AN ABSTRACT OF THE THESIS OF

Kimberly Ann Frostad for the degree of Master of Science in Human Performance presented on June 5, 2000. Title: The Efficacy Of Warm-Up And Stretching On Lower Extremity Muscular Force Production And Hamstring Extensibility In Non-Power-Trained Individuals.

Abstract approved: _____________________________

Paul Borsa

Objective: To determine the effectiveness of moderate pre-exercise warm-up, static stretching, or a combination of both on muscular force production and hamstring extensibility of the lower extremity.

Design and Setting: This experiment was a counterbalanced repeated measures design of four treatment conditions. Subjects participated in warm-up, static stretching, combined warm-up and stretching, and no treatment (control) on four separate testing days. After each treatment, hamstring extensibility was measured using the Active Knee Extension Test. Muscle force production was measured using a force dynamometer and associated software. Maximal voluntary isometric contractions of the quadriceps and hamstrings were assessed for the dominant limb.

Subjects: Seventeen moderately physically active males and females with no history of lower extremity injury within the past six months were used in this study.

Measurements: Angular displacement in degrees was assessed to determine hamstrings extensibility. Peak muscle force production (PFP) and peak rate of force production (PRFP) was assessed for quadriceps and hamstring isometric contractions. The differences between treatments were analyzed using a repeated measure analysis of variance.

Outcomes: ANOVA revealed no statistically significant difference between treatments for the variables of hip flexion range of motion and peak force.
production. Post hoc comparisons revealed that peak rate of force production was significantly less for the stretching treatment compared to the other three conditions. The outcomes showed no difference between the active treatments and the control condition in muscle force production and range of motion.
The Efficacy Of Warm-Up And Stretching On Lower Extremity Muscular Force Production And Hamstring Extensibility In Non-Power-Trained Individuals

by

Kimberly Ann Frostad

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Chair of Department of Exercise and Sport Science

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Dean of Graduate School

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Kimberly Ann Frostad, Author
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Introduction

Anecdotal evidence suggests that warm-up and stretching prior to athletic activity are effective in preventing injury and enhancing athletic performance. However, there is limited scientific proof substantiating these claims (1-5). Recent research has studied the effects of active muscle contraction, increased tissue temperature, and type of stretch on increased range of motion (6-15). Other studies have measured the effects of varying intensities of warm-up on muscle power (6,16-20). These studies give indirect support of active warm-up and stretching, yet few studies have examined the direct effects of stretching and warm-up on muscle performance (22,23).

The few studies published recommend varied intensities of warm-up for anaerobic activity and suggest little concerning increases in range of motion due to warm-up and pre-exercise stretching (6,16,17). DeBruyn-Prevost and Lefebvre (16) conclude that the intensity of the active warm-up must be light to improve performance, while Stewart and Sleivert (6) state that a warm-up must be of an intensity of 60-70% VO₂ max to be effective. There are more general studies such as Houmard et al. (17) whose only suggestion is that any warm-up is beneficial. The only consistency between studies is the generalization that some is better than none.

Warm-up can be defined as either passive or active. Warm-up can be local, from the application of heat packs to a specific area, or systemic, from an increase in core body temperature, which affects all bodily tissues. This rise in temperature causes two characteristic changes. The metabolic rate, defined as the Q₁₀ effect, increases logarithmically with an increase in body temperature. This causes an increase in the utilization of energy within each human cell. In addition,
viscoelasticity is the strongest known factor in support of the theory of a pre-exercise stretching and warm-up routine. As connective tissue, specifically collagen, increases in temperature its fluid (viscous) characteristics dominate. The result is more permanent changes in the tissue characteristics. (3,23,24).

There are two main characteristics that physiologists believe describe this deformation in tissue. Stress relaxation refers to the decrease in intramuscular force at a constant length or angular displacement. Creep refers to the changes seen when a constant load, or stress, is applied. (25)

We believe that stress relaxation and creep deformation describes the changes seen with the common forms of muscle and joint stretching. The connective tissue, after initial stretch of the tissue from applying a load, theoretically “creeps” or continues to deform and/or relax until the load is released. Permanent gains in range of motion have been theorized to be at least partly due to viscoelasticity (25).

Another factor in length changes is due to the ultimate failure of biological tissue. With any significant force applied over time on living tissue, there is always some amount of fatigue or failure. This is termed the first-cycle effect (25). Ultimately, the tissue under stress will behave differently the first time a force is applied than if the force is repeated. Theoretically more cycles of stress cause the tissue to change semi-permanently from the introduced force. (24-26).

The objectives of this study were to determine the efficacy of warm-up and stretching on lower extremity muscular force production and hamstring extensibility in non-power-trained males and females. We hypothesize that active warm-up, passive static stretching, and/or a combination of both treatments will significantly enhance lower extremity muscular force production and active hamstring extensibility compared to no treatment.
METHODS

SUBJECTS AND DESIGN

Seventeen physically active subjects, 8 males and 9 females, volunteered to participate in the study. The mean age of the subjects was 22.7 years, and the mean weight was 74.4 kg. Subjects had not engaged in any power training regimens in the past six months and were free of any musculoskeletal-related injury to the lower extremity.

A repeated measures design was used to analyze selected muscular force production characteristics and hamstring extensibility of the dominant limb under four experimental conditions: warm-up, stretching, warm-up and stretching combined, and control. The conditions were counterbalanced within subjects and between sessions. Appointment times were scheduled with at least 24 hours of rest between testing sessions. Each subject completed testing within a period of three weeks.

Procedures for testing were approved by the Oregon State University Institutional Review Board (IRB) for protection of human subjects. Subjects were provided a verbal and written explanation of the testing protocols followed by completion of the informed consent and pre-test questionnaire (Appendix B).

INDEPENDENT VARIABLES

Warm-up (WU)

The pre-exercise warm-up routine was performed on a stationary bicycle ergometer at an intensity within the subject’s heart rate range, and 60-70 rpm. The
target heart rate range was defined using the Karvonen formula for between 60-75% of their age-predicted heart rate maximum (27, 28). Each subject pedaled at this intensity for ten minutes. Heart rate was assessed using a Uniq ProTrainer heart rate monitor (Computer Instruments Corporation, Hempstead, NY). Body temperature was taken initially, at five minutes, and at the completion of testing using a tympanic thermometer.

**Stretching (S)**

The pre-exercise muscular stretching routine was performed using a partner-assisted, static stretching technique for the major biarticular muscle groups of the lower extremity (hip extensors/knee flexors and hip flexors/knee extensors). The co-principal investigator (KAF), who is a certified athletic trainer (ATC) with extensive experience in the art and science of lower extremity stretching, applied the stretching protocol. Each muscle group was consistently stretched using a preset frequency (4 repetitions), duration (15 seconds), and tensile force (10% body weight) (see Appendix C for specific stretching exercises) (5,12,13,29,30). For each repetition the investigator slowly and passively moved the limb at a tensile force of 10% body weight (BW) using a commercially available force applicator scale (Chatillon, NY), and held there for the remainder of the stretch.

**Combined (WU/S)**

The combined routine consisted of the warm-up immediately followed by the stretching routine.

**Control (C)**

The subject did not perform any of the pre-exercise routines, except for the mandatory pre-exercise warm-up.
INSTRUMENTATION

**Kin-Com Isokinetic Dynamometer (Chattecx Corporation, Chattanooga, TN)**

This dynamometer is a testing device for muscle force output. It is a multi-joint machine with a computerized database for specific isometric and isokinetic testing protocols of numerous joints in the human body. All protocols for using this machinery are in accordance with valid and reliable methods of use for simple voluntary contraction testing of the lower extremity (33-36).

**Leighton Flexometer (Leighton, Spokane, WA)**

This device was used for the measurement of hamstring extensibility. The reliability of the flexometer has been reported to be at least 0.90 (8,37,38). The flexometer is a gravity-based measuring device that uses a free moving, weighted needle and dial to measure angular displacement. Both dial and needle can be locked in any position to set the flexometer at certain degrees of movement. The flexometer is used to measure extremities’ positions in relation to a horizontal zero baseline. All measurements are accomplished with the subject lying prone on a standard treatment table (7).

DEPENDENT VARIABLES

**Angular Displacement (AD)**

The Active Knee Extension Test (AKET) protocol was used in accordance with Sullivan et al. (7). The subject was supine on a padded table with the dominant leg extended and stabilized. Subject’s dominant leg was stabilized at 90° of hip flexion. The subject’s pelvis and other leg were stabilized to the table to
isolate only the hamstrings of the dominant leg for testing. A Leighton flexometer was locked with a baseline zero set exactly in alignment with the horizontal plane, which corresponds directly with 90° of knee flexion. Dials for the flexometer were then fixed with strapping tape to avoid additional errors due to movement of the flexometer needle. The flexometer was then strapped onto the tested leg directly over the fibular head, and the foot was allowed to remain in relaxed plantar flexion. The subject was instructed to slowly extend the knee (approximately 15°/sec) maximally while retaining the right angle of the hip. A stationary box was attached to the table and subject's femur to maintain 90° of hip flexion during testing. Angular displacement (°) was measured as the maximal knee extension angle from the starting knee position of (90°). Only dominant side measurements were recorded. Data used for analysis was the mean of the subject's third and fourth trial (7). Data were used to assess the extensibility of the hamstrings.

**Peak Force Production**

Peak force production (PFP) is the maximum voluntary isometric force produced during the muscle action. PFP was measured using the Kin Com 500 H dynamometer (Chattecx Corporation, Chattanooga TN) at three flexion angles (30, 60, 90°). Three angles were used for the isometric testing to best simulate the overall range of motion of the knee joint. In studies done to compare muscle force characteristics between isometric and isokinetic muscle contractions, strong relationships have been found between the two conditions. (31,32). From this, we can assume that the isometric testing data can reasonably carry over to physical activity.

Subjects were seated with their dominant leg strapped to the dynamometer arm. Each subject performed three voluntary isometric contractions of the quadriceps at each set angle, held for two seconds. Following these sets, each subject performed three voluntary isometric contractions of the hamstrings at each set angle, held for two seconds. A 30-second rest was given between all sets. The values were recorded as peak force in Newtons (N). A composite peak force
production value was calculated as the average score of the quadriceps and hamstrings force production at the three angles. This composite value was recorded as the criterion measure for comparison.

**Peak Rate of Force Production**

Peak rate of force production (PRFP) is the steepest slope of the force-time curve, and represents the muscle’s ability to rapidly generate force or tension (N/s). In order to determine this value, raw data from the force-time curve was stored using an executable program from Visual Basic 4.0 software.

**EXPERIMENTAL PROCEDURES**

In order to prevent muscular injury, the American College of Sports Medicine recommends that a warm-up be performed before any vigorous exercise to minimize the risk for muscle injury and prepare the muscles for exercise. Each subject was required to pedal at a low intensity (60 rpm) for five minutes before each condition using a stationary bicycle ergometer (Monark).

**Core Body Temperature Assessment Protocol.**

Core body temperature has been defined as the temperature of the hypothalamus. Tympanic temperature readings are accepted as accurate representation of core body temperature. This is due to the close location of the ear from the hypothalamus (27). Tympanic body temperature readings were taken during the active warm-up and combined protocols. Temperatures were recorded at the initiation of cycling, at 5 minutes, and the instant the 10 minutes of active warm-up were completed. All readings were recorded. We can assume that an increase in core temperature was achieved due to the initiation of sweating (27).
We cannot assume that an increase in core temperature correlates with muscle force or range of motion due to the inaccuracy of the readings.

**Active Knee Extension Test Protocol.**

After the last treatment, the patient was positioned supine on a treatment table and stabilization box was positioned to maintain the dominant hip flexed at 90° for testing. The pelvis and non-dominant leg were stabilized to the table with Velcro straps to eliminate any movement that could skew the range of motion test. The baseline zero was then set and locked on the flexometer and the apparatus was strapped onto dominant leg directly over the fibular head. Subject was instructed to actively extend his/her knee, with maximum extension signaled by the maximum angle that the patient could hold for five seconds. Flexometer readings were recorded at that point. Four trials were performed with a 30-45 sec rest period between sets. Data from third and fourth trials were averaged for analysis.

**Kin-Com Isometric Testing Protocol.**

Each subject was seated upright with dynamometer arm on the side of the dominant leg. The chair was adjusted so that subject’s knee joint is at end of the seat. The subject was stabilized in chair with Velcro strapping around dominant leg and torso. Chair distance from dynamometer arm was adjusted so that the knee joint line was even with the axis of rotation of dynamometer arm. The dynamometer arm was strapped to tibia at the level of two finger widths (approximately two inches) above medial malleolus. The dynamometer protocol for isometric contraction at 30, 60 and 90 degrees of knee flexion was initiated. Three trials of 2-second isometric quadriceps contractions at each angle were completed, with 45 seconds rest between contractions. The protocol for isometric hamstring contractions was then initiated for the three angles. Subject data from the test were saved both on the hard drive of the dynamometer computer and floppy disc under the file of treatment conditions and subject identification number.
STATISTICAL PROCEDURES

Three separate one-between (condition) and one-within (time) univariate ANOVAs with repeated measures were used to reveal statistically significant mean (± SD) differences between treatment conditions over time. Scheffe post hoc analysis was used to identify significant interaction effects. Statistical significance was set at 0.05. With 17 subjects, four trial repeated measures, the significance level set at 0.05, and an expected effect size of 1.1, a statistical power of 0.80 was estimated (21,35,36,39-42).

Inter-trial reliability was determined from intra-class correlations obtained from the repeated measures. Data was analyzed using Abacus Concepts, Statview software.
RESULTS

HAMSTRING EXTENSIBILITY
ANOVA revealed no statistically significant difference between treatments \[ F(3,48) = 1.2, p = 0.32, \text{ICC} = 0.94 \] of knee extension range of motion as measured by the AKET.

MUSCLE FORCE PRODUCTION
ANOVA for peak force production revealed no statistically significant difference between treatments \[ F(3,303) = 2.5, p = 0.057, \text{ICC} = 0.98 \]. ANOVA for peak rate of force production revealed a statistically significant difference between treatments \[ F(3,303) = 2.7, p = 0.046, \text{ICC} = 0.90 \]. Post hoc comparisons revealed that peak rate of force production was significantly less for the stretching condition \( p < 0.05 \) compared to the other three conditions.
Table 1: Mean (±1 SD) data for the Active Knee Extension Test (degrees)

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<thead>
<tr>
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<th>Mean Value</th>
<th>SD</th>
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<tr>
<td>Warm-Up</td>
<td>60.24</td>
<td>10.94</td>
</tr>
<tr>
<td>Stretching</td>
<td>58.12</td>
<td>10.74</td>
</tr>
<tr>
<td>Combined</td>
<td>58.35</td>
<td>10.76</td>
</tr>
<tr>
<td>Control</td>
<td>56.91</td>
<td>12.32</td>
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Figure 1: Mean (±1 SD) data for the Active Knee Extension Test (degrees)
Vertical Lines indicate 1 SD
Table 2: Mean (±1 SD) values for Peak Force Production (N)

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<th></th>
<th>Mean Value</th>
<th>SD</th>
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<tr>
<td>Warm-Up</td>
<td>585.7</td>
<td>270.5</td>
</tr>
<tr>
<td>Stretching</td>
<td>608.4</td>
<td>285.6</td>
</tr>
<tr>
<td>Combined</td>
<td>583.3</td>
<td>277.3</td>
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<tr>
<td>Control</td>
<td>604.8</td>
<td>296.0</td>
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Figure 2: Mean (1 SD) values for Peak Force Production
(N) Vertical lines indicate 1 SD
Table 3: Mean (±1 SD) for Peak Rate of Force Production (N/s)

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<th>SD</th>
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<tr>
<td>Warm-Up</td>
<td>375.04</td>
<td>182.71</td>
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<tr>
<td>Stretching</td>
<td>346.42</td>
<td>132.68</td>
</tr>
<tr>
<td>Combined</td>
<td>360.86</td>
<td>148.07</td>
</tr>
<tr>
<td>Control</td>
<td>376.30</td>
<td>152.50</td>
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Figure 3: Mean (1 SD) for Peak Rate of Force Production (N/s)
Vertical lines indicate 1 SD
Asterisks indicate statistical significance
DISCUSSION

We hypothesized that a pre-exercise warm-up and/or stretching routine would significantly increase hamstring extensibility and muscle force production compared to a control (no treatment) group. Additionally, we expected to find no significant difference between the three treatment groups. Our findings revealed no significant improvement in hamstring extensibility or muscle force production as a result of the three treatment conditions (warm-up only, stretching only, or combination of warm-up and stretching). Our results also show no significant difference between the three treatment conditions.

HAMSTRING EXTENSIBILITY

We expected hamstring extensibility to increase significantly due to the theory of stress relaxation and creep (25). We assumed that an active warm-up of moderate intensity and duration would increase tissue temperature sufficiently, thereby making the hamstrings muscle group more elastic (or less stiff). In addition, we felt that the application of a static stretch would increase extensibility because of autogenic inhibition and subsequent muscle fiber elongation. We also assumed that our applied force of 10% body weight (BW) during the static stretch was of sufficient magnitude to activate the Golgi tendon organ (GTO) inhibition reflex. Several plausible explanations as to why our hypothesis was not supported by the experimental model will be provided.

The inability to detect differences in hamstring extensibility may have been due to the variability of flexibility in our subject pool. Most other studies restricted their subject pool to individuals who were inflexible or had less than average joint ROM (43-46). We did not set inclusion criteria for our subjects based on level of flexibility. Using a more homogeneous sample may have eliminated the
confounding influences of inter-individual differences and ceiling effects, and therefore may have assisted in detecting significant between-subject differences.

Other possible sources of error may have been the type of stretching technique, duration of the stretch, and the magnitude of applied force. We used a static stretching technique held for 15 seconds (4 repetitions) with an applied force of 10% BW.

Proprioceptive neuromuscular facilitation (PNF) stretching techniques have gained wide acceptance in the sports medicine community. In addition to applying a static stretch to the muscle group, PNF techniques use a “contract-relax” or stress-relaxation technique aimed at decreasing the inhibiting effects of muscle tension (guarding) on tissue elongation. One possible explanation for our results is that the static stretching technique in our study was not adequate for producing sufficient muscle relaxation. Research is not conclusive as to which technique is most effective in producing increased range of motion. PNF stretching has been shown in several studies to be superior to static stretching for producing gains in ROM (21, 47-49), while other studies demonstrate no significant difference between the two techniques (7,10-12,34,48).

The stretch duration is another important factor that has varying opinions in the literature. Bandy et al. (44) found that for best results, static stretches should be held for at least 30 seconds. More recently, Bandy et al. (43) found no significant difference in ROM gains using various stretch durations. Other researchers have found that ROM gains are most detectable within the period of 15-30 seconds (5,12,13,29,30). Our duration of 15 seconds per stretch, for four stretches of each muscle group may not have been enough to induce sufficient hamstring extensibility. Future research should include static stretching of varying durations to detect if any differences due to duration are present.

Our method of force application for the static stretching treatment was based upon a pre-determined force of 10% of subject’s body weight (BW). Most studies use applied forces equal to the subject’s perceived level of “tightness” or “mild discomfort” (43,51-53). These forces are subjectively derived and therefore
not consistent or reproducible. Our protocol was chosen to provide objectivity and internal consistency of the treatment between conditions and days of testing. However, our chosen force level of 10% BW may not have been great enough to cause significant viscoelastic effects. In contrast, stretching a muscle to the point where the tensile force causes elongation may produce a reflex muscle guarding effect, thus preventing any gains in ROM. Our applied force of 10% BW may not have been a sufficient tensile load to cause tissue elongation. We may also contend that the applied force may have been too large, therefore invoking a muscle guarding protective reflex in order to prevent muscular injury. Moore and Hutton (48) found that EMG activity increased with increased discomfort. This increase in EMG activity suggests that muscle contractions (reflex guarding) were initiated in response to the high level of discomfort in order to protect the muscle from injury. Whether our applied force was too small or great remains speculative.

Recent studies used applied forces to the subject’s perceived level of “tightness” or “mild discomfort” (43,51-53). The advantage of this technique is that the force is tailored to the individual’s level of flexibility and pain tolerance. This technique ensures that sufficient resistance is being maintained while at the same time minimizing the subject’s pain or discomfort level. Future research should determine which technique is more effective in producing increases in range of motion.

Static stretching, in theory, assumes no or minimal muscle activity. There are several recent studies conducted on passive hamstring stretching that included EMG activity as a dependent variable (45,48,51,53-58). Several researchers report EMG decreases with stretching (48,49,59-61,63). Others report that EMG activity increases (45,48,49,64), or does not change (46,51,53,56,58) with stretching. McHugh et al. (63) found that EMG activity increased at the subject’s maximal level of tolerance to stretch and decreased over the duration of the stretch (45 seconds). This finding may be applied to our study in that our stretch may not have been held long enough to let these initial increases in EMG activity decrease. Therefore, a lack of stress/creep relaxation may be a causative factor in our study.
Researchers have begun to investigate the effects of pain tolerance on ROM gains. Magnusson et al. (53-58) has attributed gains in ROM to both viscoelasticity and pain tolerance. He contends that for an increase in range of motion to be due to viscoelasticity, passive tensile force must be measured. If an increase in joint ROM is detected in the presence of the same tensile force as the previous stretch, it can be assumed that increased ROM is due to an increase in pain tolerance. However, if the joint is held at the same angle as previous testing and the tensile force is decreased, it can be assumed to be due to viscoelastic elongation. Results from their later studies as well as those from other researchers support their claim (46,52,53,56,57,60,65).

The majority of research on stretching evaluates the effect of stretching over consecutive days of a stretching protocol on a muscle's length, not a one treatment session such as this study (43,44,59,66,67). Because of this, the conclusions made in this study of hamstring extensibility should not be taken out of context. There are recent studies that found no effects from acute stretching (68,69). The results of our study agree with this literature, but we are limited to the conclusion that one session of static stretching was not sufficient to create significant gains in hamstring length.

FORCE PRODUCTION

We hypothesized that warm-up and stretching would significantly enhance muscular force production characteristics. We postulated that warm-up would increase intramuscular metabolic processes, otherwise known as the Q10 effect (23), which in turn would increase energy sources available to working muscle causing an increase in force production. Stretching was thought to improve muscle force production by increasing the functional range of motion of the joint performing
work. Also, a greater potential for cross-bridge formation would be expected with the muscle in a lengthened state.

In light of our expected outcomes, our findings revealed no significant increase in force production as a result of warm-up and/or stretching. However, our results did show a significant decrease in the peak rate of isometric muscular force production after the treatment of stretching alone. A review of the available literature reveals conflicting data; however, most published reports indicate a decline or no change in force production characteristics from stretching (59,70-73).

Kokkonen et al. (70) studied the effects of acute stretching on force production of knee extension and flexion. They found that force production significantly decreased after stretching when compared to a no stretching treatment. They theorized that a stiff musculotendinous unit (MTU) is a more effective mechanical system for muscle contraction (70). By stretching the MTU, a significant reduction in stiffness resulted. Similarly, Wilson et al. (71) found that stretching significantly reduced bench press performance. They indicated that the increase in compliance (decrease in stiffness) of the muscle and connective tissues from stretching diminishes the efficiency of the contractile mechanism (71). Hortobagyi et al. (59) studied the effects of stretching over a 7-week time period, and found that the maximum voluntary isometric contraction decreased, while hip flexion and extension range of motion increased. Viktorsson-Moller et al. (72) studied the effects of warm-up and stretching on force production of isometric and isokinetic knee flexion/extension. They found no difference compared to a control condition. Bohannon and Gibson (73) found that knee extension torque did not increase after quadriceps femoris stretch.

Other studies have demonstrated an increase in force production as a result of stretching and/or warm-up. Worrell et al. (74) was able to show that stretching the hamstrings increased isokinetic muscle performance. However, they detected no statistically significant increase in hamstring flexibility. Similarly, Thomson and Chapman (75) found that stretching momentarily increased contractile properties in forearm musculature.
The concept of muscular stiffness has received considerable interest recently. Current experimental models have characterized muscular stiffness by dividing the muscle’s change in force (torque) by the change in length (displacement). We used the peak rate of force production as our quasi-stiffness experimental model. By using an isometric action, the length (displacement) was held constant, while the rate of force production quantified the change in force over time. Therefore, using our quasi-stiffness model, a higher peak rate of force production measure is indicative of increased muscular stiffness.

The ability to generate muscle force quickly is dependent upon the rate of motor unit activation, reflex motor unit stimulation, and recruitment of high threshold (large) motor units (76). Both reflex motor unit stimulation (H-reflex) and motor unit recruitment (EMG) have been measured in stretching and warm-up studies (45,49,60-61,63,77).

Several studies, including our current study, have found that stretching causes neural inhibition resulting in decreased stiffness (45,49,60,61,64,77). We found that stretching had a negative effect on the rate of muscle force production. Guissard et al. (77) demonstrated neural inhibition (H-reflex suppression) during repeated bouts of stretching. Interestingly the inhibition was quickly reversed after the stretch was released. Thigpen et al. (78) in a similar study was able to show that the H-reflex remains depressed over time in the triceps surae. Condon and Hutton (45) compared the H-reflex of static stretching versus PNF techniques. They found that H-reflexes were lower in some PNF forms than static stretching. Rosenbaum and Hennig (61) found that EMG activity during warm-up or stretching of the Achilles tendon decreased significantly when compared to no treatment condition. Avela et al. (79) found that the H-reflex decreased by eight percent following repeated passive stretch. In addition, they found that maximum voluntary contractions decreased an average of 20%. Collectively, we are able to view these findings as evidence that static stretching of moderate to high intensity has an inhibiting effect on muscle force production characteristics. Specifically, we postulate that stretching activates high threshold GTOs, thereby inhibiting alpha
motor neuron facilitation to the target muscle. As a result motor unit activation is suppressed resulting in a diminished ability to generate force quickly.

Coincidentally, we found it peculiar that a significant increase in hamstring extensibility did not correspond with the significant decrease in force production. Our assumption was that with the decrease in motor unit facilitation, the muscle would be more relaxed and therefore exhibit increase extensibility. More research needs to be done in order to determine the relation between the effects of stretching on force production and tissue extensibility.

Our data also indicate that an active warm-up does not significantly enhance muscle force production. One possible explanation is that the warm-up routine used by our subjects was not intense enough to cause a significant increase in body temperature. A similar study by Cornwall (21) found no significant increase in force production of the wrist extensors as a result of superficial heat application. Tymppanic readings taken in this study were too sporadic to be considered accurate reflections of each subject’s true core temperature during warm-up. The actual change in tissue temperature from the warm-up was not accurately measured in our study, therefore our comments remain speculative.

We also cannot discount the possibility that the combined effect of stretching and warm-up caused some mild muscle fatigue, thereby decreasing muscle force production. We used a moderate intensity for the warm-up treatment that utilized the large lower extremity muscles to increase the heart rate and body temperature. This amount of work may have tired the quadriceps and hamstrings before the maximum performance testing.

We cannot assume that the results we collected using an isometric testing protocol were as effective as an isokinetic testing series. A full range of motion for testing would better mimic athletic performance. Several authors have stated that there are correlations between isometric readings and low speed isokinetic testing (31,36, 80). Isometric testing cannot be assumed an accurate replacement for isokinetic measurements. Further studies will clarify whether isokinetic testing will find different results.
CONCLUSIONS

From the results of our study, we conclude that pre-exercise warm-up and/or stretching does not significantly improve hamstrings extensibility or force production in non-power-trained individuals. Our results do, however, suggest that pre-exercise stretching cause a decreased rate of force production. This may possibly be from a neural inhibition effect from GTO stimulation.


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APPENDIX A
LITERATURE REVIEW

INTRODUCTION

In the sports medicine and athletic arenas, a combined stretching and warm-up routine is considered essential not only for the prevention of injuries, but as the extra edge that allows athletes to reach their body's maximal physiologic capabilities. Although these factors have not been proven to enhance performance and increase range of motion, there is literature that supports pre-exercise warm-up and stretching routines.

Warm-up is used to increase blood circulation in order to deliver oxygen and other nutrients to working tissues, and to increase muscle tissue temperature. Stretching is used to increase soft tissue extensibility, which causes an increase in range of motion (ROM) of joints. Health and physical education associations such as the American College of Sports Medicine promote stretching and warm-up prior to and after activity as a means of preventing musculotendinous strains. An increase in joint ROM is believed to decrease the number of muscle and tendon tears associated with physical activity, although there is little scientific proof to support these claims of injury prevention. The studies completed lack any correlation between increased range of motion and heightened performance. However, known human physiologic processes, and characteristics of skeletal muscle structures during exercise support the theories behind these claims.
CONNECTIVE TISSUE

There are five main types of human connective tissue. Blood is considered the liquid connective tissue, bone is the rigid structural connective tissue, cartilage is the avascular, aneural connective tissue that endures more stress than the other types of connective tissue, and loose connective tissue are structures such as adipose cells and tissues that comprise internal organs such as the kidneys. The last of the five types, dense connective tissue, is the structurally significant connective tissue that has the capabilities of skeletal movement, rigidity, and elasticity. There are two subtypes of dense connective tissue. Regular, dense connective tissue has an orderly parallel arrangement that resists tension in one direction. This is the structure of tendons, ligaments, and aponeuroses. Irregular dense connective tissue does not have an orderly arrangement. The fibers are in a weblike meshwork instead of a patterned arrangement. Irregular tissue is designed to stabilize other structures within the body’s arrangement. It comprises the fascia that surrounds muscle fibers and groups of muscles, the periosteum, and joint capsule (81). For the purposes of this study, the two types of connective tissue under examination will be regular and irregular dense connective tissue. These structures are involved with the joint range of motion through both the restrictions caused by the joint capsule itself, as well as the fascia and tendons that involve the muscles connecting to the joints under stretch.

Joint motion during exercise is structurally due to the anatomical unit known as muscle. To describe the contractile and lengthening properties of muscle, the tendon must be taken into consideration as being part of the active segment, thus together the muscle and tendon are defined as the musculotendinous unit (MTU). The active components of the MTU are two proteins called actin and myosin whose form and function are responsible for muscle action when broken
down into the smallest workable unit. The passive structure of muscle is the webwork of connective tissues that play no active part in muscle contraction.

Deep fascia of irregular connective tissue is extensive within the body. It holds muscles together into functional units, so that the contractions are more efficient for movement. Attached to this deep fascia are three parallel layers of connective tissue that both comprise and support the muscle structure. The entire muscle is wrapped with a substantial quantity of irregular dense connective tissue called the epimysium. Surrounding bundles of muscle fibers, holding them together in groups as well as creating workable units of muscle tissue is the sublayer called the perimysium. Finally, surrounding each individual muscle fiber is the smallest layer of connective tissue called the endomysium. Each of these layers is continuous into the muscle tendon, creating the connective tissue support of the overall structure of the musculotendinous unit (81).

Muscular and tendinous connective tissue is described by the term viscoelastic. This term depicts the duality of the tissue to react with both elastic and liquid behaviors. Elastic defines the deformation of the structure that is directly proportional to the tensile force applied. More generally, it describes the ease of which the compound deforms and returns to its original state. Viscous deformation is when the rate of deformation, not the deformation itself, is directly proportional to the forces applied. Like a liquid, the deformation is plastic, where there is no return to the previous form (3,4,9,10,23,63).

Increased tissue temperature positively affects viscoelastic tissues. The elastic and plastic components are both thought to increase in extensibility when heated (11,82). Collagen, the major component of connective tissue, although stiff at normal body temperature, becomes pliable in the range of 102-110 degrees Fahrenheit (°F) (83). The proportions of elastic and plastic reactions to stretch therefore depend upon tissue temperature, the amount of tensile force applied, and the duration that the force is applied (10). Studies have shown that with an increase in temperature of one degree Celsius (°C), physiologic changes in muscle, tendon, and connective tissues occur (5).
Muscle activity is temperature dependent. Peak tension has a negative correlation to temperature (22). For all mammals, peak force production, also called twitch tension, occurs at the normal body temperature of 20 °C. Tetanic or constant state tension has been shown to decrease at any temperature below 25 °C. Overall, the rates of force generation muscle contraction and relaxation, and the tension produced on contraction are effected by temperature variance (84,85). The increase in temperature causes more efficient actions by the contractile elements. The cross bridges of actin and myosin are thought to form and break at an increased rate, which causes faster tension generation (86). In a study of the function of the hamstrings in elderly men, both the peak muscle force production and the rate to that peak force increase significantly when heated (18).

No conclusions can be made as to whether temperature increases within muscle positively affect muscle performance, yet the majority of studies show that the possibility does exist. Davies et al. (19) found that the speed of force production was greatest at the increased temperature of 39 °C. In another study by Cornwall (21), forearm musculature, heated by warm water immersion created no difference in peak muscle force or rate to peak force.

To illustrate viscoelasticity into a workable model, Taylor et al. (9) describes these properties using two different physical examples. The elastic component is described as Hooke's model of the perfect spring. The length of the spring is directly proportional to the amount of tension pulling on it. The viscous characteristic is described using Newton's model of a hydraulic piston called a dashpot. The liquid (viscous material) controls the change in position of the piston due to the inherent attraction between liquid molecules. It is a time dependent characteristic, where the speed at which the tensile force is applied determines the final length. Within one segment of connective tissue, elastic and plastic behaviors are present. Together, these two models explain the reaction of muscle tissue to different loads and different applications of those loads (9). The research objective for stretching is to find the conditions and load that provide both the greatest gains in joint range of motion and tissue extensibility.
There are three major qualities of viscoelastic tissue. Stress relaxation and creep deformation describe in greater detail the result of the elastic and plastic qualities. Stress relaxation is the decrease in tension that occurs when a constant load is applied (9,87). Creep deformation is variation in the length of the tissue that occurs with a constant amount of tension. The third quality, hysteresis defines the transfer of heat that occurs during the deformation of the connective tissues. Within the MTU, more energy is absorbed within the tissues when the tissue is stretched than is released when the load is withdrawn. Current studies have proven creep deformation and stress relaxation using laboratory stretching techniques similar to the static stretching routines commonly performed by athletic trainers. The results imply that slower stretches allow for more relaxation of the connective tissue (i.e. more plastic deformation) causing an increase in joint ROM (5,9,12,87).

WARM-UP

Warm-up is defined as either passive or active. Increase in body temperature can be caused by an external influence, or by the energy released within the body from active participation of muscles. Warm water immersion and moist heat packs are passive modalities that increase tissue temperature in a localized area while the subject is resting. Active warm-up is both a generalized and local increase in tissue temperature due to physical activity. Muscle activity and a localized increase in bloodflow cause the temperature increase within muscle tissue associated with active warm-up. Muscle actions create heat from the energy of activation, and the thermoelastic heat that is released when the muscle returns to a relaxed state (88). Although research has shown that passive warming causes physiologic effects on the MTU (4), active is often preferred over passive warm-up. This is because unlike active warm-up, superficial heat modalities are not capable of warming tissue beyond the most superficial layer (82).
Mean core body temperature increases significantly with a moderate to intense warm-up, and this increase in temperature is directly correlated to the increase in intensity (6). Warm-up causes a rise in tissue temperature in two ways: by the immediate release of chemical irritants, and the peripheral feedback to the central nervous system (CNS) that occurs over the first thirty minutes of physical activity. DeVries (89) states that there are rapid increases in working muscle temperature within the first five to ten minutes of exercise. This is due to an estimated 2.2 liters of blood redistributed from the circulation through the splenic area, kidneys, and skin to meet the increased demands of working muscle (90). To further support the theory of immediate muscle temperature increase, Asmussin and Boje (91) found that the majority of performance improvements occur when muscle tissue temperature is increasing at a rate much faster than the core temperature rate. Heat is the natural by-product of all biochemical reactions, including muscle activity, because these reactions are not 100% efficient (76). Approximately 75% of the energy from human metabolic reactions is lost to heat (76). During exercise, when more metabolic reactions occur to produce more work, a considerable increase in the amount of heat is released (76). Thus, it appears that the localized muscle temperature increase is the greatest benefit of general warm-up on performance enhancement. This reveals the importance of involving the same muscle groups in warm-up as the athletic event demands, for maximum efficiency.

The 75% inefficiency of metabolic reactions is somewhat counteracted by the positive effect that heat has on chemical reactions. Within any living tissue, a rise in temperature causes the rate of metabolic processes to increase by a factor known as the \( Q_{10} \) effect (22). Heat activates the enzymes that catalyze metabolic reactions. The assistance of enzymes decreases the level of energy necessary within the system to initiate any reaction, whether the net outcome is endothermic or exothermic. Within physiologic tissue the direct hydrolysis of ATP is responsible for the majority of useable energy within the human body. This exothermic reaction is positively influenced due to the \( Q_{10} \) effect. In association
with increased efficiency of energy transfer within biological tissue when heated, muscle tissue can perform with heightened activity and increase coordination (76).

Chemical irritants are also a factor in temperature regulation. During exercise, this occurs via the release of catecholamines from the adrenal glands (76). At levels of exercise above 50% VO\textsubscript{2} max, blood catecholamines increase dramatically. There is no significant rise in blood concentration below 50%, however, increases can occur via the sympathetic nervous system. In anticipation of a strenuous bout of exercise, the body prepares for the activity by releasing high levels of catecholamines. These high levels aid in cellular metabolism by speeding the metabolism of glucose for energy (76).

In a study of forearm and leg musculature, the tensile forces that the muscle could withstand before failure were measured to detect the effects of warm-up. Using isometric contractions, the study found that preconditioned muscles could withstand a greater maximal force, and could stretch to a longer length than muscles that have not warmed up (5). This was hypothesized to be due to the increase in temperature that occurs during muscle contraction. The temperature increased the extensibility of the collagen fibers of the muscular network of connective tissues (5).

The question of whether warm-up causes an increase in joint range of motion has been studied. It is logical to assume that with the increase in muscle temperature, the extensibility of the tissues would also significantly increase. Increased extensibility of the separate tissues is thought to cause generalized increases in total ROM. However, this has not been the case under research conditions. In one study, it was found that hip range of motion was significantly improved only at intense levels of activity above 80% VO\textsubscript{2} max, although trends were noted at 60-70% VO\textsubscript{2} max. This shows little support for pre-exercise warm-up, because it is rarely at such intense levels of activity. In addition, Cornelius and Hands found that five minutes of active warm-up did not increase ROM for PNF contract relax techniques (82).
Few studies have been done that focus on the effects of active warm-up on fast, exhaustive, anaerobic activity. The studies that exist not only evaluate anaerobic performance, but the level of warm-up that is most beneficial. Findings indicate that high intensity warm-up above 75% VO₂ max is detrimental (6, 16). Most researchers agree that moderate levels warm the tissues without decreasing performance (6,16,17). Two authors agree that with a period of no activity of five minutes between warm-up and testing the benefits from heat no longer exist (6,16).

Warm-up increases circulation, causing increased oxygen uptake. Although aerobic processes do not dominate in the initial stage of exercise, the presence of oxygen allows the anaerobic system to contribute to the energy supply for a longer period of time (6). High intensity warm-up diminishes the glycogen stores needed for anaerobic performance (20). In addition, it has been shown to cause greater lactate accumulation, which decreases pH levels. This acidic condition diminishes the membrane potential for the sodium potassium channels, which are needed for the contraction of the fast twitch type II muscle fibers which dominate in explosive activities (6).

STRETCHING

Stretching can be separated into three major categories: ballistic, static, and proprioceptive neuromuscular facilitation (PNF). The musculotendinous unit (MTU) is equipped with built in reflexive defense mechanisms to prevent micro-trauma from over-stretching. The ballistic method is comprised of a series of bounding movements at the endpoint of a joint’s range of motion. This method is not considered acceptable stretching (92). The MTU reflexes are triggered by bouncing to contract and shorten the muscle. Mechanical lengthening coupled with the shortening caused by muscle contraction causes a greater risk of injury (4).
Static stretching inhibits the neurological reflex by using gentle, slow, sustained tension that stretches a muscle to a point just short of discomfort. It is usually done on a subjective level, where the patient’s perception of pain is the sign for the optimal point of stretch. Static stretching is the most common method utilized in the athletic setting. This is both due to the widespread acceptance of the method, and the fact that the positive results can be accomplished when athletes stretch themselves.

The theory behind PNF techniques is to utilize the neurologic mechanisms for increased muscle tissue length. PNF uses the inverse stretch reflex mechanism of the Golgi tendon organ, whose activation causes relaxation and inhibits damaging muscle contraction, to cause further muscle lengthening (4,63). There are several methods, which use a series of stretching with contractions of agonists and antagonists to maneuver increased muscle tendon stretch. Although various methods and variations of stretch have been studied, the basic biomechanical properties that are responsible for increased muscle tissue extensibility have been addressed little in sports medicine (4,9).

In theory, both the MTU and the surrounding connective tissues are responsible for defining the limits in range of motion. Only a small percentage of gains occur due to the MTU lengthening (29). Most immediate progress measured from stretching is temporary and due to the transitory lengthening of the actin/myosin complex (4,29). Johns and Wright (62) state that joint capsule and skin compose 49% of resistance to ROM gains, and muscle and tendon compose 51% of resistance (62). The dramatic viscoelastic properties of the connective tissues surrounding and within the muscle structure are responsible for the majority of the results.

Tension within muscle tissues is separated into active and passive qualities. The passive component can be defined as the starting length of the MTU. The active component is the tension placed upon the tissue itself either by the stretch placed upon it, or the contraction of the muscle from the elements within its structure (9,29). It is the structures that are more easily influenced by heat and
external stresses whose elongation is dominant. The viscoelastic properties of connective tissue make it the major initial contributor to gains in ROM. In addition, when the muscle is not in a state of contraction, the dominant resistance to tensile stresses is the connective tissue. Starring et al. (10), states that the spider-web of connective tissue that runs throughout the muscle and the sheath that surrounds it creates most of the resistance to the stretch. An increased percentage of connective tissue within the muscle structure creates more plastic, thus, permanent elongation of the muscle. This is evident when comparing genders. In a study of hamstring flexibility, females demonstrate greater gains in ROM, and are known to have a greater percentage of connective tissue when compared to males (10).

Tendons exhibit immediate gains in length that are directly proportional to the tension load applied. To achieve maximal permanent lengthening, tendons respond best to smaller loads applied over extended periods of time (87). For permanent MTU lengthening, the stretching session must consist of multiple repetitions of stretching (10,29). The duration of each stretch, to provide maximal gains in ROM, have been stated anywhere from ten seconds to twenty minutes. Several studies suggest that the greatest gains in stretching are due to the connective tissues, which respond significantly within 15-40 seconds (5,12,13,29,30). The majority of the gains in ROM made within a stretching session are due to the first four sets, which explains the 15-20 minute length of time for the session (5,12,13).

The rate at which the stretch is applied to the muscle is also important (29). Lamontagne et al. (92) found that the faster the load was applied, the more the tissues acted with resistive torque. Any speed faster than 60 degrees per second initiated this response which was proportional to increasing speed. There is more tension produced within the tissue to counteract stretch when the tensile load is applied at a faster rate (9,10).

Although PNF is thought by many to be the best method of stretch, current research has compiled data that refutes those beliefs. By comparing innervated and
deinnervated muscle fibers, Taylor et al. (9) found that there are no significant differences in elongation due to neurological influences. Another study comparing static stretching with mid-range PNF of the hip found that PNF subjects experienced only 50% of the gains of static stretching (14). From these findings, it is suggested that PNF is a good technique to be used with neurological and orthopedic conditions, where placing the joint at its endpoint is contraindicative. With healthy athletes, static stretching is recommended over PNF (14). Sullivan et al. (41) concluded in their study of hamstring flexibility methods that anterior pelvic positioning was a more significant factor for gains in range of motion than the method of stretch used. They suggest that the gains in range of motion with PNF techniques that are greater than static stretching are due to the viscoelastic effects of the greater forces exerted within the MTU rather than the Golgi tendon organ inhibition (41).

Current research with isometric contractions further refutes the claims of neurological inhibition. Taylor et al. (12) found that both isometric contractions and tensile stretches of the same force produce the same amount of elongation. Safran et al. (5) found the same results and contributed it to stress relaxation of the passive structures. During an isometric contraction, which is defined by a contraction with the length of the muscle maintained, the contractile elements shorten and the tendons are fixed at their origin and insertion, so to maintain the length, the connective tissue must elongate (5,12). This is supported by another study, who found that the process of stretching creates elongation in passive structures regardless of the type of load applied. When comparing isometric contractions and stretching protocols, the only common denominator is connective tissue lengthening, which reaffirms the stress relaxation theory (13). Rather than neurological inhibition of the Golgi tendon organs as the mechanism for benefits of contract-relax techniques, it can be hypothesized that it is instead the addition of isometric contractions added to the static stretching which causes some further lengthening of the connective tissue structures (12,13).
COMBINED STRETCHING AND WARM-UP

Few studies have been done that analyze combined pre-exercise methods. Results from these studies are a good basis for further research, but their limited findings cannot be used as facts to support the methods of pre-exercise activity. In one study by Asmussen et al. (91), flexibility of the hamstrings was measured after warm-up and stretching. A significant increase in overall range of motion was measured in the combined group. Henricson et al. (11) found that the combination of heat and stretching increased hip flexion ROM. In addition, they found that these results were maintained thirty minutes after the treatment was finished.

In a different study that distinguished the effects of warm-up and stretching on hip range of motion, it was concluded that warm-up had little to do with the increased movement of the hip. In this study, the stretching group significantly increased ROM when compared to the warm-up and the combined groups (15). This suggests that tissue temperature and collagen extensibility may not have as much to do with increased joint motion as is commonly thought.

SUMMARY

The study of the significance of pre-exercise preparation is presently in the experimental stage. There are many theories of what works best, and there is strong physiological basis behind these theories. The lack of strong evidence to support any claims suggests the strong need for further research in this area. With more background data, it will be possible to distinguish why pre-exercise activity is important, and will define the best protocols for maximum injury prevention and athletic performance. At this time, it is recommended that some method of pre-exercise activity is important for athletic competition. Further research will prove
what athletes, coaches, and athletic trainers have suspected for many years: that warm-up in some form enhances athletic performance.
APPENDIX B
ADDITIONAL METHODS

INFORMED CONSENT

A. Title of the Research Project.

The Efficacy Of Warm-Up And Stretching On Lower Extremity Muscle Force Production and Hamstring Extensibility In Non-Power-Trained Individuals

B. Investigators.

Principal Investigator: Paul Borsa, Ph.D., ATC, Assistant Professor
Department of Exercise and Sport Science, College of Health and Human Performance, Oregon State University

Co-investigators: Kimberly Frostad, ATC

C. Purpose of the Research Project.

The purpose of this study is to determine if a stretching, or warm-up, or combined warm-up/stretching routine performed before exercise significantly improves muscular performance.

D. Procedures.

I am being asked to participate in a controlled experiment because I am between the ages of 18 and 30 years of age with no history of any condition that should prevent me from heavy exercise, such as heart disease, or any musculoskeletal injury to my lower extremity in the past six months.

I will be asked to report to the Sports Medicine Research Laboratory on four separate occasions for testing.

For each session I will perform moderate intensity exercise on a stationary bicycle, maximal voluntary isometric contractions, and hip range of motion measurements using a modified straight leg raisetest.

I will be tested for muscular force production using four different experimental conditions which will be randomly assigned prior to my participation. The four conditions are warm-up, stretching, combined warm-up and stretching and no warm-up or stretching.
Before each condition I will be required to pedal at a low intensity (1 kp) @ 60-70 rpms for five minutes in order to minimize the risk for muscle injury and prepare the muscles for exercise.

For the pre-exercise warm-up I will pedal on a stationary bicycle ergometer for ten minutes at an intensity that is within my target heart rate range. My target heart rate range will be determined using the Karvonen formula.

For the pre-exercise stretch I will be passively stretched by an experienced partner who will use a static stretching technique for the major muscle groups of my lower extremity (hip and knee joint). The partner will stretch each muscle group using four repetitions. A preset tension load for the stretch will be determined by my body weight for each stretch.

For the combined routine I will perform the warm-up followed by the stretching routine.

For the control I will not perform any of the pre-exercise routines. However, I will be required to pedal at a low intensity 60-70 rpms for 5 minutes. This will minimize the risk for muscle injury and prepare me for the exercise.

For range of motion testing, active knee extension with 90 degrees of stabilized hip flexion will be measured using a Leighton flexometer.

I understand there are foreseeable risks or discomforts to me if I agree to participate in the study. The exercise protocol may produce transient light-headedness and/or nausea. In some cases, mild muscular soreness may result. I understand that this will not be significantly different from normal training discomfort.

I understand that as a benefit from my participation in this study, I will receive information concerning my ability to perform high-intensity exercise of short duration.

I understand that there are no feasible alternative procedures available for this study.

Any information obtained from me will be kept confidential. A code number will be used to identify any test results or other information that I provide. The only individuals who will have access to this information will be the investigators and no names will be used in any data summaries or publications.
I understand the University does not provide a research subject with compensation or medical treatment in the event the subject is injured as a result of participation in the research project.

I understand that my participation in this study is completely voluntary and that I may either refuse to participate or withdraw from the study at any time without penalty or loss of benefits to which I am otherwise entitled.

I understand that any questions I have about the research study and/or specific procedures should be directed to Dr. Paul Borsa, Langton Hall 223A, Oregon State University, Corvallis, Oregon, at 737-6787. Any other questions that I have should be directed to Mary Nunn, Sponsored Programs Officer, OSU Research Office, 737-0670.

My signature below indicates that I have read and that I understand the procedures described above and give my informed and voluntary consent to participate in this study. I understand that I will receive a signed copy of this consent form.

________________________________________
Signature of Subject

_________________________ _____________
Name of Subject Date Signed

_________________________ ____________________
Subject’s Present Address Phone Number

I certify that I have explained to the above individual the nature and purpose, potential risks, and benefits of this study. I have answered any questions that have been raised, and have witnessed the above signature. Also, I have provided the subject with a copy of this signed document.

________________________________________
Signature of Principal Investigator Date
PRETEST QUESTIONNAIRE

During testing sessions, physical exertion is required. The following questions are used to determine physical ability to perform required activities.

Name ________________________________

Age ________________________________

Gender (circle one)  M  F

Dominant Side (circle one)  Right  Left

Have you had any injury to your lower extremity muscles and/or joints within the last six months? If yes, please explain.

Do you participate in any physical activities on a regular basis? If yes, please explain.

This test requires a high intensity of physical exertion. Do you have any physical conditions that may affect your ability to perform physical activity? (i.e. Asthma, heart conditions, etc.)

Are you currently taking any medication that affects your level of physical activity? (i.e. heart medication, beta blockers, etc.)

Do you have any other concerns you would like to address?

I understand that I will be performing maximum effort testing under four separate conditions. I have understood and truthfully answered the above questions that pertain to my current health status.

______________________________  ________________________________
Name of Subject                Date
APPENDIX C
STRETCHING PROCEDURES

HAMSTRINGS

1. Subject lies supine on examination table.

2. Knee immobilizer is placed on dominant leg, maintaining leg in constant full extension.

3. Tension strap is placed on ankle of dominant leg just above malleoli.

4. Tension scale is attached to strap.

5. Subject flexes hips 45 degrees and knees at 90 degrees to flatten lumbar curve.

6. Pelvis is stabilized to table with snug strap over anterior superior iliac spine.

7. Non-dominant leg is stabilized to table with snug strap over mid-thigh region.

8. Examiner stands at subject's head, facing subject's feet.

9. Subject raises dominant leg, flexing at the hip so that examiner can grab tension device.

10. Tension is slowly increased to designated force.

11. Tension is held for 15 seconds.

12. Tension is released slowly and leg is returned to table.

13. Wait 30 seconds.

14. Repeat three times.
QUADRICEPS

1. Subject lies supine on examination table.
2. Tension strap is placed on ankle of dominant leg just above maleoli.
3. Tension scale is attached to strap.
4. Dominant side of body positioned so that edge of hip is aligned with edge of treatment table.
5. Subject flexes hips 45 degrees and knees at 90 degrees to flatten lumbar curve.
6. Pelvis is stabilized to table with snug strap over anterior superior iliac spine.
7. Examiner stands beside subject’s dominant side, at subject’s head.
8. Leg is hung over the edge of the table, with knee flexed.
9. Tension is pulled to designated force.
10. Tension is held for 15 seconds.
11. Tension is released slowly and leg is returned to table.
12. Wait 30 seconds.
13. Repeat three times.