness level must be achieved for functional movement. Current literature has studied stiffness of the series elastic component, specifically during a stretch-shorten cycle, and have been conflicting in their results. Some researchers advocate a very compliant series elastic component (SEC) as optimal for performance and power[94,95], while other advocate that greater overall stiffness is better.[11,12] Still other data suggests a balance between stiffness and compliance is optimal.[14,15] Wilson et al[5] used a rebound bench press lift to determine the optimal stiffness for maximal power output with this maneuver, and the results suggest that the optimal level was one of a more compliant nature. Conversely, Wilson et al[13] found in another study examining musculotendinous stiffness in concentric, eccentric, and isometric performance, that the optimal musculotendinous stiffness for maximum concentric and isometric power output was toward a stiffer level. Generally the literature does support an individualized, task-dependent level of optimal stiffness during physical activity.

Anecdotally, situations occur in sport and activity where an individual may allow their body, particularly the lower extremity, to create a more compliant system for a controlled or partial "collapse" after a functional maneuver in order to avoid possible excess strain on passive and active structures that could otherwise not dissipate higher forces. This theory of compliance or joint unloading, would outwardly appear as a well-controlled buckling within the joints normal physiological range of motion and arthrokinematics, rather than the limited movement that would result from high stiffness levels. Is possible that this may be more beneficial to an

individual for dissipation of energy and preservation of static structure integrity during certain task-dependent situations. Contrary to a stiffening strategy, this compliance may be investigated through examination of an individual's ability to quickly deactivate their muscles, thus "unloading" a joint, during a sudden perturbation.

Neuromuscular Control

Neuromuscular control is an important determinant in the ability of an individual to perform dynamic and functional movement without injury. It has been defined as the ability of the nervous and musculoskeletal systems to coordinate motion and respond to sudden perturbations[96], while also controlling the transformation of neural information or motor commands into physical energy via muscle activation.[1] Neuromuscular control helps to achieve optimal joint stiffness through the regulation of muscle tension, allowing for control of functional movements while also contributing to dynamic restraint. This occurs through dissipation of loads in musculotendinous units, allowing maintenance of integrity of static stabilizers and allowing for stability through a full range of motion. This restraint is controlled through the regulation of muscle stiffness; the level of stiffness is influenced by characteristics such as preparatory and reactive muscle activation.[2] Feed-forward control uses pre-planned movements and anticipatory muscle activation to prepare for a joint load or perturbation based on past experiences, and thus decreases reaction times.[3,17,18] This allows for pre-activation of muscles before a load is applied to a joint and aides in proper absorption of large forces, decreasing the stress on static structures. Feedback control relies on reflex arcs from muscle and joint mechanoreceptors to constantly provide afferent information to maintain stability and maximize performance.[3] During situations of unanticipated stimuli, feedback control

may alter the pre-programmed muscle activation strategies of the feed-forward loop.[19] A disruption or delay in neuromuscular firing of these loops can lead to improper limb positioning, which may increase strains on joints.[19]

Conditioning History

The ability of a person to provide an optimal level of stiffness in order to complete functional movements can be influenced by various factors, with one of them being a person's conditioning history. This history, which typically can be linked to the type of sport that they participate in, may be influenced by muscle physiology and composition. There are two primary types of muscle fibers: type I, or slow twitch, and type II, or fast twitch. Distinct differences exist between type I and type II fibers, with type II fibers showing a higher intrinsic speed of contraction, larger peak power, reaching of peak power at high velocities of contraction, but with a quicker fatigue point than type I fibers.[25,26] While muscle fiber distribution is primarily a genetic component, certain types of activity and conditioning are associated with and can enhance those fiber types. Typically type I fiber growth and motor unit recruitment is facilitated by a training focus of high repetitions and low loads and are primarily found in endurance athletes.[24] Type II fibers are generally facilitated by a training regime of low repetitions and high loads and are commonly seen in power athletes.[24] Studies have found that certain training regimens and conditioning histories can lend themselves to a certain type of fiber, including sprint runner's having a larger percentage of type II fibers[43,97,98] and also sprint training producing a shift toward type II fibers and increase in type II fiber area.[99] Other studies have shown a direct relationship between athlete type and fiber type composition, with endurance athletes having more type I fibers and power athletes

having more type II fibers.[43,100,101,102] One study involving biopsies of various track athletes found substantially more slow-twitch (type I) fibers in endurance runners than in sprint oriented athletes.[43] Costil found that for male sprinters, their biopsy contained 24.0% slow twitch fibers, while middle distance had 51.9%, distance had 69.4%, high jump had 46.7%, javelin throwers had 50.4%, and shot-put/discus throwers had 37.7%.[43] A person's conditioning history, along with other factors, may contribute to the regulation of stiffness and choice of dynamic restraint strategy that is employed in response to a sudden perturbation. Previous studies have defined stratification profiles that distinguished different types of athletes, like power and endurance, based on variables such as leg stiffness.[27,29] Hobara et al[27] examined the difference in leg stiffness between power and endurance athletes. In the study, subjects performed a repeated two-legged hop at 1.5 and 3.0 Hz and data was collected on lower leg stiffness (comprised of knee and ankle stiffness), touchdown angle, and various mean EMG values at various points within the hopping task. The study found that power athletes had increased leg stiffness compared to endurance athletes, which was in agreement with previous studies.[28,29] However, the increased stiffness was not due to any increase in preparatory muscle activation or reflex response, which was the expected result. Instead, the endurance athletes, not power athletes, had greater EMG activation, and thus increased muscle pre-activation compared to power athletes. The variation in stiffness was attributed to intrinsic stiffness differences in the triceps surae and associated tendon, which had been seen previously but were not specifically defined.[30] While there are conflicting theories and results about the source of muscle and joint stiffness between groups, power and

endurance athletes provide a unique opportunity to examine these groups more thoroughly under highly controlled conditions.

TABLES

Table 1. Stiffness Testing Conditions

TITLE	ABBREVIATION	SUBJECT'S INSTRUCTION
Passive Non-Reactive	PS	"Stay completely relaxed during the entire perturbation"
Passive Reactive	PRS	"Stay relaxed, and when the perturbation occurs, resist it as hard and as quickly as you can"
85% Contraction Non-Reactive	AS	"Push out to [85% MVIC] prior to the move. When you feel the perturbation, maintain the same level of contraction; no more or no less"
85% Contraction Reactive	ARS	"Push out to [85% MVIC] prior to the move. When you feel the perturbation, resist it as hard and as quickly as you can."
85% Contraction Deactive	DS	"Push out to [85% MVIC] prior to the move. When you feel the perturbation, relax your muscles as quickly as you can and let the move happen."

Table 2. Subject demographics for endurance athletes (END), power athletes (PWR), and controls. [a] Body mass between END and CON significantly different ($p < 0.050$).

	END	PWR	CON
N	15	12	15
Age (yrs)	19.8 ± 1.1	20.3 ± 1.3	20.1 ± 1.6
Height (cm)	176.6 ± 8.6	175.5 ± 4.9	177.0 ± 4.3
Mass (kg)[a]	65.9 ± 8.8	74.5 ± 10.3	79.0 ± 12.8
QMVIC (Nm)	98.4 ± 33.9	113.9 ± 42.0	110.0 ± 28.4
HMVIC (Nm)	108.5 ± 33.1	118.4 ± 47.0	122.9 ± 29.0

Table 3. Short-range (0-4°) and Long-Range (0-40°) stiffness values for endurance athletes (END), power athletes (PWR), and controls (CON) across all conditions. [a]END significantly different than PWR & CON. [b] PWR significantly different than END.

	Group	Short-Range (0-4°) [Nm/°/kg]	Long-Range (0-40°) [Nm/°/kg]
PS	END	0.057±0.012[a]	0.0016±0.001
	PWR	0.052±0.013	0.0020±0.001[b]
	CON	0.047±0.008	0.0015±0.001
AS	END	0.085±0.011	0.025±0.022
	PWR	0.087±0.016	0.025±0.021
	CON	0.081±0.013	0.022±0.013
PRS	END	0.055±0.010	0.052±0.013
	PWR	0.055±0.010	0.061±0.022
	CON	0.046±0.011	0.054±0.014
ARS	END	0.084±0.014	0.051±0.017[a]
	PWR	0.088±0.015	0.037±0.015
	CON	0.080±0.020	0.033±0.011
DS	END	0.081±0.015	-0.020±0.009
	PWR	0.085±0.011	-0.020±0.012
	CON	0.079±0.019	-0.022±0.012

Table 4. Peak EMG for Endurance (END), Power (PWR), and Control (CON) groups across conditions for quadriceps and hamstrings. [a] END significantly different than PWR. [b] END significantly different than CON. [c] PWR significantly different than CON.

	Group	PS	AS	PRS	ARS	DS
VM	END	13.2±11.9	82.1±32.1	79.8±43.2	121.3±55.2[b]	63.5±14.3
	PWR	8.51±7.29	82.9±27.2	59.7±25.1	111.1±36.8	68.9±23.3
	CON	8.31±6.22	77.4±16.7	57.6±17.3	82.1±15.5	60.0±11.8
VL	END	19.0±15.5[a,b]	81.5±27.6	64.6±23.8	102.6±44.8	68.1±19.4
	PWR	8.25±7.87	96.4±82.9	71.8±48.3	118.0±65.6	55.3±22.1
	CON	9.56±10.2	75.8±16.7	56.3±16.5	87.2±21.8	65.6±19.3
MH	END	11.9±10.3	13.3±8.35	15.7±10.9	14.2±7.43	18.0±14.7
	PWR	11.6±8.79	14.4±7.28	14.6±8.45	19.0±7.80	22.9±13.7
	CON	12.8±15.3	17.3±14.0	17.5±14.4	19.1±14.3	20.9±14.2
LH	END	15.8±12.4	24.3±13.1	21.3±10.2	29.8±15.7	26.0±13.0
	PWR	10.0±10.9	23.4±16.0	18.2±10.7	26.2±13.9	25.4±14.3
	CON	17.1±17.9	48.0±45.1[a,c]	33.6±29.6[c]	48.3±41.5[c]	45.1±41.6

Table 5. Time to Peak (TTP) total values for each muscle and condition.

	VM (s)	VL (s)	MH (s)	LH (s)
PS	0.270 ± 0.305	0.610 ± 1.36	1.28 ± 2.10	0.644 ± 1.15
AS	0.215 ± 0.126	0.206 ± 0.115	0.404 ± 0.970	0.161± 0.123
PRS	0.474 ± 0.264	0.443 ± 0.248	0.378 ± 0.530	0.343 ± 0.246
ARS	0.382 ± 0.244	0.297 ± 0.184	0.262 ± 0.173	0.262 ± 0.164
DS	0.049 ± 0.035	0.053 ± 0.033	0.238 ± 0.613	0.894 ± 0.052

Table 6. AUC values for all conditions, muscles, and groups. [a] END significantly different than PWR. [b] END significantly different than CON [c] PWR significantly different than CON

		VM			VL			MH			LH		
		PRE	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*
PS	*END*	1.22±1.19	2.90±2.74	2.57±2.33	1.68±1.35	4.15±3.39	3.69±3.01	1.04±0.884	2.56±2.32	2.33±1.99	1.36±0.99	3.42±2.63	3.04±2.28
	PWR	0.750±0.614	1.87±1.60	1.70±1.49	0.749±0.679	1.81±1.73	1.59±1.57	0.996±0.728	2.56±1.95	2.30±1.77	0.877±0.91	2.19±2.43	1.95±2.17
	CON	0.719±0.531	1.85±1.39	1.62±1.23	0.806±0.837	2.11±2.26	1.86±2.02	1.07±1.19	2.81±3.41	2.50±3.03	1.48±1.45	3.84±3.95	3.40±3.55
AS	*END*	8.21±1.62	15.4±4.32	14.7±6.81	8.21±2.11	15.8±4.61	15.6±6.79	1.12±0.808	2.91±1.70	2.41±1.66	2.41±1.55	5.06±2.85	4.43±2.70
	PWR	8.34±2.66	15.7±4.20	12.5±8.33	8.60±3.89	18.5±14.9	15.8±19.9	1.39±0.881	3.20±1.56	2.68±1.44	2.35±1.92	4.94±3.54	3.84±2.37
	CON	7.84±1.62	13.9±2.72	12.9±4.14	8.45±2.47	14.9±3.98	12.4±3.36	1.50±1.28	3.49±2.97	3.34±3.03	5.70±6.29	9.78±9.23	7.60±6.15
PRS	*END*	1.25±1.16	7.49±4.91[b]	13.5±7.05	1.73±1.17[a,b]	7.02±2.84	12.4±5.74	1.04±0.896	2.98±2.40	2.55±1.78	1.28±0.94	3.87±2.23	4.10±2.29
	PWR	0.767±0.54	5.37±2.36	11.7±5.40	0.804±0.629	5.51±3.22	14.5±10.6	1.05±0.733	2.77±1.93	2.73±1.63	0.89±0.91	2.66±1.90	3.51±2.10[c]
	CON	0.719±0.49	4.77±2.18	10.5±3.54	0.943±0.784	5.27±2.78	10.2±3.47	1.08±1.25	3.35±3.38	3.37±2.66	1.45±1.44	4.82±4.05	6.43±5.47
ARS	*END*	9.36±3.16	22.0±9.37[b]	23.9±11.4[b]	8.38±3.13	18.8±7.35	21.7±10.5	1.14±0.71	2.83±1.63	2.70±1.66	2.57±1.69[b]	5.71±3.05	5.91±3.16
	PWR	9.26±3.78	20.2±6.19[c]	19.2±8.70	9.07±3.61	20.4±9.62	22.8±14.1	1.55±1.18	3.62±1.65	3.37±1.75	2.71±1.94	5.41±3.04	4.64±2.18[c]
	CON	8.40±1.63	14.5±3.32	16.1±4.34	8.97±2.34	15.8±5.54	16.3±5.30	1.50±1.25	3.80±3.16	3.85±3.22	5.12±5.03	10.1±8.94[b,c]	9.07±8.20
DS	*END*	8.62±2.29	10.8±2.88	3.95±3.26	8.49±2.25[a]	12.2±4.82[a]	5.00±3.39[a]	1.12±0.73	3.25±2.34	2.14±1.59	2.51±1.66	5.02±2.47	2.96±2.13
	PWR	8.52±2.52	11.7±5.03	3.44±2.74	7.64±3.14	9.26±3.29	2.24±1.25	1.24±0.73	4.32±2.10	2.38±1.53	2.70±2.08	5.08±2.89	2.31±1.91
	CON	8.11±2.03	9.72±2.38	2.92±1.73	8.53±2.53	11.0±4.24	3.24±2.20	1.45±1.34	4.09±3.12	2.78±2.94	5.28±5.66	8.36±6.88	4.01±3.51

7. EMD values for the muscles of the END, PWR, and CON groups.

	Group	VM	VL	MH	LH
EMD	END	74.7±23.0	81.5±21.3	103.1±23.3	109.6±21.9
	PWR	68.7±25.0	64.7±20.1	91.6±20.4	85.4±24.2
	CON	77.5±21.4	74.0±21.4	90.0±23.4	84.0±24.5

FIGURES

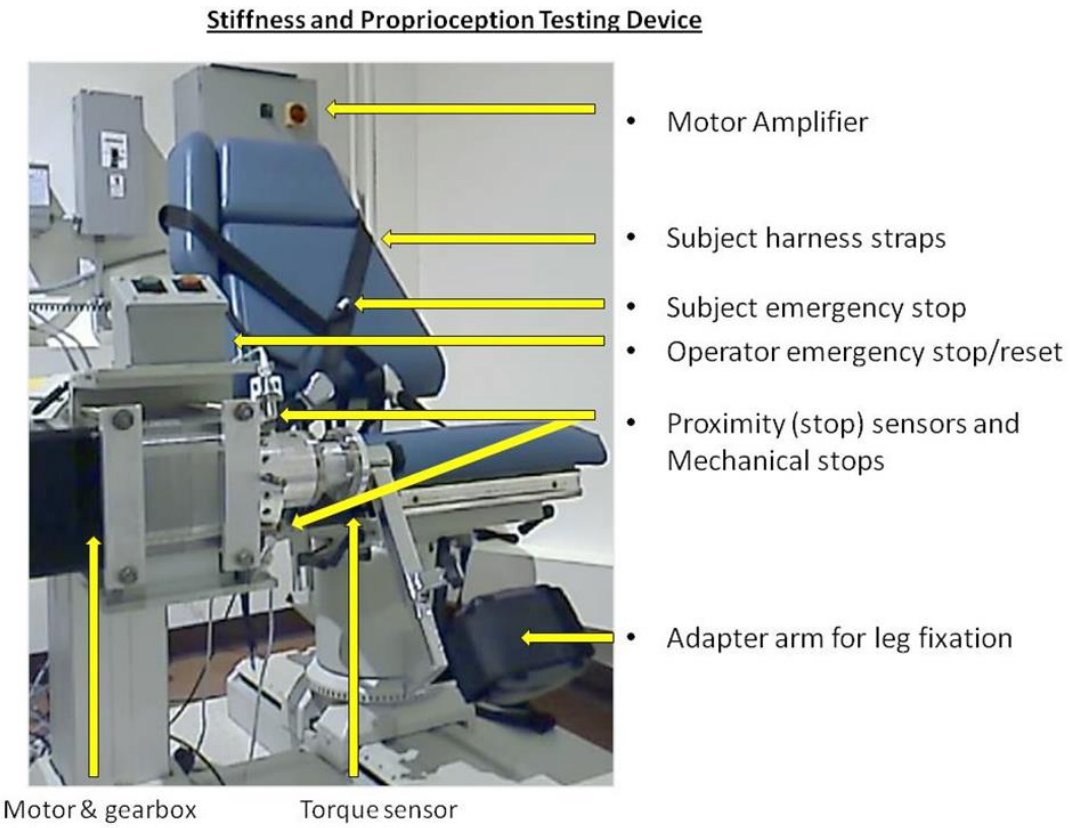


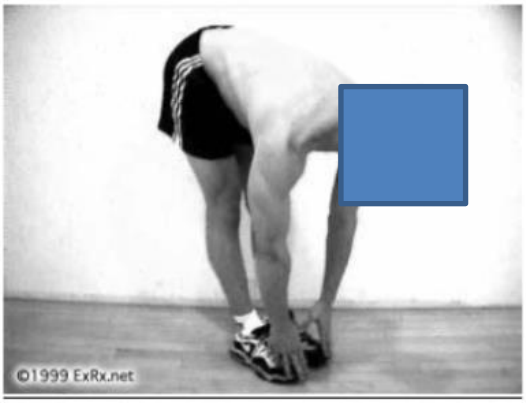
Figure 1. The Stiffness and Proprioception Testing Device (SPAD)

Quadriceps & Hamstring Stretching Techniques

QUADRICEPS:

Stand and touch the wall for support. Bend your heel towards your back and pull your ankle and forefoot to your rear end. Hold for 30 seconds and repeat 3 times.

HAMSTRINGS:



Stand and slightly bend your knees. Bend at the waist and reach towards your toes or floor and hold for 30 seconds. Repeat 3 times.

Figure 2. Quadriceps and Hamstrings Stretching Technique Handout

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Appendix A

IRB PROJECT APPROVAL

RESEARCH OFFICE

210 Hullihen Hall
University of Delaware
Newark, Delaware 19716-1551
Ph: 302/831-2136
Fax: 302/831-2828

DATE: March 5, 2010

TO: David C Oates II, BA
FROM: University of Delaware IRB

STUDY TITLE: [156420-1] Reactive Knee Stiffness Regulation Strategies
IRB REFERENCE #:
SUBMISSION TYPE: New Project

ACTION: APPROVED
APPROVAL DATE: March 5, 2010
EXPIRATION DATE: February 16, 2011
REVIEW TYPE: Full Committee Review

Thank you for your submission of New Project materials for this research study. The University of Delaware IRB has APPROVED your submission. This approval is based on an appropriate risk/benefit ratio and a study design wherein the risks have been minimized. All research must be conducted in accordance with this approved submission.

This submission has received Full Committee Review based on the applicable federal regulation.

Please remember that informed consent is a process beginning with a description of the study and insurance of participant understanding followed by a signed consent form. Informed consent must continue throughout the study via a dialogue between the researcher and research participant. Federal regulations require each participant receive a copy of the signed consent document.

Please note that any revision to previously approved materials must be approved by this office prior to initiation. Please use the appropriate revision forms for this procedure.

All SERIOUS and UNEXPECTED adverse events must be reported to this office. Please use the appropriate adverse event forms for this procedure. All sponsor reporting requirements should also be followed.

Please report all NON-COMPLIANCE issues or COMPLAINTS regarding this study to this office.

Please note that all research records must be retained for a minimum of three years.

Based on the risks, this project requires Continuing Review by this office on an annual basis. Please use the appropriate renewal forms for this procedure.

If you have any questions, please contact Elizabeth Peloso at 302-831-8619 or epeloso@udel.edu.
Please include your study title and reference number in all correspondence with this office.

Appendix B

PHYSICAL ACTIVITY AND READINESS QUESTIONNAIRE (PAR-Q)

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)

Common sense is your best guide in answering these few questions. Please read them carefully and check YES or NO opposite the question if it applies to you. If yes, please explain.

YES	NO	
☐	☐	1. Has your doctor ever said you have heart trouble? Yes, _____
☐	☐	2. Do you frequently have pains in your heart and chest? Yes, _____
☐	☐	3. Do you often feel faint of have spells of severe drowsiness? Yes, _____
☐	☐	4. Has a doctor ever said your blood pressure was too high? Yes, _____
☐	☐	5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise? Yes, _____
☐	☐	6. Is there a good physical reason, not mentioned here, why you should not follow an activity program even if you wanted to? Yes, _____
☐	☐	7. Do you suffer from any problems of the lower back (i.e. chronic pain or numbness)? Yes, _____
☐	☐	8. Are you currently taking any medications that may affect joint and muscle stiffness? Please specify. Yes, _____
☐	☐	9. Do you currently have a disability or a communicable disease? Please specify. Yes, _____

Appendix C

INFORMED CONSENT FORM

Research Study: Reactive Knee Stiffness Regulation Strategies

Investigators: David Craig Oates II, BA (Graduate Assistant) and
Charles Buz Swanik, PhD, Associate Professor
(Department of Health, Nutrition and Exercise Sciences)

1. PURPOSE/DESCRIPTION OF THE RESEARCH

Introduction

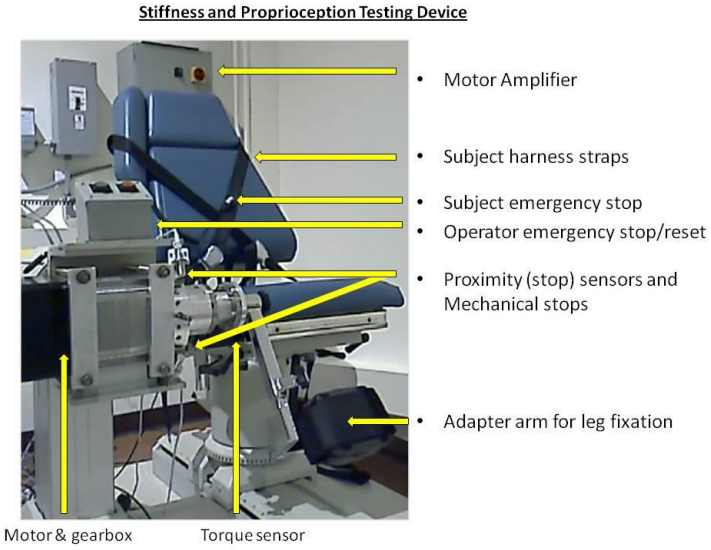
You are invited to take part in a research study to compare knee stiffness differences between people with different histories or physical activity (power versus endurance athletes) and controls. Your participation is voluntary and you are in no way obligated to take part in this testing. You may withdraw your participation in this study at anytime without penalty.

Purpose

The purpose of this research is to determine if different types of training backgrounds (power versus endurance versus control) affect how a person is able to stiffen their knee and how their muscle contractions affect stiffness of the knee. If differences exist, it may help find training factors that prevent knee or muscle injuries and improve rehabilitation.

Eligibility

Approximately 60 males will participate in this study (20 power athletes, 20 endurance athletes, and 20 controls). If you agree to participate, you will be scheduled for one testing session that will last approximately 1 1/2 hours. This testing will occur in the Human Performance Laboratory in the Fred Rust Arena at the University of Delaware, Newark DE 19716. You will be asked to wear active shorts during testing.



Procedures

We will test the stiffness of your knee joint when your thigh muscles contract and relax. Testing will be performed using the custom-built stiffness device (SPAD—shown above) that will measure your muscle contractions and how your knee resists bending. This is a seated device that will measure the strength (force) of your thigh muscles during the test.

Before testing, you will fill out a Physical Activity Readiness Questionnaire (PAR-Q) to look at your activity level. You will then warm-up on a bike for 5 minutes and stretch your muscles. In order to determine when your muscles turn on and off, we will tape small sensors over the muscles in the front and back of your thigh with adhesive skin tape after the skin over the muscles has been shaven, abraded, and cleansed with an alcohol swab. Hypoallergenic tape or non-tape methods of attaching the equipment will be available if you are allergic to adhesive skin tape. Cables from the sensors will be attached to a small box near you during the tests. The box sends information about your muscles to a computer.

To test knee stiffness, you will be seated securely in a chair with the knee (of the leg to be tested) placed in a splint, so extra movements will not occur during testing. Once your body and leg are stabilized, your leg will be in a slightly bent position. From this position you will be asked to push or pull against the device as hard as you can for a few seconds; this will be performed three times. You will then be asked to relax your muscles before the test begins. You will then perform a test in which you try to match a certain level of muscle contraction strength told to you by the tester. You will also be asked to complete 5 different stiffness testing conditions. Each condition will be tested 5 times. 2 conditions will require you to start the test with your leg muscles completely relaxed, while the other 3 will require that you start the testing at a certain level of muscle contraction. Sometime within 10

seconds of starting each condition, your leg will be moved a short degree in backward rotation (knee flexion) by the device. When you feel the motion you will either let the motion happen, react to the motion by pushing back against it, or relax your muscles immediately and let the motion occur; which response you do will be told to you in advance by the tester.

You will hold an emergency stop switch in your hand al all time so that you can turn the device off at any point during the testing procedure. The tester will also hold an emergency stop switch. Emergency stop sensors are also located in the device motor and on the attachment arm, so that your leg will not be moved beyond your normal limits.

2. CONDITIONS OF SUBJECT PARTICIPATION

You should not participate in this research study if you have a history of:

1. Previous history of fracture or surgery to the lower back, hips, knees, or ankles within the past year;
2. Any current injuries to the test leg;
3. Any hearing impairments or complications that are uncorrected; or
4. Neurological or cardiovascular problems that limit moderate physical activity

Your participation in this research is completely voluntary and you may withdraw from this study at any time without penalty.

If you are physically injured during laboratory testing procedures, you will receive immediate first aid care. If you require additional medical treatment, you will be responsible for the cost.

3. RISKS AND BENEFITS

You may experience some muscle or joint soreness within the next few days following the testing session. The soreness is similar to what you may feel following a vigorous weight lifting routine. There is minimal risk of muscle and/or joint injury (i.e. pulled muscle, joint sprain) as a result of testing. Close supervision, use of rest periods, and use of emergency stop switches during testing will minimize risks involved with this protocol.

4. CONFIDENTIALITY

Each subject will be identified by a case number and the investigators will have access to the data. Information and data will be stored on a computer until the study is completed and published. Neither your name nor any identifying information will be used in any publication or presentation resulting from this study. Following completion of the study, the data will be copied, removed from the computer, and stored in a locked cabinet indefinitely. The data may be used in the future for comparisons with data from other research studies.

4. FINCANCIAL CONSIDERATIONS

There will be no compensation for your participation.

5. CONTACTS

Any questions or concerns regarding this research study should be directed to:

Charles "Buz" Swanik, PhD, ATC
151 Human Performance Laboratory
c/o Fred Rust Ice Arena
541 South College Avenue
University of Delaware
Newark, Delaware 19716
Phone: (302) 831-2306

Any further questions regarding your rights as a participant in a research study should be directed to

Chair of the Human Subjects Review Board
Office of the Vice Provost for Research
210 Hullihen Hall
University of Delaware
Newark, Delaware 19716
Phone: (302) 831-2137

6. SUBJECT'S ASSURANCE

I have read the above informed consent document. The nature, demands, and risks and benefits of the study described above have been discussed with the investigators. I have been given the opportunity to ask questions regarding this study and they have been answered to my satisfaction. I understand that participation in this study is voluntary and that I may withdraw at any time without consequence. A copy of this consent form has been given to me.

7. CONSENT SIGNATURES

Participant's Signature:_____ Date:_____
Participant's Name (please print):_____
Investigator Signature:_____ Date:_____

Appendix D

FORCE SENSE RESULTS

Results for force sense testing are shown in the table below. The main effect of the muscle group, for time-to-matched torque was significant ($F_{1,39}$=11.12, p=0.002). Time-to-matched torque for the hamstrings was faster than quadriceps ($p < 0.050$).

Table of Force Sense values for END, PWR, and CON groups with both quadriceps and hamstring groups. [a] Time-to-Matched Torque significantly different between hamstrings and quadriceps ($p < 0.05$).

Target	Variable	END	PWR	CON
Hamstrings[a]	Relative Error (%)	26.4±18.2	36.0±16.8	33.7±20.5
	Time-to-Match (s)	2.49±0.84	2.45±0.88	2.4±1.02
	Coefficient of Variation (%)	0.02±0.01	0.02±0.01	0.03±0.01
Quadriceps	Relative Error (%)	37.6±30.3	28.8±8.4	25.2±18.3
	Time-to-Match (s)	3.0±1.02	3.0±1.22	2.54±1.30
	Coefficient of Variation (%)	0.02±0.01	0.02±0.01	0.03±0.02