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ACUTE AND CHRONIC EFFECTS OF PASSIVE STRETCHING ON VOLUNTARY AND EVOKED MUSCLE FORCE, THE LENGTH-TENSION RELATIONSHIP, ANKLE JOINT RANGE OF MOTION, AND MUSCULOTENDINOUS STIFFNESS IN THE PLANTAR FLEXORS

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ACUTE AND CHRONIC EFFECTS OF PASSIVE STRETCHING ON VOLUNTARY AND EVOKED MUSCLE FORCE, THE LENGTH-TENSION RELATIONSHIP, ANKLE JOINT RANGE OF MOTION, AND MUSCULOTENDINOUS STIFFNESS IN THE PLANTAR FLEXORS

A DISSERTATION APPROVED FOR THE
DEPARTMENT OF HEALTH AND EXERCISE SCIENCE

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"Education is not the filling of a pail but the lighting of a fire"

~ William Butler Yeats

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ABSTRACT

ACUTE AND CHRONIC EFFECTS OF PASSIVE STRETCHING ON VOLUNTARY AND EVOKED MUSCLE FORCE, THE LENGTH-TENSION RELATIONSHIP, ANKLE JOINT RANGE OF MOTION, AND MUSCULOTENDINOUS STIFFNESS IN THE PLANTAR FLEXORS

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The purposes of the present study were: (a) to examine the effects of prolonged passive stretching on the length-tension relationship under voluntary (maximal voluntary contraction (MVC) force) and evoked (single and tetanic twitch force) conditions, (b) to determine if four weeks of regular chronic stretching affects the magnitude of the stretching-induced deficit on MVC, single and tetanic twitch force, percent voluntary activation (% VA), electromyographic (EMG) amplitude of the soleus (SOL) and medial gastrocnemius (MG), range of motion (ROM), musculotendinous stiffness (MTS), and corrected calf girth, and (c) to examine the effects of acute versus chronic stretching on changes in ROM and MTS. Twenty-six healthy men volunteered for this investigation and were randomly assigned to either a stretch training (STR) group (mean±SD age = 22 ± 2 yrs; stature = 175 ± 8 cm; mass = 74 ± 12) or control (CON) group (21 ± 2 yrs;

stature = 176 ± 7 cm; mass = 76 ± 11) for four weeks. At baseline (week 1) and post-testing (week 5) all subjects completed a flexibility and strength (voluntary and evoked) assessment prior to and following 20 min of passive stretching of the plantar flexor muscles on a custom-built load cell apparatus attached to a calibrated isokinetic dynamometer. To determine ROM and MTS, the dynamometer passively dorsiflexed the foot at $5^{\circ} \cdot s^{-1}$ from -20º of dorsiflexion to the maximum tolerable ROM (as acknowledged by the subjects). For the strength assessments, subjects performed a MVC and underwent a single and tetanic stimulus at each randomly-ordered joint angle (-19º, -9º, 1º, and 12º of dorsiflexion, where 0º = neutral ankle joint angle). The stretch training included four 135-s constant-torque passive stretches 3 times per week for 4 weeks. At weeks 1 and 5, from pre- to post-stretching voluntary and evoked force decreased across all joint angles, ROM increased, and MTS decreased, however there were no changes in EMG amplitude or % VA. Following 4 weeks of stretch training, the STR group showed increases in voluntary (MVC) and evoked (single and tetanic twitch) force production at all joint angles, increases in ROM, decreases in MTS, and increases in MG EMG amplitude, but no changes in % VA, SOL EMG amplitude, and corrected calf girth. For the CON group, there were no changes in MVC force, ROM, MTS, %VA, SOL EMG amplitude, and corrected calf girth, but increases in single and tetanic twitch force and EMG MG amplitude. For the length-tension relationship, voluntary and evoked force increased from shorter to longer muscle lengths (19° to 1°) and decreased at the longest muscle length (12°), whereas % VA and EMG MG amplitude decreased from the shortest to the longest muscle lengths. There were no acute or chronic stretching induced changes in the length-tension relationship. The results of the present study suggest due to the lack of

changes in %VA and EMG amplitude and decreases in MTS from pre- to post-stretching, the stretching-induced force deficit may be more mechanical in origin. Chronic stretch training also does not appear to influence the stretching-induced force deficit or length-tension relationship. However, chronic stretch training appears to increase maximal force production at all joint angles, increase ROM, and decrease MTS in the plantar flexors with no changes in corrected calf girth. Therefore, it is possible chronic stretch training results in longitudinal hypertrophy (i.e. increased amount of sarcomeres in series), however future studies are needed to determine if there are in fact fascicle length changes following stretch training.

CHAPTER I

INTRODUCTION

Stretching has been traditionally recommended prior to exercise and/or athletic events with the intent to reduce the risk of injury and/or improve performance (65, 66, 68). However, many previous studies (16, 17) and recent reviews (61, 67) have suggested that pre-exercise stretching may have a detrimental effect on the muscle's ability to produce maximal force. For example, previous authors have reported stretching-induced decreases in isometric and isokinetic peak torque (6, 18, 25), vertical jump performance (9), and sprinting speed (55). As a result of these findings, previous studies (17, 18) have coined the term "stretching-induced force deficit" to describe the acute detrimental effects of stretching on performance measures. Although the precise mechanisms underlying the stretching-induced force deficit remain unclear, previous studies have hypothesized that it may be attributed to either "mechanical" or "neural" factors or a combination of both.

Evidence of an acute post-stretching neural deficit has been observed as decreases in muscle activation using both surface electromyography (EMG) and the twitch interpolation technique (8, 18, 25). Fowles et al. (25) was perhaps the first study to demonstrate the transient decrease in muscle activation after 30 min of triceps surae stretching. The authors (25) showed that maximal muscle activation was diminished after the stretching, and this factor accounted for 60%, 68%, 26%, 1%, 13%, and 13% of the stretching-induced force deficit at post, 5, 15, 30, 45, and 60 min, respectively, after

stretching. However, the specific causes of the neural deficit were not identified (25). A more recent study by Herda et al. (29) showed decreases in isometric peak torque, percent voluntary activation, and EMG amplitude following 20 min of passive stretching and Achilles tendon vibration. These findings indirectly indicated that prolonged stretching may cause neural deficits that are similar to the neural effects of prolonged vibration, which is thought to be caused by vibration-induced gamma loop dysfunction (29, 42). Therefore, the authors (29) tentatively suggested that prolonged passive stretching may also be related to gamma loop dysfunction. In addition, Cramer and colleagues (18) have reported decreases in isokinetic peak torque and EMG amplitude in both the stretched limb and the unstretched, contralateral limb, which the authors attributed to an unidentified central nervous system inhibitory mechanism.

On the other hand, alterations in the mechanical and/or contractile properties of the musculotendinous unit have also been linked to the stretching-induced force deficit. Nelson et al. (54) have suggested that stretching may increase the resting length of the sarcomeres, which may alter the muscle's length-tension relationship (28) and increase the rate of sarcomere shortening. Cramer et al. (16) reported that static stretching decreased isokinetic peak torque, but did not alter the total work or mean power generated during the isokinetic muscle action. This flattening of the isokinetic angle-torque curve may have indirectly reflected a stretching-induced change in the shape of the muscle's length-tension relationship. This hypothesis was recently supported by Herda et al. (28) and McHugh et al. (49) who reported joint-angle specific decreases in isometric torque production of the hamstrings following static stretching, which occurred only at the shortest muscle lengths. Therefore, it is possible that static stretching may

cause a rightward shift in the length-tension relationship that may reduce strength only at the shortest muscle lengths (28, 49). However, it is unclear how stretching affects muscle strength at the longest muscle lengths, because the decreases in muscle activation that seems to occur during voluntary muscle actions may limit the force-producing capabilities at all muscle lengths (49). Consequently, this factor was acknowledged by Behm et al. (8) who reported no changes in peak tetanic twitch force from pre- to post-stretching at a single joint angle, but proposed that alterations in tetanic force could be discovered at other joint angles. Therefore, more research is needed using both voluntary and evoked muscle force assessments at multiple joint angles.

Although acute stretching may result in detrimental decreases in muscular performance, chronic stretching programs have been shown to reduce musculotendinous stiffness (27) and improve isokinetic peak torque (79), maximal strength (41), and concentric bench press work (77). However, we are aware of only one study that has examined the effects of chronic stretching on the stretch-induced induced force deficit (5). These authors (5) reported that the magnitude of the stretching-induced force deficit in the leg extensors was unaffected by four weeks of chronic stretching. However, the authors did not examine how the chronic stretching program affected the length-tension relationship, muscle activation, or other underlying mechanisms. Therefore, there are three purposes of the present study: (a) to examine the effects of prolonged passive stretching on the length-tension relationship under voluntary and evoked conditions, (b) to determine if four weeks of regular chronic stretching affects the magnitude of the stretching-induced force deficit, and (c) to examine the effects of acute versus chronic

stretching on changes in range of motion and musculotendinous stiffness in the plantar flexor muscles.

Hypotheses

1. It is hypothesized that an acute bout of passive stretching will cause a right-ward shift of the length-tension relationship. Isometric MVC force will decrease at the shortest muscle lengths and remain unaltered at the longest muscle lengths under voluntary conditions. However, during a train of tetanic stimuli, there will be a similar decrease at the shortest muscle lengths, while twitch force at the longest muscle lengths will increase due to complete muscle activation and the right-ward shift of the length-tension relationship.

2. It is hypothesized that regular chronic stretching will have no affect on the magnitude of the stretching-induced force deficit (i.e. isometric MVC force, percent voluntary activation, and EMG amplitude) at a neutral joint angle (i.e., 90°) based on the findings of a previous study (5). However, it is unclear how chronic stretching may affect the potential acute right-ward shift of the length-tension relationship and the acute force losses at multiple joint angles under voluntary and evoked conditions.

3. It is hypothesized that the magnitude of the acute increases in dorsiflexion range of motion and musculotendinous stiffness after an acute bout of stretching will diminish following four weeks of regular chronic stretching.

Definition of Terms

MVC – a maximal voluntary isometric contraction that is performed for 5 s

Isometric MVC Force - the peak torque achieved during a maximal, voluntary, isometric muscle contraction; expressed in Newtons (N).

Percent Voluntary Activation – the percentage of motor units a person is able to voluntarily recruit compared to a non-voluntary evoked stimulation.

Surface Electromyography (EMG) – a recording of the muscle action potentials that sweep across the sarcolemma and pass through the surface electrode recording areas during a skeletal muscle action. The raw signal is expressed in microvolts (μV). A biopolar EMG electrode configuration results in a differentially amplified signal that represents the subtracted difference of the unique algebraic sums of muscle action potentials that pass within the recording areas of the two electrodes.

Range of Motion – degrees (°) of pain-free movement that a joint can sustain.

Musculotendinous Stiffness – the ratio of the change in passive resistance (torque or force) to the change in limb displacement (angle) during a slow ($5°\cdot s^{-1}$) controlled passive stretch. This is also mathematically defined as any tangential slope of the stress-strain curve recorded in vivo during a slow passive stretch.

Abbreviations

MVC – maximal voluntary contraction

%VA – percent voluntary activation

EMG – electromyography

ROM – range of motion

MTS – musculotendinous stiffness

SOL – soleus muscle

MG – medial gastrocnemius muscle

Delimitations

Twenty-four men between the ages of 18 and 30 years will be recruited for this study. All participants will complete a health history questionnaire and a written statement of informed consent prior to any testing and/or training. To be eligible for inclusion in this study, participants must be minimally physically active, which is defined as 1 – 7 hours per week of structured and/or recreational exercise, but they cannot be very active (defined as > 7 hours of exercise per week) or competitive athletes. Volunteers for this study must be free from any current or ongoing neuromuscular diseases and cannot have sustained an injury or had surgery to their thigh, leg, foot, knee, or ankle within the past 6 months. The participants cannot be currently (or recently; < 6 months) engaged in any regular resistance training program that involves the lower body, and they cannot begin any resistance training program during the four-week course of this study. Subjects cannot have taken (within 6 months) or begin taking any nutritional supplements or medications that the investigators believe may influence maximal strength, neural activation, or musculotendinous stiffness. A list of such supplements and medications may include, but are not limited to the following: creatine, beta-alanine, branched chain amino acids, and weight loss diet pills (i.e. Ephedra or pseudoephedrine).

Assumptions

Theoretical Assumptions

1. Subjects will accurately answer the health history questionnaire.
2. Five minutes of rest following pre-stretching strength and flexibility assessments will prevent the confounding effects of post-activation potentiation during post-stretching strength and flexibility assessments.
3. All equipment will function properly for all testing sessions.
4. The subject's knowledge of recent research findings regarding the acute effects of static stretching on strength and performance will not influence the outcomes of the post-stretching strength and flexibility assessments.

Statistical Assumptions

1. The population from which the samples are drawn is normally distributed.
2. The sample is randomly selected and the treatment order is randomly placed.
3. The data meets the assumption of sphericity. Sphericity requires that the repeated measures data demonstrate both homogeneity of variance and homogeneity of covariance.

Limitations

1. Subjects will be recruited as students from several departmental courses and responded to advertisements located within and around the Huston Huffman Center; therefore, the process of subject selection may not truly be random. In addition, the sample will be volunteers, therefore not meeting the underlying assumption of random selection.

2. Because of the time constraints of the post-stretching condition and the transient nature of the stretching-induced force deficit, only one isometric MVC and tetanic twitch will be conducted at each of the four joint angles prior to and following the acute stretching protocol. Although this may likely increase the within-subject variability of the measurements, this is a limitation that may be necessary given the purpose of this study.
3. The acute and chronic stretching protocol may not reflect every-day stretching, because it will only occur in one leg and it will be done in a modified dynamometer that most people do not have access to. This will limit the external validity of the stretching-related findings of this study.

CHAPTER II

REVIEW OF LITERATURE

The biomechanical effects of stretching on the muscle-tendon unit.

Taylor, Dalton, Seaber, and Garrett (1990)

The purpose of this investigation was to examine the acute effects of passive stretching on the viscoelastic properties of the muscle-tendon unit (MTU). A total of 60 rabbit MTU's (n=40, extensor digitorum longus; n=20, tibialis anterior) were used in this study. This study consisted of 3 parts: (I) viscoelastic response of the MTU during 10 repeated cyclic stretches to a pre-determined length (10% beyond its resting length), (II) viscoelastic response during ten 30-s static stretches to a pre-determined tension (78.4 N), and (III) viscoelastic response of cyclic stretching to a pre-determined length at varying rates (0.01, 0.1, 1, and 10 cm/sec) and while the MTUs were innervated and denervated. For part I, there was a 16.6% overall decrease in peak tension from the 1^{st} to 10^{th} cycle, however, only the first four stretches were significantly ($P<0.05$) different from the remaining stretches. Part II indicated that the stress relaxation curves for stretch 1 and 2 were significantly ($P<0.05$) different from the remaining stretches. In addition, the MTU increased by 3.46% of its initial length following the 10 stretches, with 80% of the length changes occurring during the first 4 stretches. The results for part III indicated that peak tensile force (N) and energy absorbed (N·cm) increased ($P<0.0001$) with increasing

stretch rates. However, the MTU responded similarly under both innervated and denervated conditions. These results suggested that the rabbit MTU exhibits viscoelastic properties during stretching techniques commonly used in clinical and athletic settings. Specifically, the decline in peak tension and increase in MTU length under the same tension demonstrate stress relaxation and muscle creep properties, respectively. It was also hypothesized (72) that ballistic stretching may be more dangerous than more traditional static stretching routines due to the greater peak tension and absorbed energy following the increased stretch rates.

Taylor, Brooks, and Ryan (1997)

The purpose of this investigation was to compare the effects of repeated muscular contractions and repeated passive stretches on the viscoelastic properties of the muscle-tendon unit (MTU). The tibialis anterior muscles of eight male rabbits were used in this study. One of the hind limbs was randomly assigned to the stretching (STRETCH) group, while the contralateral limb was assigned to the contraction (CON) group. Passive tension of each muscle was evaluated at a neutral length prior to and after the STRETCH or CON conditions. The STRETCH condition involved 10 repeated stretches from the muscle's shortest to largest length (in vivo). The CON condition involved ten 1-s stimulated contractions repeated every 10 s. Passive tension at a neutral length was decreased from 1.16 ± 0.17 N to 0.67 ± 0.09 N for the STRETCH group and from 0.88 ± 0.22 N to 0.42 ± 0.08 for the CON group. The decreases in passive tension were similar between conditions (P=0.24). These results suggested that the changes in passive tension were due to the magnitude of the load placed on the MTU and independent of how the

load is applied (i.e. centrally with an isometric contraction or peripherally with a stretch). The authors (71) hypothesized that the reduction in passive tension is connective tissue-mediated, because lengthening of the connective tissue is the only commonality between both conditions.

Morse, Degens, Seynnes, Maganaris, and Jones (2008)

The purpose of this investigation was to examine the changes in the muscle-tendon unit (MTU) during passive lengthening of the medial gastrocnemius (MG) and to determine which component of the MTU contributes to the increase in range of motion following an acute bout of repeated passive stretching. Eight healthy recreationally active men volunteered for this study. All subjects were secured in the prone position in a Cybex (Cybex Norm, Cybex International Inc., NY, USA) isokinetic dynamometer. The myotendinous junction (MTJ) of the MG was identified by B-mode ultrasonography while the ankle was dorsiflexed at $1^{\circ}\cdot s^{-1}$ to each subjects maximally tolerable end range of motion. Passive torque, muscle fascicle length, and pennation angle were also measured before and after five 1-min static stretches. Prior to the stretching protocol, increases in dorsiflexion range of motion resulted in a 2.19 cm increase in the length of the MTU. The muscle (proximal to the MTJ) and tendon (distal to the MTJ) accounted for 47% and 53% of the total elongation of MTU, respectively. Following the stretching protocol, range of motion increased by $4.6 \pm 1.5^{\circ}$ (17%) and passive stiffness was reduced from 16.0 ± 3.6 to 10.2 ± 2.0 Nm (47%). In addition, there was a 0.33 cm increase in the whole MTU at the end range of motion, with a 0.34 cm increase in the distal displacement of the MTJ, which accounted for the entire increase in elongation of

the MTU. However, the stretching protocol had no influence on the length or pennation angle of the fascicles. Increases in joint range of motion prior to the stretching protocol may have been attributed to similar increases in both the muscle fascicles and tendon components of the MTU. However, the increased length and decreased stiffness of the MTU following stretching may have been due to alterations within the muscle. The authors (53) hypothesized that these changes may have resulted from altered connective tissue properties (i.e. perimysium).

The acute effects of stretching on voluntary and evoked neuromuscular function, range of motion, and musculotendinous stiffness.

Fowles, Sale, and MacDougall (2000)

The purposes of this study were to assess maximal isometric strength, voluntary activation, electromyographic (EMG) amplitude, passive stiffness, and twitch characteristics before and after an acute bout of passive stretching (PS_{max}). Ten college-aged subjects (6 males and 4 females) volunteered for this study. The PS_{max} protocol consisted of thirteen 135-s stretches of the plantarflexors for a total of approximately 30 min of time under stretch. A control period (CON) was also used and consisted of no stretching. Isometric strength (maximum voluntary contraction = MVC), EMG amplitude of the soleus, and voluntary activation were assessed immediately post, and at 5, 15, 30, 45, and 60 min after the PS_{max} or CON condition. In a separate trial, twitch properties were examined at 3 different joint angles of dorsiflexion (0°D, 10°D, and 20°D) immediately post, 15, 30, 45, and 60 min after the PS_{max} condition. The results

demonstrated a 28% decrease in MVC which still remained 9% below the pre values 1 hour after PS_{max}. Percent voluntary activation and EMG amplitude were significantly decreased ($P<0.05$) following PS_{max}, however, they returned to baseline by 15 min post-stretching. In the separate trial, passive torque was significantly ($P<0.05$) reduced up to 30 min following PS_{max}, while peak twitch torque remained depressed up to 1 hour and the angle of optimal peak twitch shifted temporarily from 10°D to 20°D immediately following PS_{max}. These findings suggested that prolonged passive stretching (30 min) decreases voluntary isometric strength for up to 1 hour due to decreases in both impaired muscle activation and impaired muscle contractile ability. However, the decreases in muscle activation returned to baseline within 15 min following PS_{max}. The authors (25) hypothesized that the neural changes following PS_{max} may be due to specific neuromuscular feedback responses that include the Golgi tendon organs, mechanoreceptors (type III afferent), nociceptors (type IV afferent), and/or fatigue responses. In contrast, the authors (8) suggested that the mechanical changes after PS_{max} may have been due to alterations in the length-tension relationship and/or connective tissue plastic deformation.

Behm, Button, and Butt (2001)

The purpose of this investigation was to examine the factors underlying the decreases in force production following an acute bout of passive stretching of the leg extensor muscles. Twelve healthy male subjects (age: 20-43 years) volunteered for this experiment. The passive stretching protocol was preceded by a 5-min submaximal warm-up on a cycle ergometer and included 5 sets of 45-s stretches with 15 s of rest

between stretches (20 min of time under stretch). The stretching exercises consisted of a standing quadriceps stretch, hurdler quadriceps stretch, kneeling hip extension stretch, and an assisted prone quadriceps stretch. All stretches (unassisted and assisted) were held at the point of discomfort. Isometric strength testing included 2 tetanic and 3 maximal voluntary contractions (MVCs) with 1 min of rest between contractions. The twitch interpolation technique was performed during the MVCs to determine the extent of muscle inactivation. Surface electromyographic (EMG) electrodes were placed over the rectus femoris and biceps femoris. Assessments were performed (Pre) and 6-10 min after (Post) the stretching and control conditions. For the stretching protocol, MVC was significantly decreased ($P<0.05$) by 12% with a significant ($P<0.05$) increase in muscle inactivation by 2.8%. Surface EMG activity significantly decreased ($P<0.05$) by 20.2% for the quadriceps and non-significantly decreased by 16.8% for the hamstrings. However, passive stretching did not influence peak tetanic force. The authors (8) hypothesized that the lack of tetanic force changes post-stretching suggested that decreases in voluntary force production were more affected by changes in muscle activation rather than changes in muscle elasticity. However, it was also suggested (8) changes in tetanic force following stretching may be seen at other joint angles.

Herda, Ryan, Smith, Walter, Bemben, Stout, and Cramer (2008)

The purpose of this study was to examine the acute effects of prolonged passive stretching (PS) versus prolonged vibration (VIB) on voluntary peak torque (PT), percent voluntary activation (%VA), peak twitch torque (PTT), passive range of motion (PROM), musculotendinous stiffness (MTS), and surface EMG and MMG amplitude of the medial

gastrocnemius (MG) and soleus (SOL) muscles during isometric maximal voluntary contractions (MVCs) of the plantar flexors. Fifteen healthy recreationally-active men volunteered for this investigation. Each participant performed a MVC and a PROM assessment prior to and after 20 min of PS, VIB, and a control condition. The passive stretching protocol consisted of nine 135-s stretches for a total duration of 20 min of time under stretch. The VIB protocol was applied to the Achilles tendon using a Foredom Percussion Hammer (Bethel, CN) at 70 Hz for the entire 20 min. The PS and VIB conditions resulted in significant decreases ($P<0.05$; 5-10%) in voluntary PT, nonsignificant ($P=0.081$; 2-3%) decreases in %VA, and 9-23% decreases ($P<0.05$) in EMG amplitude of the MG and SOL muscles, respectively. There were no changes ($P>0.05$) after the control condition. In addition, PROM increased ($P<0.05$) by 19% and MTS decreased ($P<0.05$) by 38% after the PS condition, however there were no changes ($P>0.05$) in PROM and MTS after the VIB and control conditions. The authors (29) hypothesized that the similar stretching- and vibration-induced decreases in isometric PT and muscle activation may have been due to decreases in Ia-afferent feedback from the muscle spindles (i.e. gamma loop dysfunction). However, the increased PROM and decreased MTS observed in the PS condition suggested that "mechanical factors" may also have contributed to the stretching-induced decreases in muscle strength.

Cramer, Housh, Weir, Johnson, Coburn, and Beck (2005)

The purpose of this study was to examine the acute effects of static stretching on peak torque (PT), the joint angle at PT, mean power output (MP), electromyographic (EMG) amplitude, mechanomyographic (MMG) amplitude of the vastus lateralis (VL)

and rectus femoris (RF) muscles during maximal voluntary isokinetic leg extensions at 60 and 240°·s^{-1} (Cybex 6000 dynaometer, Division of Lumex, Inc., Ronknonkoma, NY) of the stretched and unstretched limbs. Twenty-one subjects (mean age ± SD, 21.5 ± 1.3) volunteered for this study. Immediately following the pre-stretching isokinetic strength testing, each subject underwent four static stretching exercises (1 unassisted and 3 assisted) of the dominant limb only. Each stretching exercise was held for 30 s to the point of mild discomfort with 20 s of rest between each stretch. After stretching, the isokinetic strength tests were repeated. There were significant decreases ($P<0.05$) in PT for the stretched limb at 60 and 240°·s^{-1} and for the unstretched limb at 60°·s^{-1}. EMG amplitude of the VL and RF also decreased ($P<0.05$) from pre- to post-stretching for both the stretched and unstretched limbs. However, there were no stretching-induced changes ($P>0.05$) for the joint angle at PT, MP, or MMG amplitude. Given that PT and EMG amplitude decreased in both limbs, the authors (18) hypothesized that the stretching-induced decreases may have been due to an unidentified central nervous system inhibitory mechanism. Although no changes were seen for the joint angle at PT, it was suggested (18) that the stretching-induced changes in the length-tension relationship may have compensated for the decreases in PT by maintaining the area under the torque vs. range of motion curves.

Evetovich, Nauman, Conley, and Todd (2003)

The purpose of this study was to examine the effects of an acute bout of static stretching of the biceps brachii on peak torque, electromyography (EMG), and mechanomyography (MMG) during maximal concentric isokinetic muscle actions.

Eighteen (men, n = 10, women, n = 8) adult subjects (mean ± SD, age = 22.7 ± 2.8 years) performed 3 maximal isokinetic forearm flexion muscle actions at 30 and 270°·s^{-1} following either a stretching or non-stretching condition. The highest torque value for each velocity was used for analysis. EMG (Quinton Quick Pre Ag-AgCl; Quinton, Bothello, WA) and MMG (Hewlett-Packard, Andover, MA; model #21050, bandwith 0.02-2,000 Hz) sensors were placed over the non-dominant biceps brachii. The stretching protocol consisted of 3 static stretches, which were held for 30 s, repeated 4 times, and with 15 s of rest between stretches. For peak torque, the non-stretching condition resulted in significantly greater ($P<0.05$) torque when compared to the stretching condition at both velocities. MMG amplitude was significantly greater ($P<0.05$) for the stretching condition; however, EMG amplitude was not different ($P>0.05$) between conditions. The authors (23) hypothesized that the stretching-induced decreases in musculotendinous stiffness may have accounted for the decreases in peak torque at both velocities. These findings suggested that for athletic performances that include maximal muscle actions of the upper body, stretching may be detrimental to performance.

Ryan, Beck, Herda, Hull, Hartman, Stout, and Cramer (2008)

The purpose of this study was to extend the findings of Fowles et al. (25) and examine the time course for the acute effects of 2, 4, and 8 min of more practical passive stretching durations on isometric peak torque (PT), percent voluntary activation (%VA), electromyographic (EMG) amplitude, peak twitch torque (PTT), rate of twitch torque

development (RTD), and range of motion (ROM) of the plantar flexors. Thirteen volunteers (mean±SD age=22±3 yrs) participated in 4 randomly-ordered experimental trials: control (CON), 2 min (PS_2), 4 min (PS_4), and 8 min (PS_8) of PS. Isometric strength and passive ROM tests were conducted before (pre), immediately after (post), and at 10, 20, and 30 min post-stretching. The PS trials included varied repetitions of 30-s passive stretches with 20 s of rest between stretches. The passive stretches were repeated until the specific time under stretch for each condition was completed (i.e. PS_2 involved four 30-s stretches for a total of 2 min of time under stretch). The CON condition consisted of a 15 min resting period, equivalent to the total duration of the 8 min PS condition (including rest between stretches). PT, %VA, EMG amplitude, PTT, and RTD were assessed during the twitch interpolation technique during each voluntary isometric strength test, while ROM was quantified as the maximum tolerable angle of passive dorsiflexion. Isometric PT decreased ($P<0.05$) immediately after all conditions (CON (4%), PS_2 (2%), PS_4 (4%), and PS_8 (6%)), but returned to baseline at 10 min. EMG amplitude and %VA were unaltered ($P>0.05$) after all conditions. PTT and RTD decreased ($P<0.05$) immediately after the PS_4 (7%) and PS_8 (6%) conditions only, however returned to baseline at 10 min post-stretching. ROM also increased immediately after stretching for the PS_2 (8%), PS_4 (14%), and PS_8 (13%) conditions, however returned to baseline after 10 min. More practical durations of stretching (2 – 8 min) did not decrease isometric PT compared to the CON, however, the stretching did temporarily improved ROM. In addition, there were dose-dependent decreases in potentiated twitch properties (PTT and RTD) for the PS_4 and PS_8 conditions, which suggested that the mechanical properties of muscle contraction may have been altered, yet these changes

were not sufficient to alter voluntary PT possibly due to the joint angle tested. The authors (63) concluded that the results of this study questioned the overall detrimental influence of PS on performance.

Egan, Cramer, Massey, and Marek (2006)

The purpose of this study was to examine the acute effects of static stretching on peak torque (PT) and mean power output (MP) during maximal, voluntary concentric isokinetic leg extensions at 60 and 300°·s^{-1} in National Collegiate Athletic Association Division I Women's Basketball players. Eleven members of a Division I women's basketball team volunteered for this study. Isokinetic strength testing was completed prior to and after (post) the static stretching exercises at 5, 15, 30, and 45 min post-stretching. The strength testing consisted of 3 or 4 submaximal warm-up trials followed by 3 maximal muscle actions at both randomly-ordered velocities. The repetition with the greatest amount of work was selected for analysis. Following the initial isokinetic strength testing, each subject stretched their dominant leg extensors using 1 unassisted and 3 assisted static stretching exercises. Each stretch was held for 30 s to the point of mild discomfort, which was repeated four times with a 20 s rest between stretches (8 min of time under stretch). The results indicated that static stretching had no influence on PT (P=0.161) or MP (P=0.088) from pre- to post-stretching for any of the post-stretching time intervals. The authors suggested (22) that trained athletes may be less susceptible to the stretching-induced force deficit than untrained, non-athletes.

The acute effects of stretching on voluntary neuromuscular function and the length-tension relationship.

Cramer, Beck, Housh, Massey, Marek, Danglemeier, Purkayastha, Culbertson, Fitz, and Egan (2007)

The purpose of this study was to examine the acute effects of static stretching on peak torque (PT), work, the joint angle at peak torque, acceleration time, isokinetic range of motion, mechanomyographic (MMG) amplitude, and electromyographic (EMG) amplitude of the rectus femoris during maximal concentric isokinetic leg extensions. Ten women (mean ±SD: age=23±2.9 yrs) and eight men (age 21.4=21.4±3.0 yrs) volunteered for this study. Isokinetic strength testing was performed before and after the static stretching and consisted of maximal concentric muscle actions of the dominant leg extensors using a calibrated Biodex System 3 dynamometer (Biodex Medical Systems, Inc., Shirley, NY) at randomly ordered velocities of 1.04 and 5.23 $rad \cdot s^{-1}$. The repetition resulting in the greatest amount of work was selected for analysis. The static stretching protocol consisted of 4 repetitions of 1 unassisted and 3 assisted stretching exercises held for 30 s to the point of mild discomfort. Between stretches the leg was returned to a neutral position for a 20-s rest period. EMG bipolar surface electrodes (Moore Medical, Ag-AgCl) and MMG accelerometers (EGAS-FS, Entran, Inc., Fairfield, NJ) were placed along the longitudinal axis of the rectus femoris. PT, acceleration time, and EMG amplitude decreased ($P<0.05$) from pre- to post-stretching at 1.04 and 5.23 $rad \cdot s^{-1}$. However, there were no changes ($P>0.05$) in work, joint angle at peak torque, isokinetic range of motion, or MMG amplitude. These findings indicated that static stretching did

not change the area under the angle – torque curve (work), but caused significant decreases in PT. The authors (4) suggested that the stretched caused a "flattening" of the angle-torque curve such that decreases PT occurred, but greater force production also occurred at the extreme ends of the angle-torque curve. The authors (16) hypothesized that the flattening of the angle-torque curve and increased limb acceleration rates provided indirect support of the hypothesis that static stretching alters the angle-torque relationship and/or sarcomere shortening velocity.

Herda, Cramer, Ryan, McHugh, and Stout (2008)

The purpose of this study was to examine the acute effects of static versus dynamic stretching on peak torque (PT), electromyographic (EMG), and mechanomyographic (MMG) amplitude of the biceps femoris (BF) muscle during isometric maximum voluntary contractions (MVCs) of the leg flexors at four different knee joint angles. Fourteen men (mean ±SD: age=25±4 yrs) volunteered for this study. Strength testing was performed prior to and immediately after the dynamic and static stretching routines of the right leg flexors. Isometric PT was measured at 4 randomly-ordered knee joint angles of 41°, 61°, 81°, and 101° below full extension on a Biodex System 3 isokinetic dynamometer (Biodex Medical Systems, Inc., Shirley, NY). Two 4 s isometric MVCs were performed at each joint angle with 30 s of rest between each MVC and joint angle. The higher of the two MVC trials was used for the analysis. The static stretching routine included 4 repetitions of 1 unassisted and 2 assisted stretches held for 30 s at the point of mild discomfort with 15 s of rest between stretches. The dynamic stretching routine included 4 sets of three exercises designed to stretch the same muscles

as the static stretching routine. Each set was completed in a slow and controlled manner for 30 s which resulted in 12-15 repetitions per set with a 15 s rest period between sets. The repetitions, sets, and rest periods were chosen in order to equalize the volume of static and dynamic stretching (mean±SD: 9.2±0.4 min for the static stretching and 9.1±0.3 min for the dynamic stretching). Bipolar surface EMG electrodes (Ag-Ag Cl, Quinton Quick Prep, Quinton Instruments Co., Bothell, WA) and MMG accelerometers (EGAS-FS-10-/VO5, Measurement Specialties, Inc., Hampton, VA) were placed along the longitudinal axis of the BF muscle. PT decreased from pre- to post-static stretching at 81° ($P=0.019$) and 101° ($P=0.001$), but resulted in no strength changes at the other joint angles. PT was unaltered ($P>0.05$) from pre- to post-dynamic stretching. EMG amplitude was unchanged after static stretching ($P>0.05$), however, it increased following dynamic stretching at 81° ($P<0.001$) and 101° ($P<0.001$). MMG amplitude increased following static stretching at 101° ($P=0.003$), while dynamic stretching increased MMG amplitude at all joint angles ($P<0.05$). These findings suggest that decreases in strength following static stretching may be due to mechanical rather than neural mechanisms for the BF muscle, which are evident at the shortest muscle lengths (81° and 101°). The authors (28) concluded that an acute bout of dynamic stretching appears to be less detrimental to maximal force production and may even cause post-activation potentiation due to the increases observed in EMG and MMG amplitude of the leg flexors.

McHugh and Nesse (2008)

The purposes of this study were to determine if the stretch-induced force deficit is muscle length dependent (study 1) and determine if performing passive stretching prior to

eccentric exercise affects strength loss and pain on subsequent days (study 2). Ten men (mean ±SD: age=33±9 yrs) volunteered for study 1. Strength testing of the leg flexors was performed isometrically at six joint angles (80°, 65°, 50°, 35°, 20°, and 5° below full extension) and isokinetically (concentric and eccentric) on a Biodex System 2 dynamometer. The subjects were tested with the thigh flexed at 20° and the trunk flexed to 90° creating a trunk-to-thigh angle of 70°. Two isometric maximal voluntary contractions (MVCs) were performed at each joint angle with 1 min between test angles. Following the isometric contractions, each participant performed 4 maximal isokinetic eccentric and concentric muscle actions at $60°\cdot s^{-1}$. The stretching protocol consisted of six 90-s static hamstring stretches (9 min of time under stretch) where the leg was passively stretched to full extension at $5°\cdot s^{-1}$. Passive torque at full extension was recorded at the start of the first and sixth stretch. For study 2, the dominant and nondominant legs of 8 men (age=34±9 yrs) were randomly assigned to either a stretch (six 60 s stretches) or control condition prior to eccentric hamstring exercise. Isometric strength at five joint angles (89°, 76°, 63°, 50°, and 37°) and pain were assessed prior to, immediately after, and 3 days after 10 unilateral isokinetic eccentric hamstring at 100% of an isometric MVC. The control period included no stretching and the contralateral limb performed the eccentric exercise 8 min after the isometric contractions. For study 1, following 9 min of stretching, isometric strength was decreased by 17% at 80°, 11% at 65°, 5% at 50°, 7% at 35°, 8% at 20°, and increased by 6% at 5° ($P<0.01$). In addition, there was an 8.3% decrease in the resistance to stretch from the first to the sixth stretch ($P<0.05$). Isokinetic eccentric and concentric peak torque and the angle at peak torque were unaffected by the static stretching protocol ($P>0.05$). For study 2, isometric

strength following eccentric exercise was greater for the stretched versus the unstretched control limb at 37° ($P<0.05$), but was unaffected at the other joint angles ($P>0.05$). There was also no difference ($P=0.94$) in reported pain between the stretched and unstretched limb. These results suggested that the stretching-induced force deficit was dependent on muscle length, with decreases in isometric strength occurring at the shortest muscle lengths, however, there was no change in strength in the lengthened position. Stretching prior to eccentric exercise offered little prevention of strength loss or perceived pain, however, stretching did prevent strength loss with the muscle in its most lengthened position. The authors (49) concluded that the stretch-induced preservation of strength with the muscle in a lengthened position following eccentric exercise may be important for resisting injuring during training when muscle damage is present such as during preseason training.

Nelson, Allen, Cornwell, and Kokkonen (2001)

The purpose of this study was to determine if the decreases in isometric strength following an acute bout of static stretching are joint angle-specific. Thirty female and 25 male college students (mean ±SD: age=22±6 yrs) volunteered for this study. Each participant performed maximal isometric leg extensions of the dominant leg on a Cybex II isokinetic dynamometer (Lumex, Inc., Bay Shore, NY) at five different knee joint angles (90°, 108°, 126°, 144°, and 162°) prior to (pre) and following (post) the static stretching protocol . Each participant performed four maximal voluntary contractions (MVCs) at each randomly-ordered joint angle. The average peak torque of the four contractions was used for subsequent analyses. The static stretching protocol consisted

of two stretching exercises of the dominant leg extensors. Each stretch was repeated four times and held for 30 s with 20 s of rest between stretches (4 min of time under stretch). Following the static stretching protocol, average peak torque was reduced ($P<0.01$), but only at a knee joint angle of 162°, whereas the average peak torque was unchanged ($P>0.05$) following the static stretching protocol for the remaining joint angles. These findings suggested that an acute bout of static stretching reduces isometric strength at the short muscle lengths. The authors (54) hypothesized that the stretching protocol inhibited strength by placing sarcomeres at a less-than-optimal length much sooner in the full range of motion.

The effects of regular chronic stretch training on the stretching-induced force deficit.

Behm, Bradbury, Haynes, Hodder, Leonard, and Paddock (2006)

The purpose of this study was to determine if initial flexibility (study 1) and gains in range of motion following a chronic stretching program (study 2) would diminish the stretch-induced force deficit. Eighteen subjects (mean ±SD: age=25±8 yrs) volunteered for study 1. Each subject performed isometric maximum voluntary contractions (MVCs) of the leg extensors and a drop jump prior to (pre) and after (post) an acute bout of static stretching. In addition, hip flexion, hip extension, and plantar dorsiflexion range of motion (ROM) measurements were taken at pre- and post-stretching. Twelve subjects (22±2 yrs) volunteered for study 2. Each subject performed isometric leg extension and flexion MVCs, a drop and countermovement jump, and ROM assessments prior to and

following an acute bout of stretching, which was then repeated after four weeks of flexibility training. The ROM assessments for plantar dorsiflexion were replaced in the training study by the sit and reach test. The acute stretching protocol included four separate stretches for the quadriceps, hamstrings, and plantar flexor (leg extended and flexed) that were repeated 3 times, held for a duration of 30 s, and with 30 s of rest between stretches. The stretch training program included the same stretches that were performed in study 1, and were completed five days per week for four weeks. The results indicated that an acute bout of static stretching reduced leg extension (6.1-8.2%, $P<0.05$) and flexion (6.6-10.7%, $P<0.05$) strength, drop jump contact time (5.4-7.4%, $P<0.01$), and countermovement jump height (5.5-5.7%, $P<0.01$). The findings from study 1 showed no significant relationship between ROM and the stretching-induced force deficits ($P>0.05$). Stretch training increased sit and reach (11.8%, $P<0.01$), hip extension (19.7%, $P<0.01$), and hip flexion (13.4%, $P<0.01$) ROM. However, stretch training had no significant ($P>0.05$) effect on the stretching-induced force deficit. The authors (7) suggested that since all stretches were held to the point of discomfort with all testing, the relative stress on the muscle was unaffected by training, initial flexibility, or stretch tolerance.

Nelson, Kokkonen, and Eldredge (2005)

The purpose of this study was to determine if chronic stretch training would attenuate the maximal strength losses seen following an acute bout of static stretching. Thirty-one college students (18 women and 13 men) enrolled in physical activity classes volunteered for this study. Each participant had engaged in at least 30 min of supervised

daily stretching for at least 10 weeks and had a percentile rank greater than 60 for sit-and-reach norms prior to the start of the study. The stretch training routine consisted of 15 different static stretches which were designed to target all of the major lower extremity muscles. The strength testing protocol consisted of a 1 repetition max (1RM) for the leg flexors and leg extensors on two successive days. The 1 RM protocol included repeated maximal attempts with set (i.e. 10 lb) increases in weight until each participant failed to complete a lift. Prior to each treatment, each participant randomly completed either static stretching of the hip, thigh, and calf muscles for 20 min or 10 min of quiet sitting. In addition, a sit-and-reach test was performed before and after each treatment using an Acuflex I sit-and-reach flexibility test (Novel Products, Rockton, IL). The results indicated that sit-and-reach performance increased from pre- to post-stretching; however sit-and-reach performance did not improve following 10 min of quiet sitting. Following static stretching, leg flexion 1 RM and leg extension 1 RM were significantly ($P<0.05$) less than the non-stretching condition (3.6 and 5.7%, respectively). The authors (56) concluded that acute static stretching can inhibit maximal strength of the leg flexors and extensors regardless of previous stretching history.

The effects of regular chronic stretch training on musculotendinous stiffness and range of motion.

Magnusson, Simonsen, Aagaard, Sorensen, and Kjaer (1996)

The purpose of this study was to examine the effects of a long-term stretching regimen on the tissue properties and stretch tolerance of human skeletal muscle. Seven female participants (mean ±SD: age=26±6 yrs) volunteered for this study. Each participant completed two protocols before and after three weeks of stretch training. The first protocol consisted of a slow stretch (0.087 $rad \cdot s^{-1}$) to a pre-determined angle, which was held for 90 s for both the stretched and contralateral control leg. At the end of the holding phase, each participant performed a maximal voluntary contraction (MVC) to normalize the passive peak torque values and electromyographic activity (EMG) data. Stiffness ($Nm \cdot rad^{-1}$), passive energy (area under the curve), and EMG amplitude (μV) were calculated during the slow stretch maneuver. During the holding phase, initial peak torque, rate of torque decline, and EMG amplitude were calculated. Protocol 2 was similar to protocol 1, however the stretch was continued to the point of pain (stretch tolerance). The stretch training consisted of two daily sessions, one in the morning and one in the afternoon, for 20 consecutive days. Each session consisted of five 45-s stretches with 15–30 s of rest between stretches. The total stretching duration was 150 min with each participant completing on average 94% of the training (141 min). The results for protocol 1 indicated no significant differences in stiffness (P=0.86), passive energy (P=0.61), and EMG amplitude (P=0.24) following 3 weeks of stretch training when compared to the control leg. However, for protocol 2, there were significant increases in maximal joint angle (P=0.018), peak toque (P=0.018), and passive energy (P=0.018) for the stretched leg only. EMG amplitude was also unchanged for both the control and stretched leg following training. These results suggested (46) that reflex surface EMG activity may not limit range of motion during slow controlled stretches and

increases in range of motion following three weeks of stretch training may not be due to increases in stretch tolerance, rather than a change in the mechanical or viscoelastic properties of the muscle.

Guissard and Duchateau (2004)

The purpose of this study was to determine the contributions of neural and mechanical mechanisms that influence range of motion (ROM) about a joint and how these are influenced by chronic stretch training. Twelve participants (8 men and 4 women) volunteered for this study. The electrical and mechanical properties of the right plantar flexor muscles were measured in the prone position before, after 10, 20, and 30 training sessions, as well as 30 days after the end of training. Each session included a progressive stimulation of the tibial nerve to determine the Hoffmann (Hmax) reflex and the maximal direct motor response (Mmax). The tendon (Tmax) reflex was determined from a clinical reflex hammer dropped onto the Achilles tendon from a constant height. Each participant then performed three 5-s isometric maximal voluntary contractions (MVCs), 5 ballistic submaximal (70% of MVC) isometric contractions, and a maximal dorsiflexion range of motion assessment to determine flexibility and passive stiffness of the angle-torque curve. Maximal peak torque and rate of torque development were determined during the maximal isometric MVCs and submaximal ballistic MVCs, respectively. The stretch training program consisted of 30 total sessions performed five days per week for six weeks. Four different static stretches were repeated five times, held for 30 s, with 30 s of rest between each stretch. Each stretch was held to the point of discomfort for a total of 10 min of time under stretch. The stretching training resulted in

a 30.8% increase ($P<0.01$) in maximal ankle dorsiflexion, with 56% of this gain observed after only 10 sessions. Thirty days following the training, 74% of the increases in flexibility were still present. Passive stiffness decreased by 33% ($P<0.001$) after 30 training sessions and remained decreased ($P<0.001$) 30 days after training. There were no changes ($P> 0.05$) in peak isometric MVC torque or rate of torque development across all time periods. Mmax was not modified by training at all time periods ($P>0.05$), although the Hmax/Mmax ratio decreased ($P<0.01$) after 30 training sessions and the Tmax/Mmax ratio decreased ($P<0.05$) after 20 and 30 sessions. However, these changes were transient and returned to baseline 30 days after training. The results from this study suggest that 30 sessions of static stretching of the plantar flexor muscles increase dorsiflexion range of motion and these improvements are partially maintained up to one month after training. The improved flexibility was associated with decreases in passive stiffness and reduced Hmax and Tmax amplitudes, however only changes in passive stiffness were maintained following training. The authors (27) suggested that the increases in flexibility resulted from reductions in both passive stiffness and tonic reflex activity, however, these adaptations may show different time courses.

Mahieu, McNair, Muynck, Stevens, Blanckaert, Smits, and Witvrouw (2007)

The purpose of this study was to determine if static and ballistic stretching programs have different effects on passive resistive torque and tendon stiffness. Eighty-one healthy participants volunteered for this study. All participants were randomly assigned to either a static stretching group (n=31), ballistic stretching group (n=21), or a

control group (n=29). Before and after 6 weeks of stretching or normal daily activity, each participant was evaluated for ankle range of motion (ROM), passive resistive torque of the plantar flexors, and tendon stiffness of the Achilles tendon. Dorsiflexion ROM was measured with a universal goniometer by the same investigator. Passive resistive torque of the plantar flexors was quantified with a Biodex System 3 isokinetic dynamometer that moved the ankle through four continuous cycles from 20° plantar flexion to 10° dorsiflexion at $5°\cdot s^{-1}$. The peak passive resistance torque (Nm) during the four cycles was analyzed. Achilles tendon stiffness was calculated from the ratio of muscle force and the elongation of the Achilles tendon. In addition, each subject performed three 5-s isometric maximal voluntary contractions (MVCs), where the highest peak torque was used to calculate muscle force in the tendon stiffness calculation. The static and ballistic stretching programs consisted of a single daily stretching exercise for six weeks. The static stretching group repeated the stretching exercise five times for 20 s with 20 s of rest between stretches. The ballistic stretching group completed an identical stretching protocol, however, when the subjects reached the initial stretching position, they moved up and down at a pace of one movement per second. The results indicated that dorsiflexion ROM increased ($P<0.01$) for all groups. The static stretching group resulted in significant decreases ($P<0.01$) in passive resistive torque, with no change in Achilles tendon stiffness ($P=0.231$). The ballistic stretching group resulted in no changes in passive resistive torque ($P=0.609$), however, there were decreases in Achilles tendon stiffness ($P<0.01$). There were no changes ($P>0.05$) in passive resistive torque or Achilles tendon stiffness for the control group. These findings demonstrated that static and ballistic stretching techniques have different effects on passive resistive torque and

Achilles tendon stiffness. The authors (47) also suggested that both types of stretching should be considered for training and rehabilitation programs due to their unique adaptations to the muscle-tendon unit.

The effects of regular chronic stretch training on muscular performance.

Hortobagyi, Faludi, Tihanyi, and Merkely (1985)

The purpose of this study was to examine the effect of a lower body stretch training program on muscular performance and range of motion (ROM). Twelve healthy male secondary school students (mean ±SD: age=15±0.5 yrs) volunteered for this study. Each participant performed three isometric maximal voluntary contractions (MVCs), six to eight fast isometric contractions, and five maximal concentric muscle actions at 25, 50, 75, 100, and 125 kg of the leg extensors on two occasions separated by seven weeks. Rate of torque development and half-relaxation time were calculated during the fast isometric contractions. In addition, maximal stride frequency was determined during a sprinting test. Flexibility tests included a front-to-rear split, supine hamstring stretch, and a side split while supine. The stretch training program was performed three times a week for seven weeks. Each participant completed six stretches that were held for 10 s and were repeated two times. The results indicated increases ($P<0.05$) in flexibility from pre- to post-stretch training for all flexibility tests. There were no changes in isometric MVC peak torque. However, rate of torque development, half relaxation time, and maximal stride frequency improved ($P<0.01$) following training. Peak concentric velocity was

also increased ($P<0.01$) for only the three lowest loads (25, 50, and 75 kg). These results suggested that seven weeks of stretch-training increased flexibility and improved the velocity-specific characteristics of isometric and concentric muscle actions, as well as sprinting stride frequency. The authors (33) suggested that these adaptations may be due to stretch-induced increases of sarcomeres in series.

Wilson, Elliott, and Wood (1992)

The purpose of this study was to examine the effects of flexibility training of the upper body musculature on bench press performance. Sixteen experienced male powerlifters volunteered for this study and were randomly assigned to a control (n=8) and experimental group (n=8). Each participant was tested on two occasions separated by eight weeks. Each participant performed a maximal rebound bench press, concentric-only bench press, musculotendinous stiffness measurement, and a measure of upper body (gleno-humeral joint) static flexibility. Musculotendinous stiffness was measured using a damping oscillation technique that required each subject to hold a standard Olympic bar 3 cm above their chest. An external force or perturbation at 15, 30, 45, 60, and 70% of their maximal bench press was then applied to the bar and the subsequent force response was recorded and used to determine musculotendinous stiffness. Each lift was also recorded with a high speed camera to determine velocity, instantaneous power, and the amount of work done. The flexibility training program consisted of four exercises held for 8-20 s. Two sets of six to nine repetitions were performed for each exercise twice a week for eight weeks. The results indicated that gleno-humeral joint flexibility increased ($P<0.01$) and musculotendinous stiffness was reduced ($P<0.05$) from pre- to post-

flexibility training for the experimental group only. Musculotendinous stiffness decreased ($P<0.05$) from pre- to post-flexibility training in both the 70% and maximal load conditions. Maximal rebound bench press (5.4%, $P<0.05$) and concentric-only bench press (4.5%, $P=0.10$) increased following training for the experimental group. In addition, the flexibility-training group increased (20%, $P<0.05$) the amount of work produced in the initial 0.37-s of the concentric phase of the rebound bench press lift. There were no changes ($P>0.05$) for the control condition from pre- to post-testing. The increase in rebound bench press performance following eight weeks of flexibility training was attributed to a reduction in musculotendinous stiffness. The authors (77) suggested that this reduction may have allowed subjects to store and release more elastic strain energy, which consequently may have improved the initial concentric performance.

Worrell, Smith, and Winegardner (1994)

The purposes of this study were to determine the most effective stretching technique to improve flexibility and to examine the effects of improving flexibility on isokinetic peak torque. Nineteen healthy participants [males (n=10) and females (n=9)] volunteered for this study. Each participant performed a flexibility and isokinetic strength testing assessment prior to and after three weeks of stretch training. Hamstring flexibility was assessed with an active leg extension test. Maximal isokinetic eccentric and concentric muscle actions were performed on a Biodex isokinetic dynamometer (Biodex, Shirley, New York, NY). Eccentric and concentric peak torque values were recorded at 60 and 120°·s^{-1} for each leg. Each participant stretched both legs; one leg was randomly assigned a static stretching method and the other leg was stretched using a

contract-relax proprioceptive neuromuscular facilitation (PNF) method. The static stretching method included four repetitions of 15-20 s stretches with 15 s of rest between stretches. The PNF method included four repetitions of 20 s bouts. Each repetition consisted of a 5-s maximal isometric hamstring contraction, 5 s of rest, a 5-s maximal isometric quadriceps contraction, and a 5-s rest. The stretch training program was performed five days per week for three weeks (15 sessions). The results indicated non-significant increases (P=0.082) in flexibility for the static (8°) and PNF (9.5°) stretching methods. Maximal isokinetic eccentric peak torque increased ($P<0.05$) at 60 and $120°\cdot s^{-1}$, while maximal isokinetic concentric peak torque increased ($P<0.01$) at $120°\cdot s^{-1}$. These results indicated that three weeks of chronic stretch training of the hamstrings did not significantly improve hamstring flexibility, however, stretch training did significantly improve maximal isokinetic eccentric and concentric peak torque. The authors (79) suggested that the improvements in hamstring performance may be due to a more compliant series elastic component allowing for greater stored potential energy.

Nelson, Kokkonen, Eldredge, Cornwell, and Glickman-Weiss (2001)

The purpose of this study was to examine the influence of chronic stretch training on submaximal running economy. Thirty-two (16 females, 16 males) participants volunteered for this study. Each participant performed a VO_{2max} graded exercise test, a running economy test, a sit-and-reach test on two occasions separated by 10 weeks. Each participant's VO_{2max} was determined at the initial visit as the highest 20-s average obtained during the last 4 min of each test. On the subsequent day, each participant performed a 10-min running economy test at 70% of his/her VO_{2max}. Running economy

was quantified as the average of all the VO_2 readings during the last 5 min of the submaximal test. Prior to performing the running economy test, each participant performed a sit-and-reach test (Acuflex I, Novel Products, Inc., Rockton, IL). Following all pre-testing measures, all participants were randomly assigned to either a stretching (STR) or non-stretching (CON) group. The stretch training program consisted of 15 assisted and unassisted different static stretches targeting all the lower body musculature. Each stretch was held for 15 s and repeated three times with a 15-s rest period between stretches. Each session lasted about 40 min and was completed three days per week for 10 weeks. The results indicated a significant ($P<0.05$) increase (9%) in sit-and-reach performance for the STR group with no change ($P>0.05$) in the CON group. There were no significant ($P>0.05$) differences from pre- to post-stretch training for VO_{2max} or running economy for both the STR and CON groups. These findings suggest that 10 weeks of chronic stretch training will improve flexibility, but will not influence submaximal running economy. The authors (57) also suggest that the lack of changes in running economy may be due to the fact that chronic stretching does not alter musculotendinous stiffness.

Kokkonen, Nelson, Eldredge, and Winchester (2007)

The purpose of this study was to determine the influence of chronic stretch training on muscle strength, endurance, and power. Thirty-eight participants volunteered for this study. Each participant performed a battery of exercise tests over a 3-day period on two occasions separated by 10 weeks. Day 1 included a sit and reach test, standing long jump, 20-m sprint, and leg extension and leg flexion one repetition maximum (1

RM) tests. Day 2 included a vertical jump test and a leg extension and leg flexion muscle strength-endurance test. Day 3 included a graded treadmill VO_{2max} test. Following all pre-testing measures all participants were randomly assigned to either a stretching (STR, n=11 females, n=8 males) or control (CON, n=11 females, n=8 males) group. The stretching program consisted of 15 assisted and unassisted static stretches targeting all the lower-extremity musculature. Each stretch was held for 15 s, repeated three times with a 15-s rest period between stretches. Each session lasted 40 min and was performed three days per week for 10 weeks. For flexibility, the results indicated an 18% increase ($P<0.001$) in sit and reach performance for the STR group with no change ($P>0.05$) in the CON group. For muscular power, the STR group improved standing long jump distance (2.3%, $P<0.001$), vertical jump height (6.7%, $P<0.01$), and 20-m sprint time (1.3%, $P<0.001$) when compared to the CON group. For muscular strength and endurance, the STR group improved leg extension (32.4%, $P<0.001$) and leg flexion (15.3%, $P<0.001$) maximal strength and leg extension (28.5%, $P<0.001$) and leg flexion (30.4%, $P<0.001$) strength-endurance when compared to the CON group. These findings suggested that a structured, chronic stretch training program may improve muscular flexibility, strength, and power. The authors (41) also suggested that the increases in strength and power may have been attributed to stretch-induced muscle hypertrophy.

Bazett-Jones, Gibson, and McBride (2008)

The purpose of this study was to examine the influence of chronic stretch training on hamstring flexibility, vertical jump height, and sprint performance in trained athletes. Twenty-one division III women's track and field athletes (mean±SD, age ± 18.57)

volunteered for this study. Each participant performed an active leg extension test, a 55-m sprint, and a vertical jump test on three occasions separated by three weeks. Maximal 55-m sprint speed was determined using wireless timing gates (Brower Timing Systems, Draper, UT) and vertical jump height was determined using a jump force platform (Kistler Instrument Corp., Buffalo, NY). Following all pre-testing measures, all participants were randomly assigned to a stretching (n=10) or control (n=11) group. The stretch training consisted of one static hamstring stretch that was performed four days per week for six weeks. The static stretch was repeated four times and held for 45-s with a 45-60-s rest between stretches for both legs. No significant changes ($P>0.05$) were observed for left and right leg flexibility, vertical jump height, or 55-m sprint speed for either the stretching or control group. However, there were non-significant increases ($P>0.05$) in active leg extension range of motion for the left (1.7%) and right (2.1%) leg. These findings suggest that six weeks of chronic stretch training has little influence on performance in trained track and field athletes. The authors (4) also added that competitive athletes with normal flexibility may not benefit from regular chronic stretch training.

LaRoche, Lussier, and Roy (2008)

The purpose of this study was to examine the effects of chronic stretch training on thigh extension peak torque (PT), rate of torque development (RTD), work (W), and the angle at peak torque (PTA). Twenty-nine males (age, 18-60 years) volunteered for this study. All participants were randomly assigned to a control (n=10), static (n=9), or ballistic (n=10) stretching group. Each participant performed four maximal thigh

extensions at $60°\cdot s^{-1}$ on a custom thigh extensor torque apparatus that was connected to a Cybex II isokinetic dynamometer on two occasions separated by four weeks. PT was quantified as the highest torque value generated, RTD was the slope of the linear region of the torque vs. time curve, W was calculated by integrating the area under the angle-torque curve, and PTA was the hip angle at which PT occurred. The stretching program consisted of 10 sets of a single hamstring stretching exercise, which was performed three times per week for four weeks. Each stretch was held for 30 s with 30 s of rest between stretches. The static stretching group held the position for the duration of the stretch, whereas the ballistic stretching group moved in and out of the stretch each second. The results indicated no significant ($P>0.05$) differences between the static and ballistic stretching groups when compared to the control group for PT, RTD, W, or PTA. These findings suggested that a short (four weeks) chronic stretch training program has little influence on hamstring strength. In addition, the authors (45) suggested that the lack of changes in PTA may have indicated that muscle length or the length-tension relationship was unaltered.

CHAPTER III

METHODS

Participants

Twenty-six healthy men (mean±SD age = 21 ± 2 yrs; stature = 175 ± 8 cm; mass = 75 ± 11) volunteered for this investigation. None of the participants reported any current or ongoing neuromuscular diseases or musculoskeletal injuries specific to the ankle, knee, or hip joints. All participants reported engaging in 1 ± 1 $hr \cdot wk^{-1}$ of aerobic exercise, 2 ± 2 $hr \cdot wk^{-1}$ of resistance exercise, and 2 ± 2 $hr \cdot wk^{-1}$ of recreational sports. None of the participants were competitive athletes, however, due to their reported levels of aerobic exercise, resistance training, and recreational sports (above), these individuals might be best classified as normal, moderately-active, recreationally-trained participants. This study was approved by the University of Oklahoma Institutional Review Boards for Human Subjects, and all participants completed a written informed consent form (Appendix A) and a Pre-Exercise Testing Health & Exercise Status Questionnaire (Appendix B). Using the procedures described by Howell (34) for estimating sample sizes for repeated measures designs, a minimum sample size of n = 9 was required for each group to reach statistical power (1-β) of 0.80 based on the findings of Fowles et al. (25).

Research Design

A randomized, repeated measures design [acute (pre- vs. post-stretching) × chronic (week 1 vs. week 5) × condition (stretching vs. control) × angle (-19° vs. -9° vs.

1° vs. 12° dorsiflexion); 2 × 2 × 2 × 4] was used to examine the acute and chronic effects of repeated passive stretching (PS) on muscle force production at four different ankle joint angles during voluntary and non-voluntary (evoked) contractions, percent voluntary activation (%VA), surface electromyographic (EMG) amplitude of the soleus (SOL) and medial gastrocnemius (MG) muscles, dorsiflexion range of motion (ROM), musculotendinous stiffness (MTS) of the plantar flexors, and corrected calf girth. Each participant visited the laboratory three times to undergo isometric strength and flexibility assessments of the right plantar flexors. The first visit (week 0) was a familiarization trial, and the next two visits (weeks 1 and 5) were identical acute stretching protocols separated by four weeks (Table 1). The acute stretching protocols involved pre-stretching isometric strength and dynamic flexibility assessments, 20 min of passive stretching (PS), and post-stretching isometric strength and flexibility assessments. Following the first visit (week 0), each participant was randomly assigned to either a control (CON) or stretching (STR) group for the remaining four weeks of the study (Table 1). After the four-week stretching or control intervention, visit three (week 5) was performed using the same protocol as visit two (Figure 1). All laboratory testing (weeks 0, 1, and 5) was performed at the same time of day (± 2 h) for each subject.

Variables

The independent variables included: (a) acute [pre- vs. post-stretching], (b) chronic [week 1 vs. week 5], (c) treatment [CON vs. STR], (d) ankle joint angle for voluntary strength and evoked twitches [-19° vs. -9° vs. 1° vs. 12° dorsiflexion], and (e) ankle joint angle for the MTS assessments [1° vs. 5° vs. 9° vs. 13°]. The dependent

variables that were measured included: (a) isometric maximal voluntary contraction (MVC) force, (b) evoked twitch forces after single and titanic stimuli, (c) percent voluntary activation (%VA), (d) surface electromyographic (EMG) amplitude for the MG and SOL muscles, (e) dorsiflexion range of motion (ROM), (f) MTS, and (g) corrected calf girth.

Instrumentation

- A custom-built, modified McComas boot connected to Biodex Systems 3 isokinetic dynamometer (Biodex Medical Systems; Shirley, NY) was used to passively dorsiflex the foot for the ROM and MTS measurements.
- A high-accuracy low-profile load cell (Omegadyne LC402, Stamford, CT) was used to measure MVC and evoked twitch plantar flexion force.
- A Digitimer DS7AH (Herthfordshire, UK) was used to evoke the twitches by stimulating the tibial nerve.
- Pre-amplified (gain: x 350) active EMG electrodes (TSD150B, Biopac Systems, Inc.; Santa Barbara, CA) with a 20-mm inter-electrode distance was placed over the SOL and MG muscles to record surface EMG signals.
- The pre-amplified EMG signals were sampled at 2 KHz and filtered with a commercially-available acquisition unit (MP150WSW, Biopac Systems, Inc.; Santa Barbara, CA) with an analog bandpass filter of 12 – 500 Hz.
- All analog signals from the Biopac acquisition system were sampled with an external analog-to-digital converter (DAQCard 6036E, National Instruments,

Austin, TX) that was controlled by custom-written software (LabVIEW 8.5, National Instruments, Austin, TX).

- A personal computer (Dell Inspiron 8200, Dell, Inc., Round Rock, TX) was used to store all the digitized signals for off-line analysis.
- Custom-written software (LabVIEW 8.5, National Instruments, Austin, TX) was used to process the surface EMG, force, torque, and dynamometer position signals.
- A standard Gulick anthropometric measuring tape (AliMed, Dedham, MA) with a spring attachment was used to measure calf circumferences.
- A calibrated (± 1 mm) Lange Skinfold Caliper (Santa Cruz, CA) was used to obtain a medial calf skinfold.

Familiarization Trial

During week 0 (Figure 1), each participant visited the laboratory to practice the strength and flexibility assessments and become familiarized with the procedures, including the evoked twitches. During the familiarization trial, each participant identified the maximum amount of passive torque that they could tolerate during a stretch (i.e., the point of discomfort, but not pain as acknowledged by the subject). This amount of passive torque was then used to stretch the plantar flexors during all subsequent testing and training protocols.

In addition, each subject's corrected calf girth was measured during the familiarization trial using a standard Gulick anthropometric tape (AliMed, Dedham, MA) and Lange skinfold caliper (Santa Cruz, CA) (70). With the subject seated, calf

circumference was recorded horizontally at the maximum girth of the calf muscle (70). A medial calf skinfold was taken at the same location with a vertical fold (70). Corrected calf girth was then calculated by the following formula as described by Stewart et al. (70):

$$\text{Corrected Calf Girth} = \text{Calf Circumference} - (\text{Skinfold Thickness} \times \pi)$$

Muscle Force Assessments

To determine MVC force, twitch force, and %VA, each participant was seated with restraining straps over the pelvis, trunk, and thigh, with a leg flexion angle of 0º below the horizontal plane (full extension) on a custom-built, modified McComas boot (48) connected to a calibrated Biodex System 3 dynamometer (Biodex Medical Systems, Inc. Shirley, NY, 1998, Figure 2A). The lateral malleolus of the fibula was aligned with the input axis of the dynamometer. The foot was secured in a thick rubber heel cup attached to a footplate with straps over the toes and metatarsals (distal to the malleoli) so that the straps would not impede any passive foot movement at the ankle joint (Figure 2B).

Each participant performed one isometric MVC, one twitch evoked with a single stimuli, and one twitch evoked with a tetanic train of stimuli at four randomly-ordered joint angles, including -19°, -9°, 1°, and 12° of dorsiflexion (where 0° = neutral ankle joint angle = 90° between the foot and leg; negative angles describe the foot in plantar flexion; positive angles describe the foot in dorsiflexion). At each joint angle, the single-stimulus twitch and tetanic twitch were performed approximately 15-20 s apart (25) in

random order prior to the isometric MVC and twitch interpolation technique. One minute of rest was allowed between joint angles (8).

The single-stimulus and tetanic twitches were evoked with the same stimulus intensity as determined for the twitch interpolation technique (see below for the procedures to determine the stimulus intensity). The tetanic train was elicited at a frequency of 100 Hz for 500 ms. The MVC trials were used to assess peak voluntary force (before the superimposed twitch) as well as %VA (see below). For the isometric MVC's, the participants were instructed to give a maximum effort for each trial and strong verbal encouragement was provided by the investigators.

Percent Voluntary Activation

The twitch interpolation technique was used to determine %VA. Transcutaneous electrical stimuli were delivered to the tibial nerve using a high-voltage (≤ 400 V) constant-current stimulator (Digitimer DS7AH, Herthfordshire, UK). The cathode was a metal probe (8 mm diameter) with the tip covered in a saline-soaked sponge, which was pressed over the tibial nerve in the popliteal fossa. The anode is a 9 x 5 cm rectangular self-adhesive electrode (Durastick Supreme, Chattanooga Group, Hicton, TN) and was positioned just distal to the patella. Single stimuli (30 mA) were used to determine the optimal probe location by the investigator visually inspecting a computer monitor that displayed the twitch force and EMG signal for the SOL muscle after each exploratory stimulus. Once the best twitch was determined and the location of the probe was marked for all subsequent stimuli, the maximal compound muscle action potential (M-wave) was determined with incremental (5 mA) amperage increases until a plateau in the peak-to-

peak M-wave was observed. To ensure a supramaximal stimulus, 120% of the stimulus intensity (mA) that elicited the maximal M-wave was used during the evoked twitches and the twitch interpolation procedure. A single stimulus was a 200-μs duration square wave impulse, while a doublet consisted of two single stimului delivered successively at 100 Hz. Doublets were administered with the supramaximal stimulus intensity during the MVC trials to increase the signal-to-noise ratio and minimize the series elastic effects on force production (21). In accordance with the twitch interpolation procedure, a supramaximal doublet was administered 350-500 ms into the MVC plateau (superimposed twitch) and then again 3-5 s after the MVC trial at rest (potentiated twitch). %VA was calculated with the following equation (1):

$$\%VA = \left[1 - \left(\frac{\text{superimposed twitch}}{\text{potentiated twitch}}\right)\right] \times 100$$

Dorsiflexion Range of Motion

The ROM of the plantar flexors was determined for each participant during the acute stretching protocol using the isokinetic dynamometer programmed in passive mode. The isokinetic dynamometer passively moved the dynamometer lever arm at an angular velocity of $5^{\circ} \cdot s^{-1}$ from approximately -25º of dorsiflexion to the maximal tolerable ROM (as acknowledged by the subjects) and was then immediately returned to the starting position.

Musculotendinous Stiffness

For MTS, angle (°) and torque (Nm) values were sampled from the dynamometer during the flexibility assessments, which provided the passive torque-angle curves. To be consistent with previous studies (29, 46), MTS values ($Nm \cdot deg^{-1}$) were calculated as tangential slopes at specific joint angles using a fourth-order polynomial regression model that was fit to the torque-angle curves according to the procedures described and evaluated by Nordez et al. (58). MTS was determined for the right plantar flexors at every 4^{th} degree during the final 13º range of motion (i.e. 1°, 5°, 9°, and 13°), which was common to all subjects' MTS assessments with no visually identified increases in EMG amplitude (to ensure the measurements were passive). The torque values were gravity corrected with the weight of the platform being subtracted at each joint angle. Custom-written software (LabVIEW 8.5, National Instruments, Austin, TX) was used to fit the polynomial models and calculate the MTS values according to the procedures described by Ryan et al. (62).

Surface Electromyography

Pre-amplified bipolar, active surface electrodes (TSD150B, Biopac Systems Inc.; Santa Barbara, CA; nominal gain = 350; bandwidth = 12 – 500 Hz) with a fixed center-to-center interelectrode distance of 20 mm were placed on the SOL and MG muscles. For the SOL, the electrodes were placed along the longitudinal axis of the tibia at 66% of the distance between the medial condyle of the femur and the medial malleolus. The electrodes for the MG were placed on the most prominent bulge of the muscle in accordance with the recommendations of Hermens et al. (30). A single pre-gelled, disposable electrode (Ag-Ag Cl, Quinton Quick Prep, Quinton Instruments Co., Bothell,

WA) was placed on the spinous process of the 7th cervical vertibrae to serve as a reference electrode. To reduce skin impedance and increase the signal-to-noise ratio, local areas of the skin were shaved, lightly abraded, and cleaned with isopropyl alcohol prior to placement of the electrodes.

Signal Processing

The EMG (μV), force (N), torque (Nm), and angle (°) signals were recorded simultaneously with a Biopac data acquisition system (MP150WSW, Biopac Systems, Inc.; Santa Barbara, CA) during each isometric MVC, evoked twitch, and flexibility assessment. The force (N) signal from the load cell (Omegadyne, model LC402, range 0-500 lbs; Stamford, CT), angle (°) and torque (Nm) signals from the dynamometer, and the EMG (μV) signals recorded from the SOL and MG were sampled at 2 kHz for the MVC and evoked twitch assessment, while the same channels were sampled at 1 kHz for the flexibility assessment. All signals were stored on a personal computer (Dell Inspiron 8200, Dell, Inc., Round Rock, TX), and processed off-line using custom written software (LabVIEW 8.5, National Instruments, Austin, TX). The EMG signals were digitally filtered (zero-phase 4th –order Butterworth filter) with a pass band of 10-500 Hz. The force signal was smoothed with a zero phase shift 100-point moving average for the isometric and evoked twitch assessments. In addition, the force signal was gravity corrected for the weight of footplate so that the baseline force was 0 N. All subsequent analyses were performed on the filtered, smoothed, and gravity-corrected signals.

Isometric MVC force (N) was calculated as the average force value that occurred during the 0.25-s epoch taken immediately prior to the superimposed twitch.

Consequently, the same (concurrent) 0.25-s epochs were selected for the EMG signals. The time domain of the EMG signals were represented as root mean square (RMS) amplitude values.

Passive Stretching

The repeated PS of the right plantar flexor muscles were performed on the modified isokinetic dynamometer in passive mode (Figure 2). The dynamometer passively dorsiflexed the foot at $5°\cdot s^{-1}$ until the maximal tolerable torque threshold (identified during the familiarization trial) was met. The dynamometer maintained this torque, and stretched the plantar flexors for 135 s in accordance with the procedures of Herda et al. (29). The dynamometer was then returned to its starting position for 5 s and then repeated for a total stretching duration of approximately 20 min (9 passive stretching repetitions).

Stretch Training Protocol

Each participant in the STR group passively stretched their right plantar flexors in the same fashion as the PS protocol (above). They visited the laboratory three days per week (76) for four weeks and performed four 135 s stretches for a total of 9 min of time under stretch during each visit. Approximately 5 s of rest was given between stretches.

Statistical Analyses

Six separate four-way (2 × 4 × 2 × 2) mixed factorial ANOVAs [group (CON vs. STR) × joint angle (-19° vs. -9° vs. 1° vs. 12° dorsiflexion) × acute (pre- vs. post-

stretching) × chronic (week 1 vs. week 5)] were used to analyze the isometric MVC force, twitch forces, %VA, and EMG amplitude for the SOL and MG muscles. A four-way (2 × 4 × 2 × 2) mixed factorial ANOVA [group (CON vs. STR) × joint angle (1° vs. 5° vs. 9° vs. 13°) × acute (pre- vs. post-stretching) × chronic (week 1 vs. week 5)] was used to analyze the MTS data. A three-way (2 × 2 × 2) mixed factorial ANOVA [group (CON vs. STR) × acute (pre- vs. post-stretching) × chronic (week 1 vs. week 5)] was used to analyze dorsiflexion ROM. Finally, a two-way (2 × 2) mixed factorial ANOVA [group (CON vs. STR) × chronic (week 1 vs. week 5)] was used to analyze the corrected calf girths. When appropriate, follow-up analyses were performed using one-way repeated measures ANOVAs, dependent samples t-tests, and independent samples t-tests with Bonferroni corrections. An alpha of $P \leq 0.05$ was used to determine statistical significance, and all statistical analyses were performed using SPSS v. 15.0 (SPSS Inc., Chicago, IL).

In addition, to examine the magnitudes of change as a result of the acute stretching protocol, percent change scores and 95% confidence intervals were calculated from pre- to post-stretching at week 1 and 5 for the STR and CON groups. When the 95% confidence interval included 0, the percent change was not statistically different from zero (74).

CHAPTER IV

RESULTS

Isometric Maximal Voluntary Contraction Force

There was no four-way interaction (angle × acute × chronic × group, P=0.884), no three-way interactions for angle × acute × chronic (P=0.787), acute × chronic × group (P=0.812), angle × chronic × group (P=0.472), and angle × acute × group (P=0.849), no two-way interactions for angle × group (P=0.450), acute × group (P=0.969), angle × acute (P=0.653), angle × chronic (P=0.193), and acute × chronic (P=0.232), however there was a significant two-way interaction for chronic × group (P=0.043) and a significant main effect for acute (P<0.001) and angle (P<0.001). The marginal means for isometric MVC force (collapsed angle, chronic, and group) decreased from pre- to post-stretching (P<0.001, Figure 3A). The marginal means for MVC force (collapsed across acute and angle) increased from week 1 to week 5 for the STR group only (P<0.001, Figure 3B). The marginal means for MVC force (collapsed across acute, chronic, and group) at -19° was less than MVC force at -9°, 1°, and 12° (P<0.001), and MVC force at -9° and 12° was less than 1° (P<0.001, Figure 3C). The mean percent change scores for the STR (Figure 3D) and CON (Figure 3E) groups indicated that the acute stretching protocol elicited relative decreases in MVC force from pre- to post-stretching, which was unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Single Twitch Force

There was no four-way interaction (angle × acute × chronic × group, P=0.452), no three-way interactions for angle × acute × chronic (P=0.668), acute × chronic × group (P=0.207), angle × chronic × group (P=0.786), and angle × acute × group (P=0.156), no two-way interactions for angle × acute (P=0.227), chronic × group (P=0.833), acute × group (P=0.459), angle × group (P=0.801), and no main effect for group (P=0.248). However, there were significant two-way interactions for acute × chronic (P=0.019) and angle × chronic (P<0.001). The marginal means for single twitch force (collapsed across angle and group) during the pre-stretching at week 5 were greater than the post-stretching means at week 5 as well as the pre- and post-stretching means at week 1 (P<0.05, Figure 4A). The marginal means for single twitch force (collapsed across acute and group) at -19° were less than -9°, 1°, and 12° at both weeks 1 and 5 (P<0.05, Figure 4B). Single twitch force at -9° was also less than 1° and 12° at both weeks 1 and 5. In addition, the week 5 mean values were greater than the week 1 means for all joint angles (P<0.05, Figure 4B). The mean percent change scores indicated that the acute stretching protocol elicited decreases in twitch force, with larger decreases at the shorter muscle lengths (-19°, -9°, and 1°), for both the STR (Figure 4C) and CON (Figure 4D) groups at both weeks 1 and 5, which indicated that the acute stretching decrements were relatively unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Tetanic Twitch Force

There was no four-way interaction (angle × acute × chronic × group, P=0.500), no three-way interactions for angle × acute × chronic (P=0.874), acute × chronic × group (P=0.092), angle × chronic × group (P=0.214), angle × acute × group (P=0.203), no two-

way interactions for angle × acute (P=0.343), chronic × group (P=0.918), acute × group (P=0.166), angle × group (P=0.576), acute × chronic (P=0.087) and no main effect for group (P=0.301). However, there was a two-way interaction for angle × chronic (P=0.018) and a main effect for acute (P=0.008). The marginal means for tetanic twitch force (collapsed across angle, chronic, and group) decreased from pre- to post-stretching during weeks 1 and 5 at all joint angles for both groups (P<0.05, Figure 5A). The marginal mean for tetanic twitch force (collapsed across acute and group) at -19° was less than -9°, 1°, and 12° for both weeks 1 and 5 (P<0.05, Figure 5B). Tetanic twitch force at 1° was also greater than 12° at week 1, while 1° was also greater than -9° and 12° at week 5 (P<0.05, Figure 5B). In addition, the tetanic twitch force means were greater at week 5 than week 1 for all joint angles (P<0.05, Figure 5B). The mean percent change scores indicated that the acute stretching protocol elicited decreases in tetanic twitch force for both the STR (Figure 5C) and CON (Figure 5D) groups, although the stretching-induced decreases may have been smaller in magnitude than the single twitch forces (Figures 4C and 4D). The shorter muscle lengths (-19° and 1°) may have been more affected by the acute stretching protocol than the longest muscle length (12°) particularly for the CON group, but the overall percent change was relatively unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Percent Voluntary Activation

There was no four-way interaction (angle × acute × chronic × group, P=0.831), no three-way interactions for angle × acute × chronic (P=0.559), acute × chronic × group (P=0.825), angle × chronic × group (P=0.698), angle × acute × group (P=0.571), no two-

way interactions for angle × acute (P=0.488), chronic × group (P=0.950), acute × group (P=0.349), angle × group (P=0.574), acute × chronic (P=0.452), angle × chronic (P=0.772), and no main effects for group (P=0.301), acute (P=0.740), or chronic (P=0.120). However, there was a main effect for angle (P<0.001). The marginal mean for percent voluntary activation (collapsed across acute, chronic, and group) at -19° was greater (P<0.05, Figure 6A) than 1° and 12°. Percent voluntary activation at 12° was also less (P<0.05, Figure 6A) than -9° and 1°. The mean percent change scores for the STR (Figure 6B) and CON (Figure 6C) groups indicated that the acute stretching protocol did not elicit any meaningful changes in percent voluntary activation from pre- to post-stretching and was relatively unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Surface Electromyography

For the SOL, there was no four-way interaction (angle × acute × chronic × group, P=0.712), no three-way interactions for angle × acute × chronic (P=0.230), acute × chronic × group (P=0.339), angle × chronic × group (P=0.435), angle × acute × group (P=0.688), no two-way interactions for angle × acute (P=0.607), chronic × group (P=0.134), acute × group (P=0.205), angle × group (P=0.311), acute × chronic (P=0.664), angle × chronic (P=0.115), and no main effects for angle (P=0.288), chronic (P=0.127), acute (P=0.107), or group (P=0.775). In short, there were no changes or differences related to the acute or chronic stretching protocols or joint angle for EMG amplitude of the SOL muscle. The mean percent change scores for the STR (Figure 7A) and CON (Figure 7B) groups indicated that the acute stretching protocol had no meaningful effect

on EMG amplitude of the SOL, although most of the scores showed marginal increases from pre- to post-stretching. However, this pattern was unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

For the MG, there was no four-way interaction (angle × acute × chronic × group, P=0.751), no three-way interactions for angle × acute × chronic (P=0.829), acute × chronic × group (P=0.771), angle × chronic × group (P=0.667), and angle × acute × group (P=0.766), no two-way interactions for angle × acute (P=0.975), chronic × group (P=0.086), acute × group (P=0.697), angle × group (P=0.113), acute × chronic (P=0.566), angle × chronic (P=0.265), and no main effects for acute (P=0.338) or group (P=0.973). However, there were main effects for angle (P<0.001) and chronic (P=0.002). The marginal means for EMG amplitude of the MG muscle (collapsed across angle, acute, and group) were greater at week 5 than week 1 (P<0.001, Figure 8A). The marginal means for EMG amplitude (collapsed across acute, chronic, and group) were greater at -19º than -9º, 1º and 12º (P<0.001, Figure 8B). The mean percent change scores for the STR (Figure 8C) and CON (Figure 8D) groups indicated that the acute stretching protocol had no meaningful effect on EMG amplitude of the MG, although like the EMG values from the SOL, most of the scores showed marginal increases from pre- to post-stretching. However, this pattern was unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Range of Motion

There was no three-way interaction (acute × chronic × group, P=0.718), no two-way interactions for acute × chronic (P=0.365) or acute × group (P=0.489), but there was

a two-way interaction for chronic × group interaction (P=0.008) and a main effect for acute (P<0.001). The marginal means for ROM (collapsed across chronic and group) increased from pre- to post-stretching (P<0.001, Figure 9A) as a result of the acute stretching protocol. The marginal means for ROM (collapsed across acute) increased (P<0.001, Figure 9B) from week 1 to week 5 for the STR group only. The mean percent change scores for the STR and CON groups (Figure 9C) indicated that the acute stretching protocol increased ROM for both the STR and CON groups. However, the magnitudes of these relative increases were unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Musculotendinous Stiffness

There was no four-way interaction (angle × acute × chronic × group, P=0.285), no three-way interaction for acute × chronic × group (P=0.571), however, there were significant three-way interactions for angle × acute × chronic (P<0.001), angle × chronic × group (P=0.007), and angle × acute × group (P=0.047). For both the STR and CON groups, the marginal means for MTS (collapsed across group) decreased from pre- to post-stretching for all joint angles at weeks 1 and 5 (P<0.05, Figure 10A and 10B). The marginal means for MTS (collapsed across acute) decreased at all joint angles from week 1 to week 5 for the STR group only (P<0.05, Figure 10C and 10D). As expected, MTS increased with the increasing joint angles (1° < 5° < 9° < 13°) for both groups at weeks 1 and 5 (P<0.05, Figure 10C and 10D). The mean percent change scores for the STR (Figure 10E) and CON (Figure 10F) groups indicated that the acute stretching protocol

decreased MTS for both groups. However, the magnitudes of these relative decreases were unaffected by the chronic stretch training protocol (weeks 1 vs. 5).

Corrected Calf Girth

There was no two-way interaction (chronic × group, P=0.901) or main effects for chronic (P=0.461) or group (P=0.194). Corrected calf girth did not change from week 1 to 5 for either the STR or CON group (P>0.05).

CHAPTER V

DISCUSSION

Acute Effects of Stretching

The results of the present study suggested that an acute bout of prolonged, passive stretching decreases isometric MVC force (Figure 3A). These findings are consistent with many previous studies that have reported transient but detrimental decreases in muscle strength in the plantar flexors (25, 29, 75), leg extensors (8, 17, 18) and leg flexors (28, 49). For example, Weir et al. (75), Herda et al. (29), and Fowles et al. (25) reported 7%, 10%, and 28% decreases in isometric plantar flexion strength following 10 – 30 min of passive stretching. Recently, Ryan et al. (63) examined the acute effects of practical durations of passive stretching in the plantar flexors (2 – 8 min of time under stretch). The authors (63) reported that the decreases in isometric plantar flexion force following 2 min (2%), 4 min (4%), and 8 min (6%) of passive stretching were no different than a control (4%) condition, which was sitting dormant for 15 min. Therefore, our findings support these previous studies (25, 29, 63, 75) suggesting that prolonged (> 8 min time under stretch) passive stretching reduces voluntary isometric strength in the plantar flexor muscles.

Previous studies (28, 49) have suggested that the stretching-induced force deficit is muscle length-dependent with the losses in strength being more apparent at shorter muscle lengths. For example, Herda et al. (28) and McHugh et al. (49) reported that approximately 9 min of static stretching of the leg flexors decreased peak torque at the shortest muscle lengths, but had no effect or increased strength at the longer muscle

lengths. We hypothesized that 20 min of passively stretching the plantar flexors would elicit similar responses, such that isometric MVC force would be compromised at the shortest muscle lengths (-19° and -9°) and remain unaltered at the longest muscle length (12°). However, the present findings indicated that the acute stretching protocol decreased isometric MVC strength at all ankle joint angles (Figure 3A), which was contrary to our original hypotheses and the results of Herda et al. (28) and McHugh et al. (49). It should be noted, however, that a recent study by Kay and Blazevich (37) reported decreases in plantar flexion concentric strength following 3 min of passive stretching at 50%, 70%, and 90% of full dorisiflexion ROM (100%). Therefore, it is possible the differences between our results and those reported by Herda et al. (28) and McHugh et al. (49) are due to the muscle-specific differences between the hamstrings and plantar flexor muscles. However, future studies should test this hypothesis by comparing the stretching-induced force deficit at various joint angles for the leg flexors versus the plantar flexors.

Two general hypotheses have been suggested to explain the stretching-induced force deficit: a) neural factors that involve decreases in muscle activation (8, 18, 25, 29) and b) mechanical factors that involve alterations of the length-tension relationship, force-velocity relationship, and/or the viscoelastic properties of the muscle (16, 28, 49). For example, Fowles et al. (25) reported that within the initial 15-min post-stretching, the majority of force loss was due to an impaired ability to activate all available motor units. Other studies have demonstrated similar acute decreases in muscle activation using surface EMG and the twitch interpolation technique in the plantar flexors (25, 29) and leg extensors (8, 18). Fowles et al. (25) further noted that the underlying mechanisms for the

decreases in activation of the stretched muscles are related to Golgi tendon organ reflex activity, mechanoreceptor and nociceptor pain feedback, and/or fatigue-related mechanisms. In an attempt to elucidate the neural mechanisms underlying the stretching-induced force deficit, Herda et al. (29) suggested that similarities exist between the stretching- and vibration-induced decreases in muscle activation, which is likely related to a temporary impairment of gamma loop function. Cramer et al. (18) also suggested that a central nervous system mechanism may be responsible for the stretching-induced decreases in isokinetic peak torque production and EMG amplitude of the stretched leg as well as the unstretched, contralateral leg.

It is important to note, however, that not all studies have reported stretching-induced decreases in muscle activation. For example, Ce et al. (12) and Weir et al. (75) reported no changes in either %VA or EMG amplitude following 3.75 – 10 min of passive stretching of the plantar flexors. These findings agreed with Ryan et al. (63) who found no changes in %VA or EMG amplitude following 2 – 8 min of passive stretching of the plantar flexors. Thus, it is possible that the more fully activated distal muscles (39), such as the plantar flexors, require longer durations of stretching to diminish muscle activation (63). In contrast, a very recent study (37) found decreases in EMG amplitude of the plantar flexors during voluntary isokinetic muscle actions after only 3 min of passive stretching. Thus, conflicting evidence exists regarding the acute effects of stretching on the neuromuscular activation of the plantar flexors. To add to the disparity among the previous findings, the results of the current study reported no changes in muscle activation (EMG amplitude or %VA) following 20 min of passive stretching. These results are in contrast to those reported by Herda et al. (29) who found a 9%

decrease in EMG amplitude and a 3% non-significant decrease in %VA of the plantar flexors following a very similar stretching protocol as used in the present study (20 min of time under stretch). The differences among these studies and the present results is unclear, however, it is possibly related to the limitations of surface EMG. For example, Farina et al (24) suggested that the surface EMG underestimates the amount of activation sent from the spinal cord due to amplitude cancellation (cancellation of positive and negative phases of motor unit action potentials). In addition, the authors (24) suggested that ".. changes in the average rectified or root mean square EMG values after an intervention may not rigorously reflect altered levels of neural drive to the muscle" (p. 1488). Thus, although the lack of changes in %VA or EMG amplitude observed in the present study cannot rule out neural factors as one of the mechanisms responsible for the acute stretching-induced decreases in isometric MVC and twitch forces, it is likely that other mechanically-related mechanisms may have been responsible.

Another way to indirectly assess the acute effects of stretching on the ability of the central nervous system to fully activate the stretched muscles is to compare the voluntary and non-voluntary, evoked responses immediately after the stretching protocol (8). For example, Behm et al. (8) reported that 20 min of leg extensor stretching resulted in 12% decreases in MVC force and 12% decreases in single twitch force, however, there were no change in tetanic twitch force. The authors (6) concluded that if sufficient summation of twitches is provided through tetanic stimulation, there is no detectible force loss due to stretching, which implies that the loss of force due to stretching in the voluntary and single twitch responses was due to a central nervous system deficit. In contrast, the results of the present study found decreases in MVC force, single twitch

force, and tetanic twitch force at all joint angles. These findings tentatively suggested, therefore, that even when central nervous system activation is experimentally controlled, acute decreases in force production are still observed. Albeit, there was slightly less stretching-induced force deficit in the tetanic twitch forces compared to the single twitch forces (Figures 4C,4D,5C, and 5D), which suggested that if there was a neural contribution to the deficit, it was very small. Nevertheless, the present findings indicated that the stretching-induced force deficit was detectible even when muscle activation was voluntary, non-voluntary, and non-voluntarily summated, which implied that the acute stretching protocol elicited little, if any, affects on the central nervous system. It is possible that the conflicting results from this study and those reported by Behm et al. (8) are due to muscle-specific differences between the plantar flexors and quadriceps. This was also noted by Behm et al. (8) who suggested that " the greater size and differing fibre composition of the quadriceps may result in a muscle specific response (p.262)." In addition, these differences may also be related to the differences in stretching treatments, where Behm et al. (8) used a constant-angle (the angle at which the stretch occurs is held constant) stretching treatment and the current study employed a constant-torque (the torque at which the stretch occurs is held constant) stretching treatment. This was supported by Yeh and colleagues (80, 81) who reported that a constant-torque stretching treatment was more effective than a constant-angle (i.e. static stretch) treatment in reducing MTS in patients with hypertonicity. Thus, the stretching treatment used in this study may have resulted in a greater change in the compliance of the muscle-tendon unit. Overall, though, these findings suggested that there was very little (if any) contribution of

the central nervous system to the acute stretching-induced force deficit, which implies that the force deficit was largely mechanical in origin.

The other hypothesis that is often used to explain the stretching-induced force deficit involves mechanical alterations in the musculotendinous unit. Specifically, stretching-induced decreases in MTS may theoretically alter the shape of the length-tension relationship and/or sarcomere shortening velocity (16). For example, Cramer et al. (16) reported that although static stretching decreased isokinetic peak torque, it did not change the area under the isokinetic angle-torque relationship, which tentatively suggested that the stretching changed the shape of the length-tension relationship in the leg extensor muscles. This has been supported by previous authors who have reported joint-angle specific decreases in isometric torque production of the hamstrings (28, 49) and quadriceps (54) that seem to occur at the shortest muscle lengths. In theory, stretching would increase tendon and/or aponeurosis extensibility allowing greater muscle fiber shortening during an isometric contraction and therefore cause a rightward shift in the angle-torque curve (28). Therefore, the stretching-induced force deficit may only be apparent at muscle lengths shorter than the length for optimal force production. It should also be noted that the evoked twitch responses appear to be more affected at the shorter muscle lengths, as opposed to the longer muscle lengths, whereas voluntary force was affected throughout all the joint angles (Figures 4C, 4D, 5C, and 5D).

In support of the "mechanical" hypothesis there was a 21% decrease in MTS and a 17% increase in ROM from pre- to post-stretching. These findings were consistent with Herda et al. (29) and Fowles et al. (25) who found 38% and 27% decreases in MTS in the plantar flexors, respectively. The increases in ROM were also in agreement with

previous studies (29, 50, 63) examining the plantar flexor muscles that demonstrated 8-19% increases in ROM. There are several hypotheses that have been proposed to explain the acute, transient stretching-induced decreases in MTS and increases in ROM, which include alterations in tendon compliance (43), fascicle length (25), and intramuscular connective tissue (53). However, a recent study by Morse et al. (53) reported a distal shift of the musculotendinous junction in the MG following 5 min of passive stretching, which was unrelated to changes in tendon compliance or fascicle length. Therefore, it is possible that, based on the findings of Morse et al. (53), the decreases in MTS and increases in ROM may have been due to alterations in the viscoelastic properties of the intramuscular connective tissues (i.e., perimysium). Since the layers of connective tissues that surround the muscle fibers, fascicule, and muscle belly join to form the tendon (11), it is possible that any stretching-induced alterations of these tissues may affect their ability to transfer force from the contractile component to the bone, thereby reducing the muscle's ability to produce maximal force. The results of the present study supported this hypothesis and provided further evidence that the mechanical alterations in the muscle-tendon unit may contribute to the acute stretching-induced force deficits often seen during both voluntary and evoked contractions.

To date there are only two studies that have examined the effects of chronic stretching on the acute stretch-induced force deficit. Behm et al. (7) reported that the magnitude of the stretch-induced force deficit in the leg extensors was unaffected by four weeks of chronic stretching. However, Chaouachi et al. (13) recently reported that 6 weeks of stretch and sprint training diminished the detrimental effects of static stretching compared to the sprint only training group in 13-15 year-old adolescents. Our findings

supported those of Behm et al. (7) and demonstrated that 4 weeks of chronic stretch training had relatively no influence on the magnitude of the stretching induced changes in voluntary and evoked force, MTS, ROM, and muscle activation (%VA and EMG amplitude). The disparity between our study and Behm et al. (7) compared to those reported by Chaouachi et al. (13) are unclear. However, the stretch training program was longer for Chaouachi and colleagues (13) (6 vs. 4 weeks) suggesting that longer stretch training periods may be necessary to influence the acute stretching-induced force impairments. In addition, it may also be possible that the training status of the participants influences the magnitude of the stretching-induced force deficit. For example, previous studies have reported that an acute bout of stretching does not influence isokinetic peak torque (22) and vertical jump height (73) in trained women's basketball players. Although, future studies are needed to determine if longer stretch training programs influence the magnitude of stretching-induced deficits in both trained and untrained participants.

Chronic Effects of Stretching

Although many previous studies (8, 17, 18, 25, 28, 29, 49) have reported that acute stretching may result in detrimental decreases in muscular performance, a recent review by Shier (67) has suggested that chronic stretching studies have either reported improvements or no changes in performance. For example, Kokkonen et al. (41) found 15-32% increases in leg flexion and leg extension 1 RM strength, Worrell et al. (79) reported increases in leg extension eccentric and concentric peak torque at $120°\cdot s^{-1}$, and Wilson et al. (77) reported increases in rebound bench press strength. Conversely, other

authors have reported no change in isometric leg extension strength (33), leg flexion peak torque (45), and isometric plantar flexion strength (27) in response to a chronic stretching regiment. The results of the present study suggested that 4 weeks of stretch training of the plantar flexors increased isometric strength at all joint angles by 15% (Figure 3B). Therefore, despite some conflicting evidence, our findings are in agreement with previous studies (41, 77, 79) suggesting that chronic stretching enhances muscle strength. The mechanisms responsible for increases in muscle strength have been postulated by Kokkonen et al (41) to be a result of muscle hypertrophy. These hypotheses have been suggested due to a number of animal models who have demonstrated substantial stretch-induced muscle hypertrophy (14, 15, 20, 69). For example, Stauber and colleagues (69) demonstrated increases in rat soleus muscle mass and cross-sectional area increased by 13% and 30%, respectively, following stretching 3 times per week for 4 weeks. Similar results have been reported by Coutinho et al. (14) who found 16% increases in rat soleus fiber area following 40 min of stretching 3 days a week for 3 weeks. However, using corrected calf girth estimates (70) the results of the present study found no increases in calf size following 4 weeks of stretch training. Other previous studies (33, 41) have also suggested that increases in performance following stretch training are due to increases in muscle length or the addition of sarcomeres in series. Such adaptations have been reported in animal models where chronic stretching (40 min every 3 days for 3 weeks) increased muscle length by 5% and serial sarcomere number by 4%. These findings have been indirectly supported in previous human studies (47, 60) that have reported decreases in MTS following stretch training and subsequently proposed that these adaptations were also due to an increase in the number of sarcomeres in series. Theoretically, an addition

of sarcomeres in series may place more sacormeres at their optimal operating length and allow them to operate over a smaller range of their length-tension relationship (40). Therefore, it is possible that based on present findings the increases in voluntary muscle strength across all joint angles in the STR group (15%) and negligible increases in the CON group (1%) were due to the structural adaptations in the muscle by the addition of sarcomeres in series. To further support the stretching-induced changes in muscle longitudinal size, there were significant increases observed in MG EMG amplitude from week 1 to week 5, with relatively larger increases observed in the STR (26%) versus the CON (11%) group. These results tentatively suggested that the stretching induced longitudinal hypertrophy increases overall muscle activation of the MG. It may also be noted that there were significant increases in single and tetanic twitch forces for both groups from week 1 to week 5. These findings were in contrast to Guissard and Duchateau (27) who reported no increases in evoked force under both single and double stimulus (i.e., doublet) conditions. Although it is unclear the mechanisms behind the increases in twitch forces for the control condition, these findings also provide further support of the overall increase in muscle strength during both voluntary and evoked conditions following 4 weeks of regular chronic stretch training.

Perhaps the most noted reasons for participating in chronic stretching programs are related to increases in flexibility, or the pain free ROM and increase in muscle-tendon unit compliance (65). Many previous studies have reported increases in ROM for the plantar flexors (26, 27, 47), hamstrings (44, 46, 60), and gleno-humeral joint (77) following stretch training programs. The results of the present study supported these findings demonstrating a 20% increase in dorsiflexion ROM for the STR group and a 3%

decrease in dorsiflexion ROM in the CON group from week 1 to week 5 (Figure 9B). Several mechanisms have been proposed to explain the chronic increases in joint ROM, which include increases in stretch tolerance (44, 46), decreases in reflex activity (27, 35), and alterations in the mechanical properties of the muscle-tendon unit (27, 47, 60). Most notably, Magnusson et al. (46) reported increases in hamstring ROM and peak tolerable torque but no changes in the passive mechanical properties of muscle-tendon unit (i.e. stiffness and energy absorbed) or surface EMG. The authors concluded that increases in ROM are due to the increases in stretch tolerance of the subject rather than mechanical or viscoelastic changes of the muscle-tendon unit. This hypothesis was later supported by LaRoche et al. (44) who reported similar findings in the hamstrings following 4 weeks of training. Conversely, studies examining the effect of chronic stretching in the plantar flexors have reported decreases in MTS (27, 47) and reflex activities (27) (i.e. Hoffman and Tendon reflexes). These changes were supported by our findings showing 16% decreases in MTS for the STR group and 6% decreases in the CON group from week 1 to week 5 (Figure 10C and 10D). As noted earlier (53), the adaptations that occur following acute bouts of stretching (i.e. alterations in intramuscular connective tissue) are likely the mechanisms responsible for the decreases in MTS seen in the plantar flexors following chronic stretching periods. It is also possible that the differences between our findings and others examining the plantar flexors (27, 47) and those reported by Magnusson et al. (46) and LaRoche et al. (44) may be due to muscle specific differences, where larger muscles groups (i.e hamstrings) require longer stretch training programs to elicit similar mechanical adaptations as typically seen in the plantar flexors (27, 47). However, future studies are needed to examine this hypothesis.

Length – Tension Relationship

As noted in many previous (2, 31, 51, 52, 78) studies, the amount of isometric force that can be produced during a muscle contraction is dependent upon the length of the muscle (59). The length-tension relationship of a muscle or group muscles is often obtained from isometric contractions at varying muscle lengths (59). The results of the present study suggested that force production increased from -19° to 1° of dorsiflexion and then slightly decreased at 12° of dorsiflexion during both voluntary (Figure 3C) and evoked (Figure 4B and 5B) conditions. Many previous studies (32, 36, 51, 52) have reported similar trends for the length-tension relationship in the plantar flexors, where force increased from shorter to longer muscle lengths. However, MVC force was reported to be the greatest at muscle lengths ranging between 7 – 15° of dorsiflexion. It is important to note that the differences among these studies and those reported in this study may be due to subject positioning, where the subjects in the aforementioned studies were tested in the prone position and the subjects in the current study were seated at a hip angle of 135° (between the trunk and thigh). Thus, although speculative, it is possible that a greater hip angle may cause a left-ward shift in the length-tension relationship causing the maximal forces to occur at angles less than 15° of dorsiflexion. This was recently discussed by Kay et al. (37) who found ankle ROM was limited and subjects experienced discomfort behind the knee when seated at a hip angle of 105° and 120°.

In addition to the muscle length dependent changes in force, it is evident that changes in muscle length also influence muscle activation. For example, some previous studies have reported decreases (52, 64, 78) in EMG amplitude while others have

reported increases (3, 19) in muscle activation from shorter to longer muscle lengths. The results of the present study suggested that muscle activation (% VA and EMG amplitude) decreased from the shortest (-19°) to longest (12°) muscle length. Studies that have examined the plantar flexors under similar joint angle conditions (leg fully extended) have reported similar findings. For instance, Winegard et al. (78) reported that motor unit activation increased at the shorter muscle lengths and Miyamoto et al. (52) found non-significant decreases in EMG amplitude with increases in ankle joint angle. Sale and colleagues (64) reported similar findings with the leg flexed at 90°. When normalized to the maximal M-wave, plantar flexor EMG amplitude decreased with increases in muscle lengths. These findings are in agreement with the hypothesis stated by Kennedy et al. (38) suggesting that the muscle must "… be activated to a greater extent to compensate for the limited force contribution of the shortened gastrocnemius" (p. 63).

Another proposed adaptation attributed to chronic stretching is the alteration of the length-tension relationship. In theory, chronic stretch training would cause a right-ward shift in the angle-torque relationship, where the addition of sarcomeres in series would increase fascicle length and lead to less strength at shorter muscle lengths but greater strength at the longer muscle lengths. A cross-sectional comparison by Alfonso et al. (2) supported this hypothesis demonstrating that the angle-torque relationship was shifted to the left in those with less flexible hamstrings when compared to those with more flexible hamstrings. However, the results of the current study found no alterations in the length-tension relationship following 4 weeks of stretch training. It may be possible that the lack of changes in the current study were due to the plantar flexors

operating primarily on the ascending limb of the length-tension relationship (36). Such alterations in the length-tension relationship may be more noticeable in muscles such as the hamstrings, which have previously demonstrated right-ward shifts in the angle-torque relationship following eccentric strength training (10). However, future studies are needed to determine if there are muscle specific differences in the changes of the length-tension relationship following chronic stretch training.

Conclusion

Overall, our findings are consistent with many previous studies that have reported stretching-induced decreases in muscle strength (8, 17, 18, 25, 28, 49). However, the lack of changes in %VA and EMG amplitude from pre- to post-stretching are in contrast to previous studies examining similar durations of stretching (i.e. 20 – 30 min) (8, 25, 29). Furthermore, the decreases in MTS, evoked twitch forces, and increases in ROM are similar to previous studies who have proposed the presence of "mechanical factors" accounting for the stretching-induced decreases in strength (25, 29, 37). As a result of these findings, it is possible that the stretching-induced force deficit may be more mechanical in origin. Following 4 weeks of stretch training, the STR group experienced increases in maximal force production at all joint angles, increases in ROM, decreases in MTS, increases in MG EMG amplitude, and no change in corrected calf girth. These findings support the work of previous authors (41, 77, 79) who reported improvements in muscular performance following chronic stretch training regimens. However, these adaptations did not influence the magnitude of the acute stretching-induced changes in voluntary and evoked force production, muscle activation, ROM, MTS, or the length-

tension relationship. Therefore, it is possible chronic stretch training results in longitudinal hypertrophy (i.e. increased amount of sarcomeres in series) supported by the decreases in MTS and lack of changes in corrected calf girth. However, future studies are needed to test this hypothesis by examining fascicle length changes (i.e. ultrasound technology) after chronic stretch training. It may also be noted that maximal force production and muscle activation across the length-tension relationship followed a similar trend as reported in previous studies (32, 36, 51, 52), where force increased to a neutral joint angle (1°) and slightly decreased at longer muscle lengths (12°), whereas muscle activation decreased from shorter to longer muscle lengths.

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Appendix A.

Figure 1.

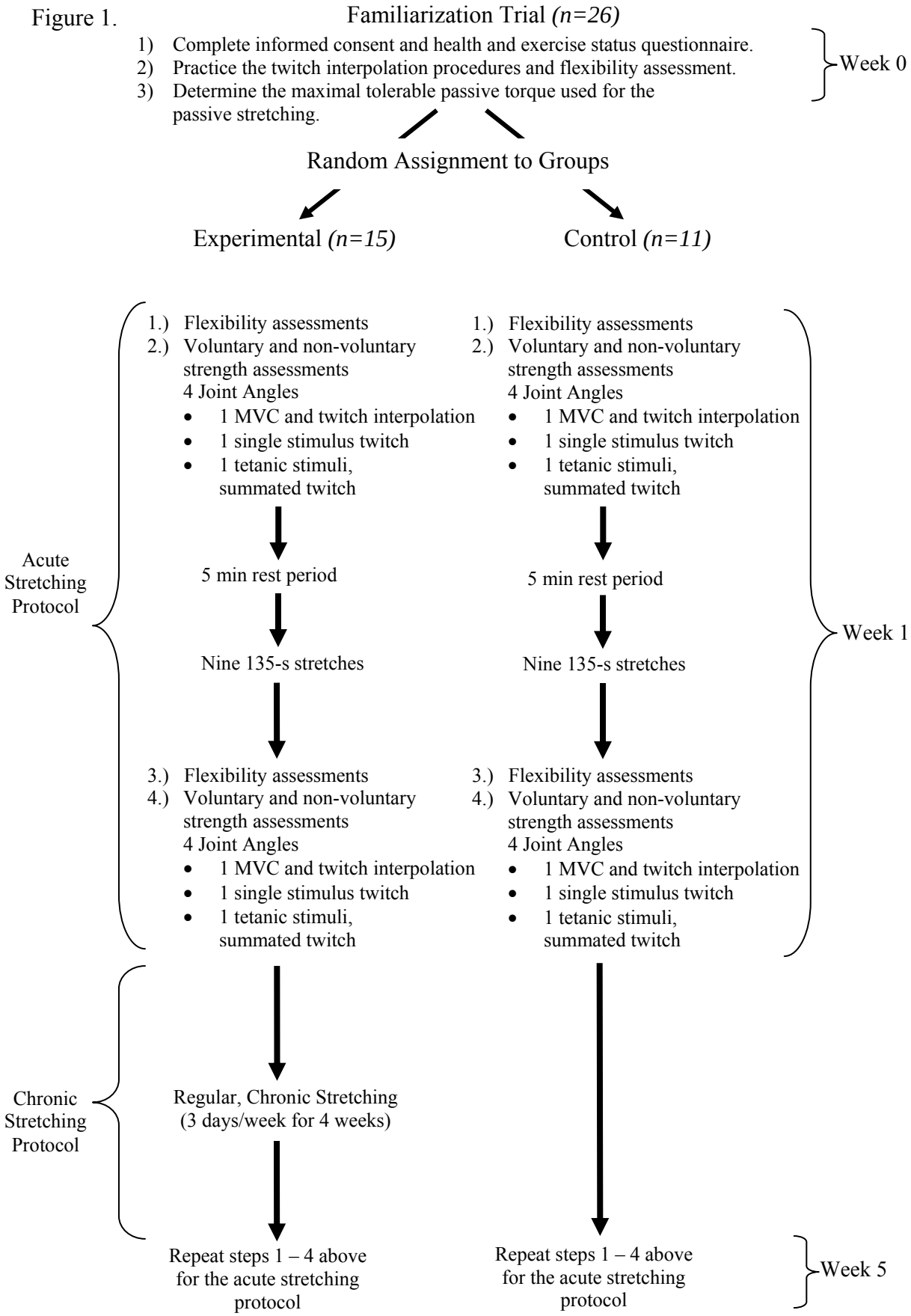

Figure 2.

A)

B)

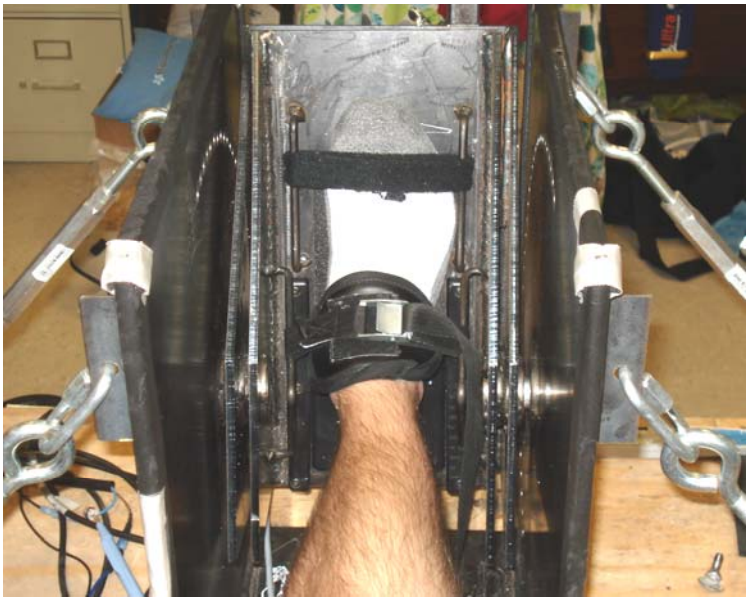

Figure 3.

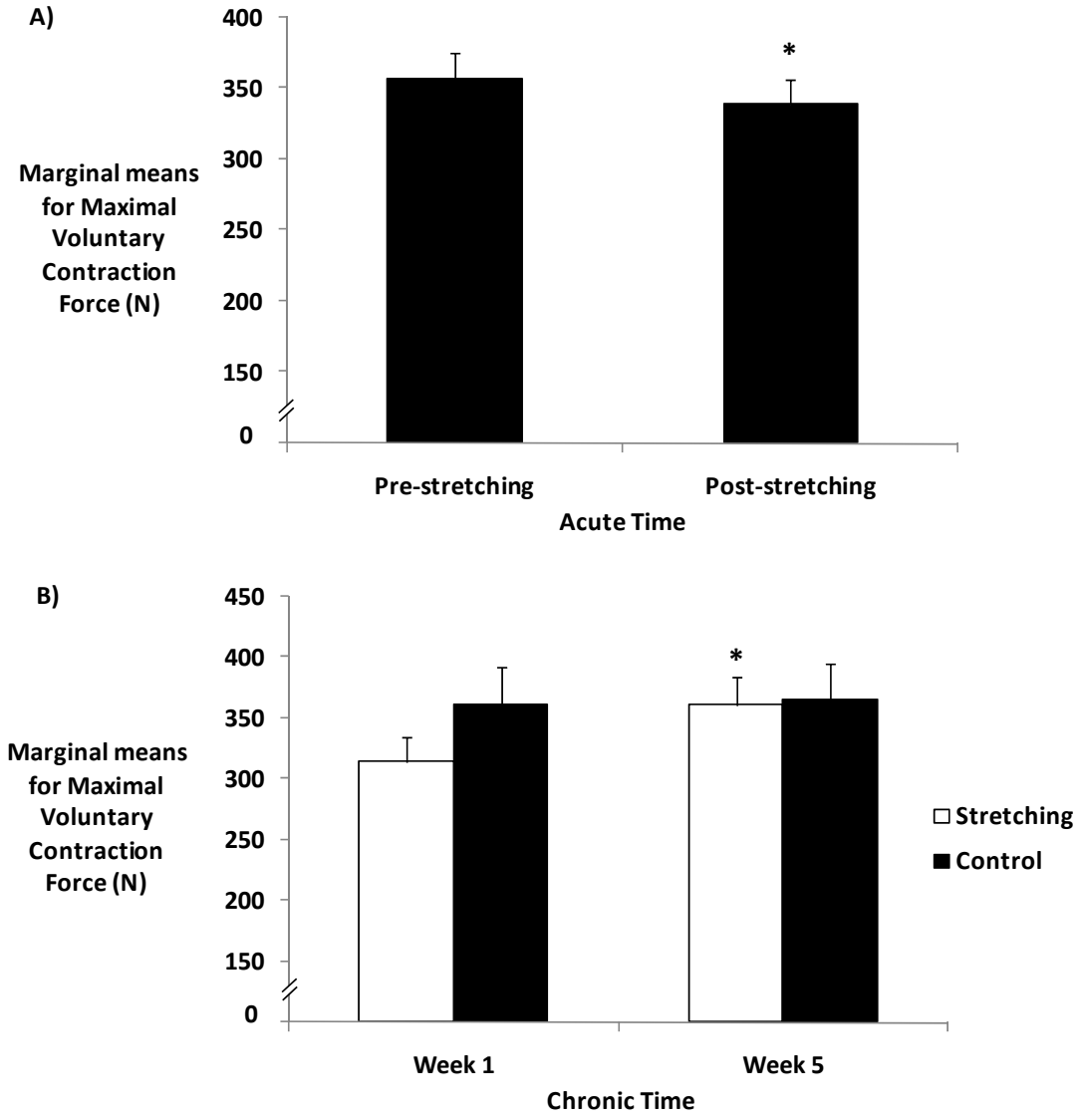
A)
400
350
300
250
200
150
0
Marginal means for Maximal Voluntary Contraction Force (N)
*
Pre-stretching
Post-stretching
Acute Time
B)
450
400
350
300
250
200
150
0
Marginal means for Maximal Voluntary Contraction Force (N)
*
Week 1
Week 5
Chronic Time
Stretching
Control

Figure 3.

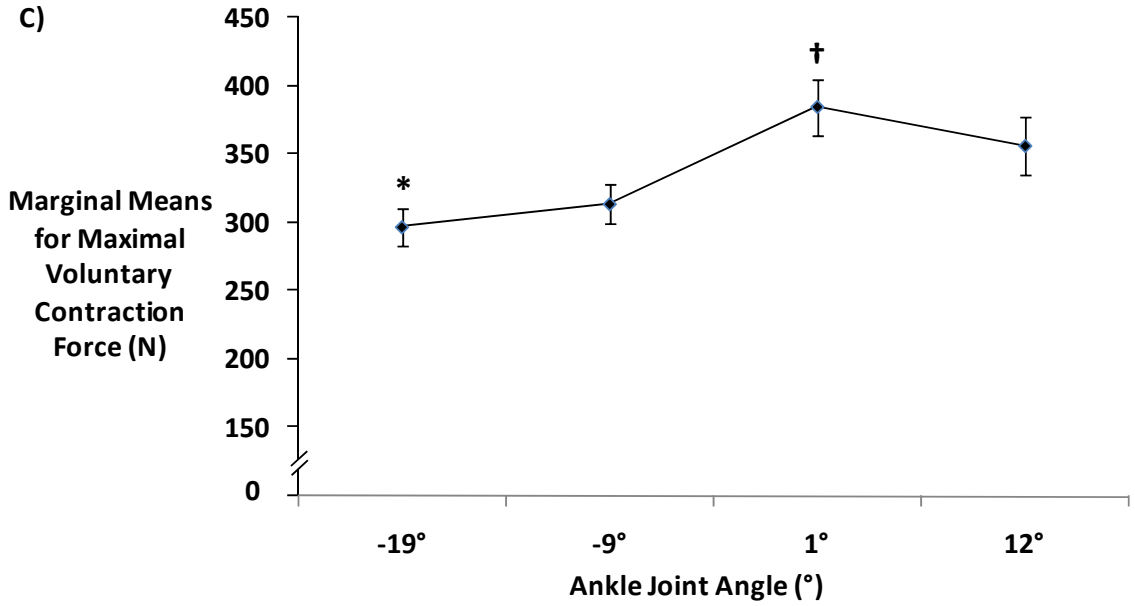
C)
450
400
350
300
250
200
150
0
Marginal Means for Maximal Voluntary Contraction Force (N)
*
†
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 3.

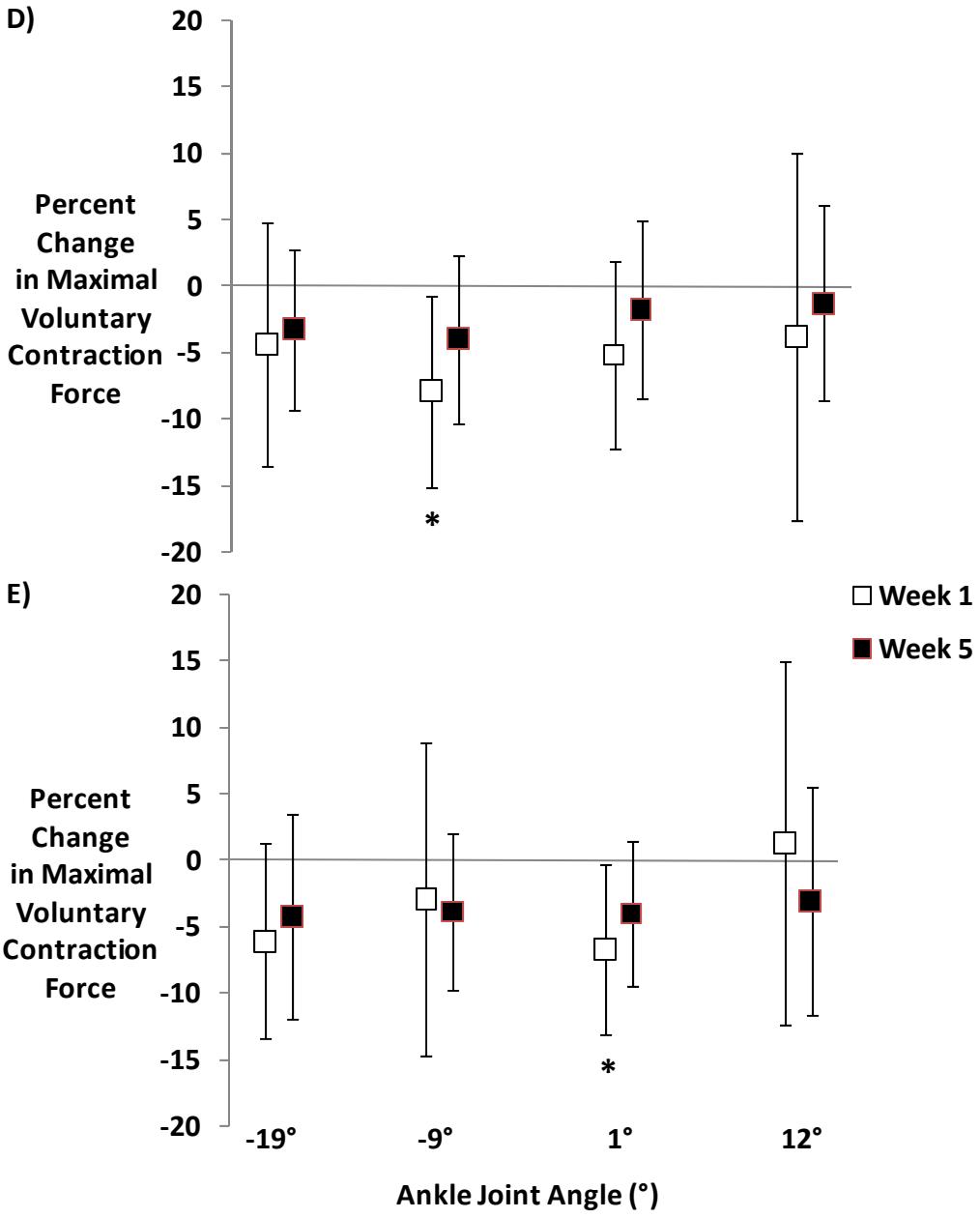
D)
20
15
10
5
0
-5
-10
-15
-20
Percent Change in Maximal Voluntary Contraction Force
*
E)
20
15
10
5
0
-5
-10
-15
-20
Percent Change in Maximal Voluntary Contraction Force
*
Week 1
Week 5
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 4.

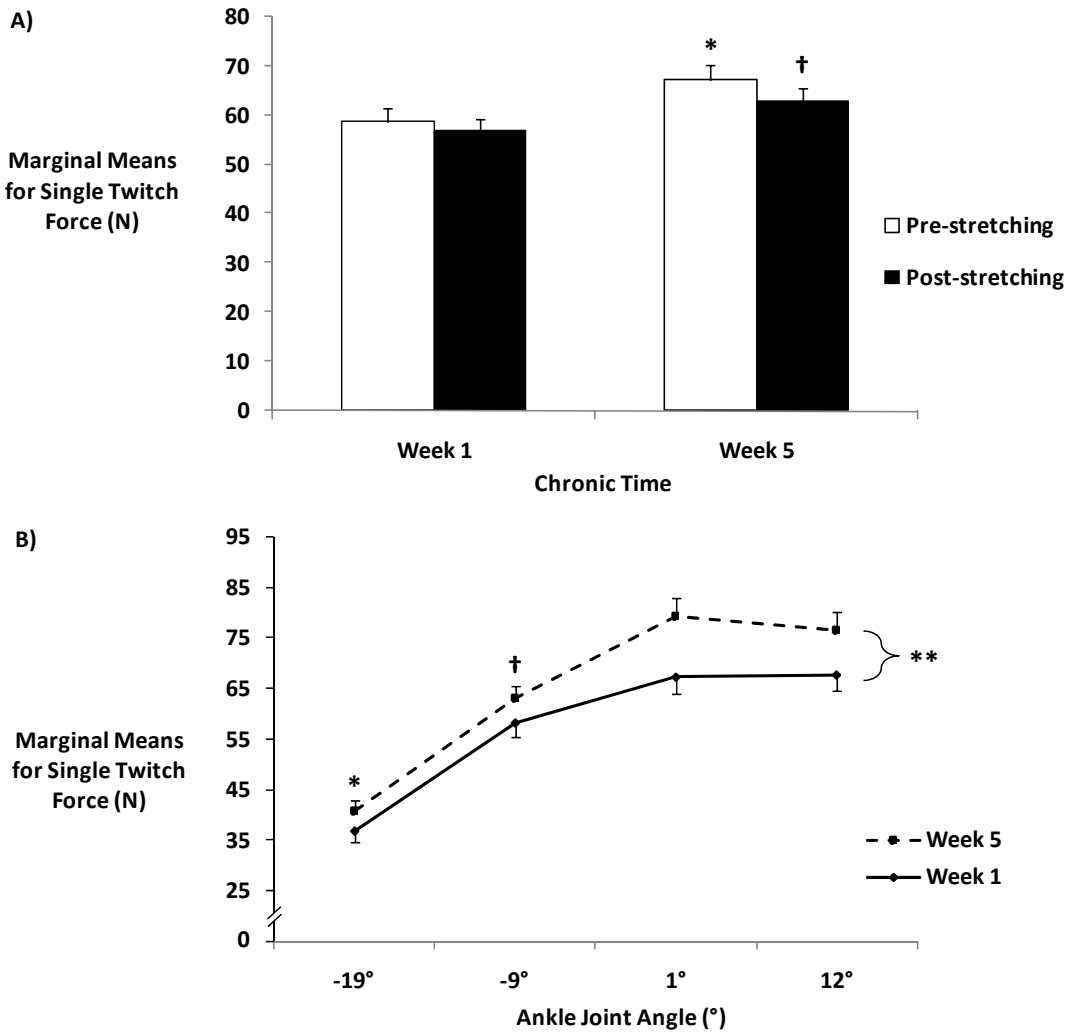
A)
Marginal Means for Single Twitch Force (N)
80
70
60
50
40
30
20
10
0
*
†
Pre-stretching
Post-stretching
Week 1
Week 5
Chronic Time
B)
Marginal Means for Single Twitch Force (N)
95
85
75
65
55
45
35
25
0
*
†
**
Week 5
Week 1
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 4.

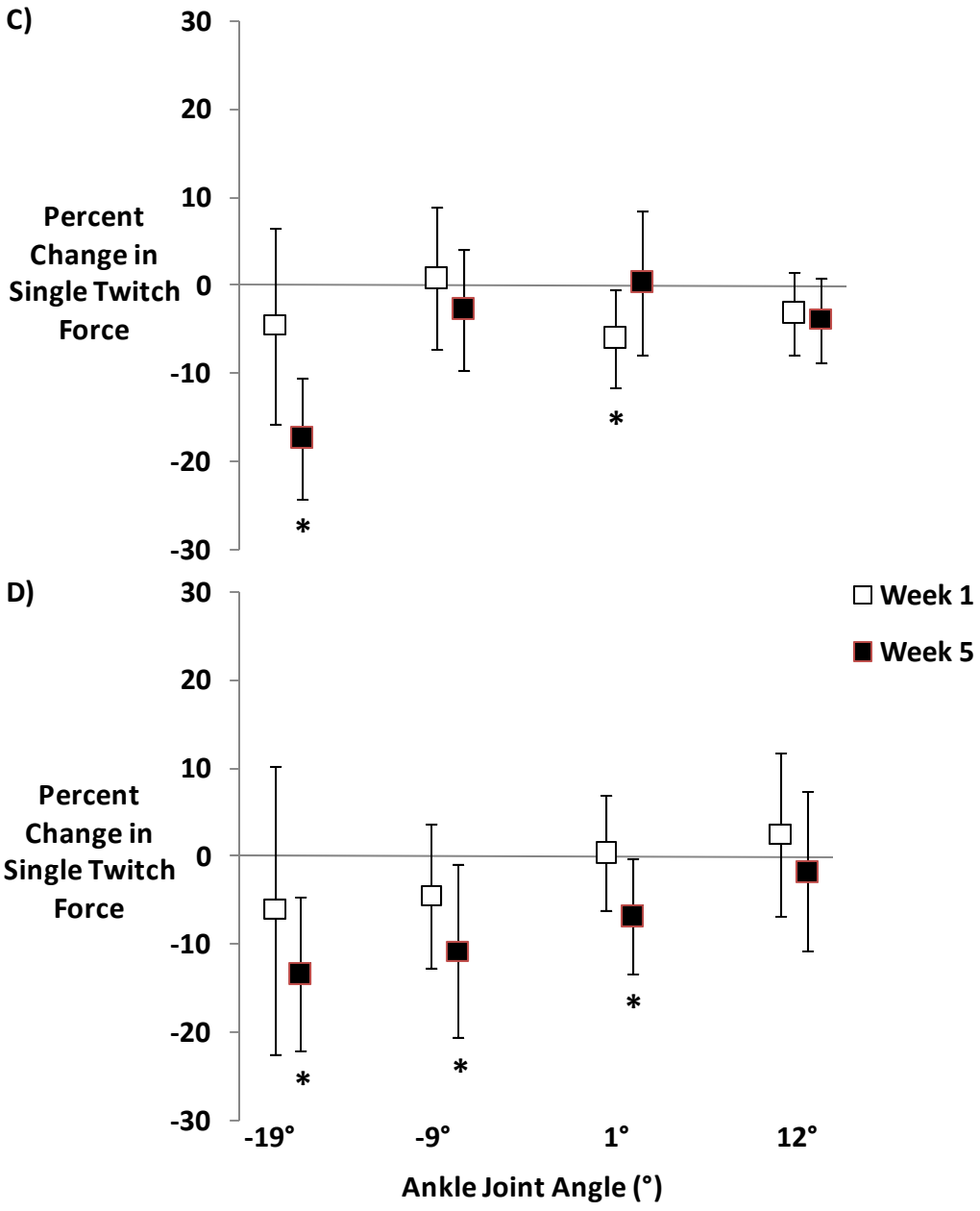
C)
30
20
10
0
-10
-20
-30
Percent Change in Single Twitch Force
*
*
D)
30
20
10
0
-10
-20
-30
Percent Change in Single Twitch Force
*
*
*
Week 1
Week 5
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 5.

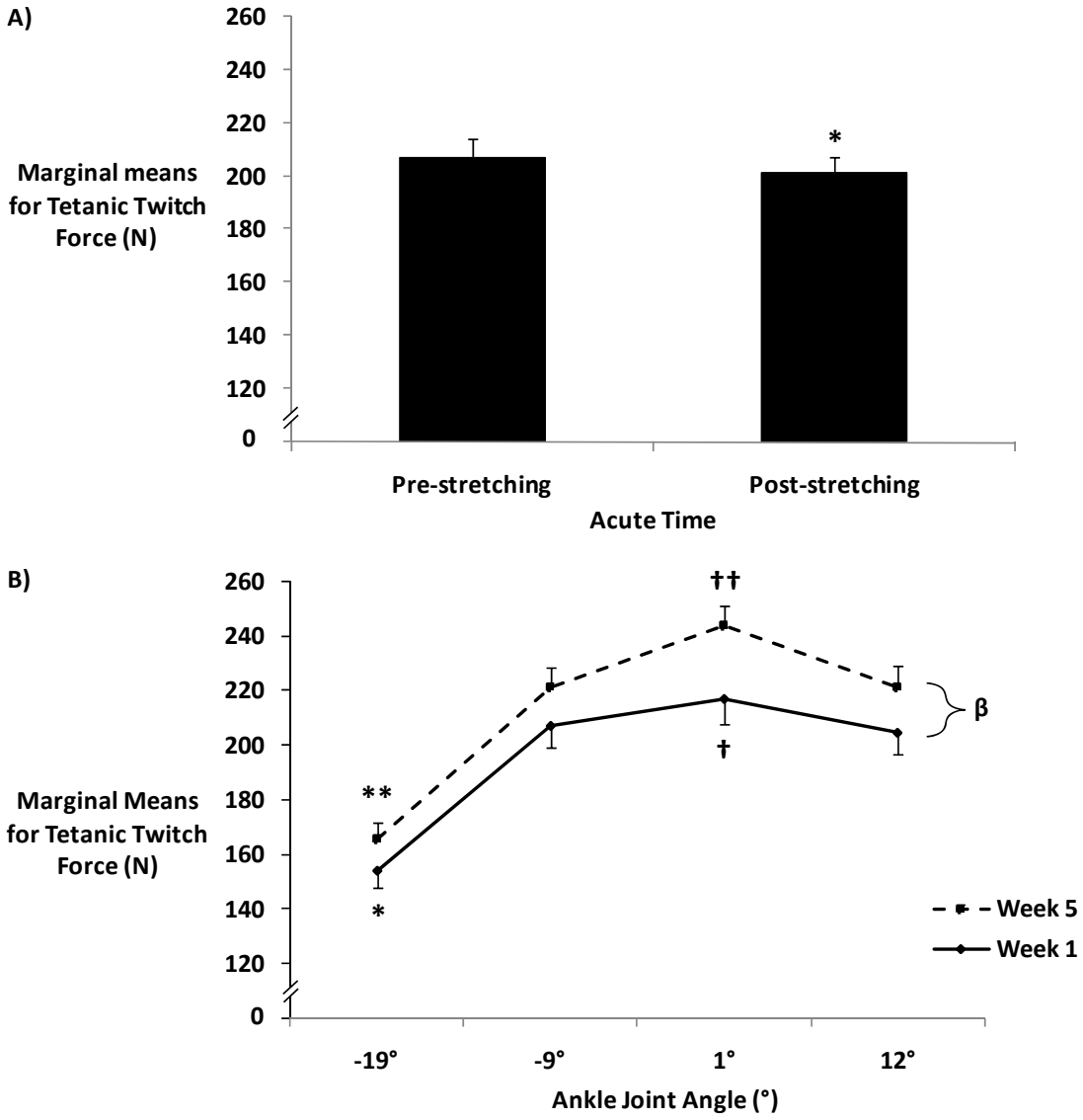
A)
Marginal means for Tetanic Twitch Force (N)
260
240
220
200
180
160
140
120
0
*
Pre-stretching
Post-stretching
Acute Time
B)
Marginal Means for Tetanic Twitch Force (N)
††
**
*
†
β
Week 5
Week 1
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 5.

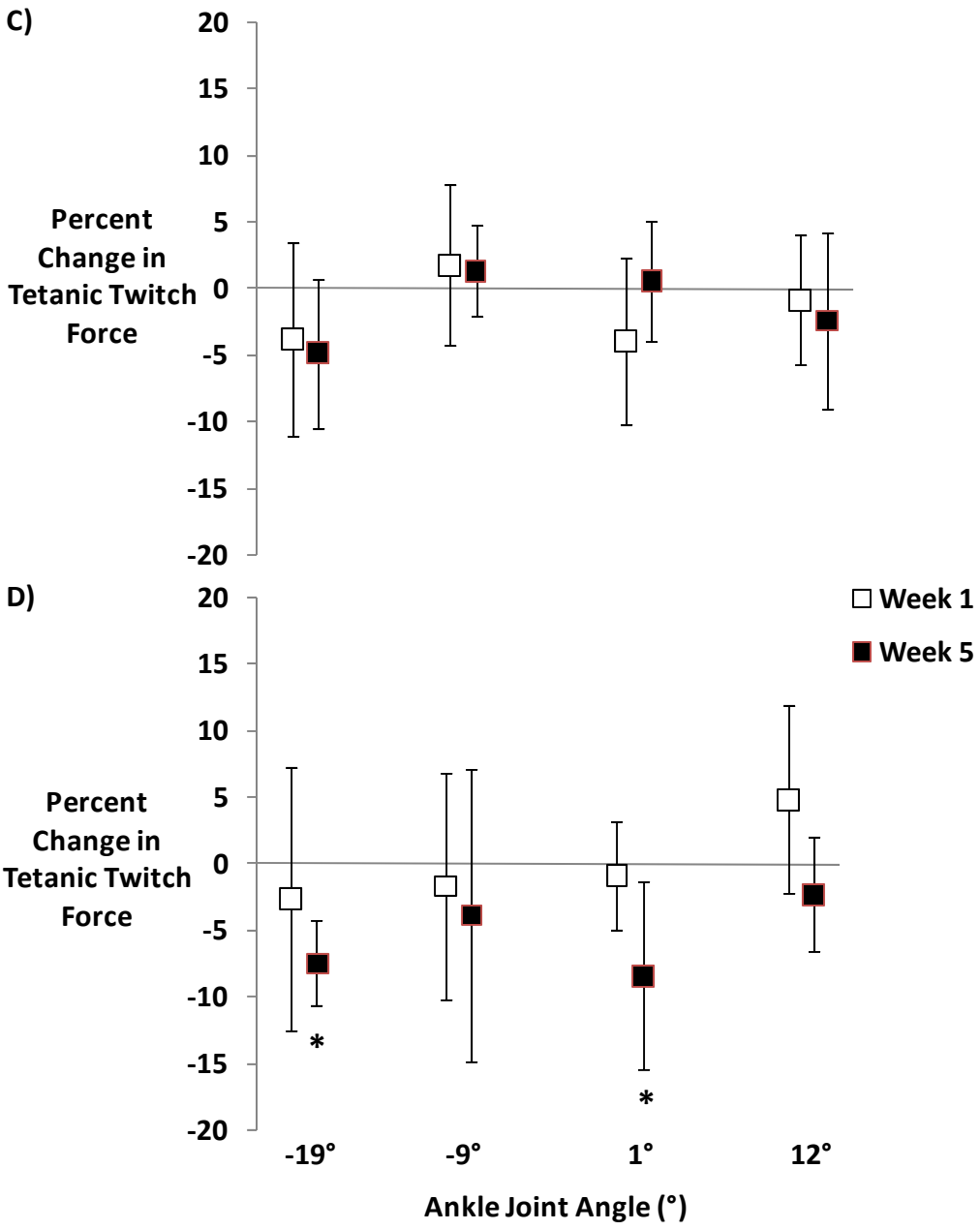
C)
20
15
10
5
0
-5
-10
-15
-20
Percent Change in Tetanic Twitch Force
D)
Week 1
Week 5
Percent Change in Tetanic Twitch Force
*
*
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 6.

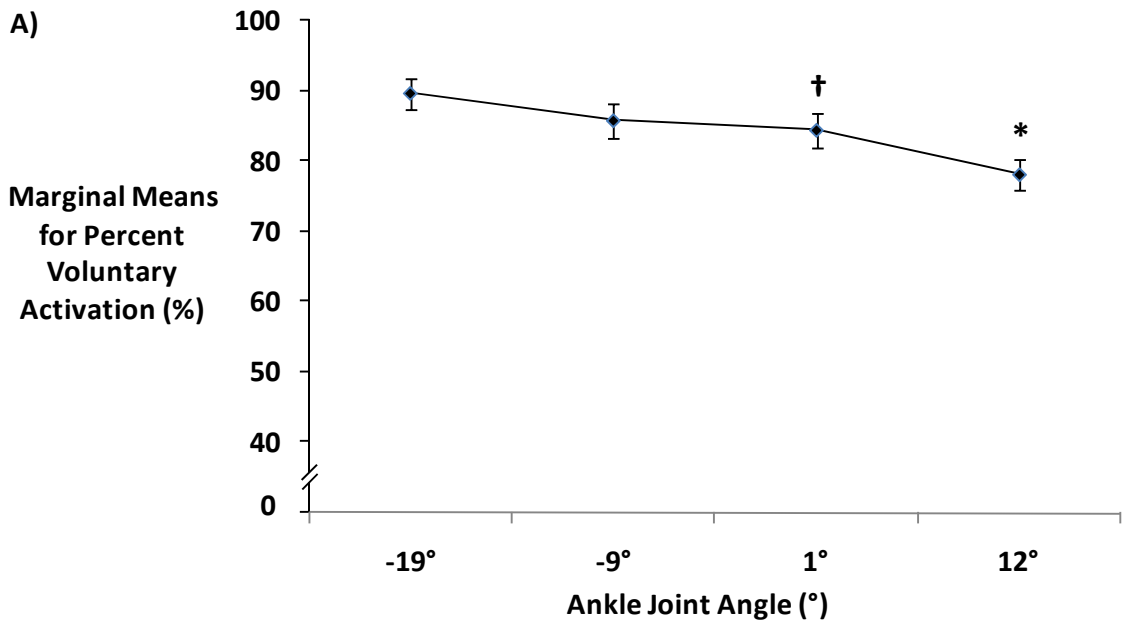
A)
100
90
80
70
60
50
40
0
†
*
Marginal Means for Percent Voluntary Activation (%)
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 6.

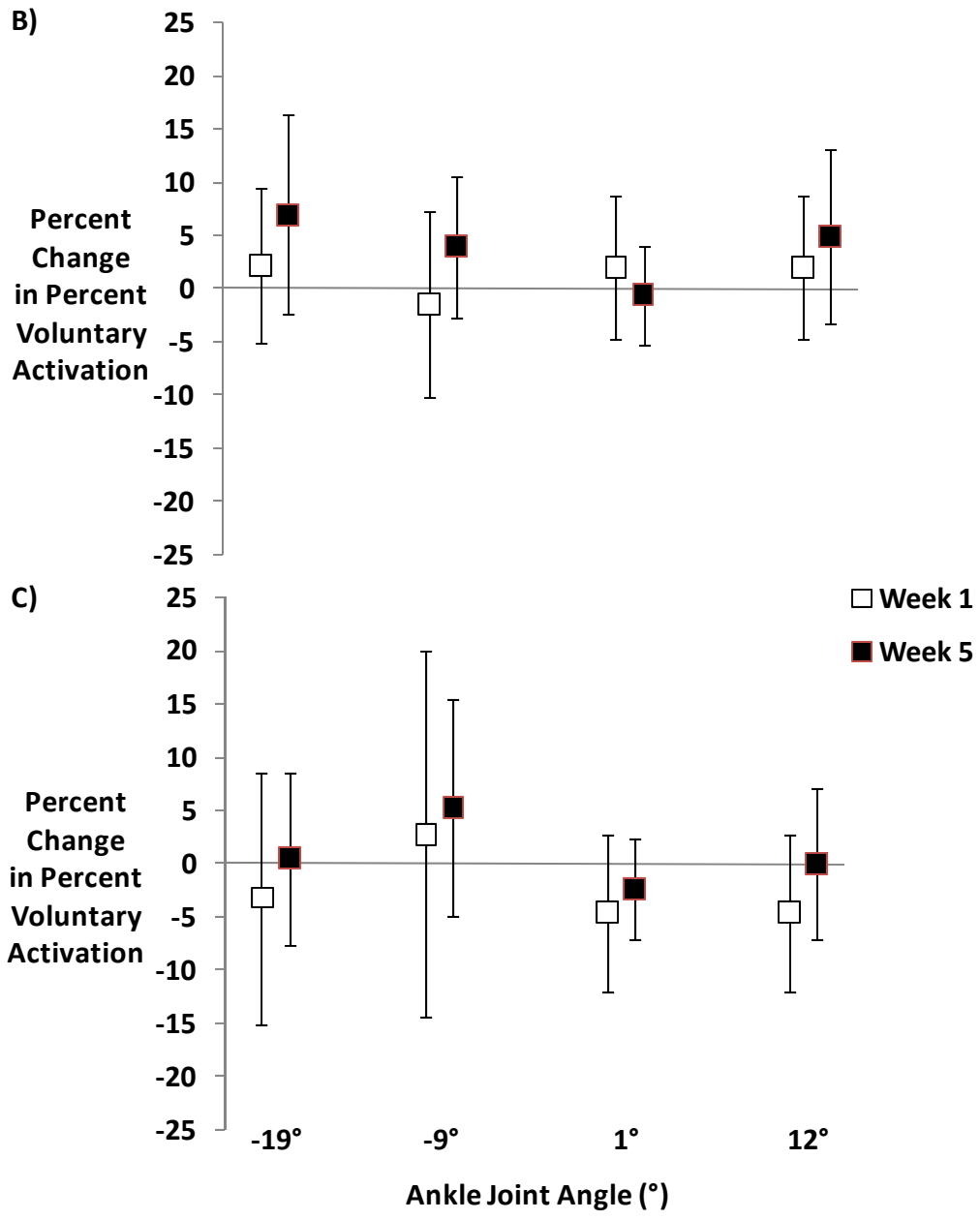
B)
25
20
15
10
5
0
-5
-10
-15
-20
-25
Percent Change in Percent Voluntary Activation
C)
Week 1
Week 5
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 7.

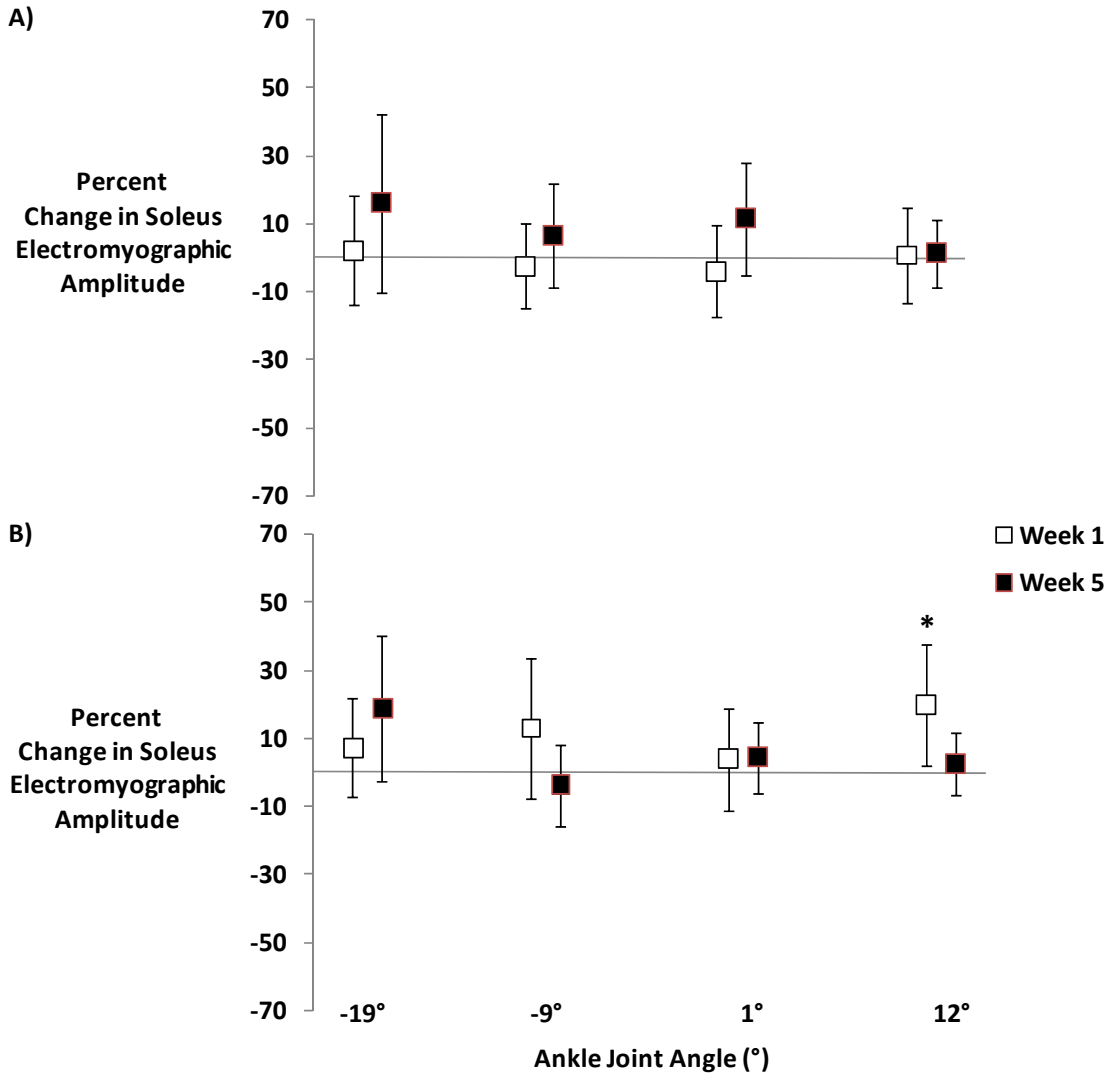
A)
B)
Percent Change in Soleus Electromyographic Amplitude
70
50
30
10
-10
-30
-50
-70
Week 1
Week 5
*
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 8.

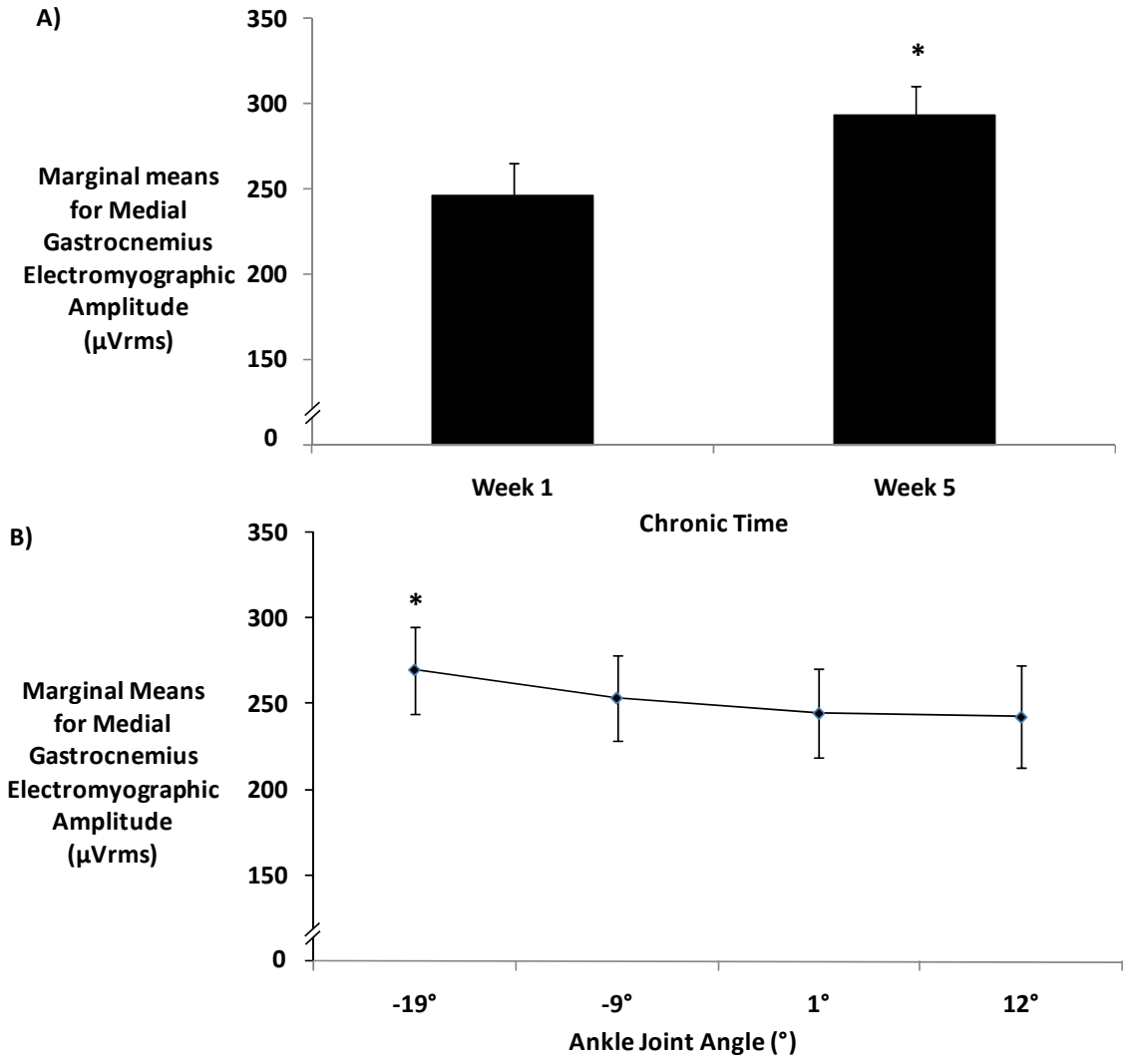
A)
350
300
250
200
150
0
Marginal means for Medial Gastrocnemius Electromyographic Amplitude (µVrms)
*
Week 1
Week 5
Chronic Time
B)
350
300
250
200
150
0
Marginal Means for Medial Gastrocnemius Electromyographic Amplitude (µVrms)
*
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 8.

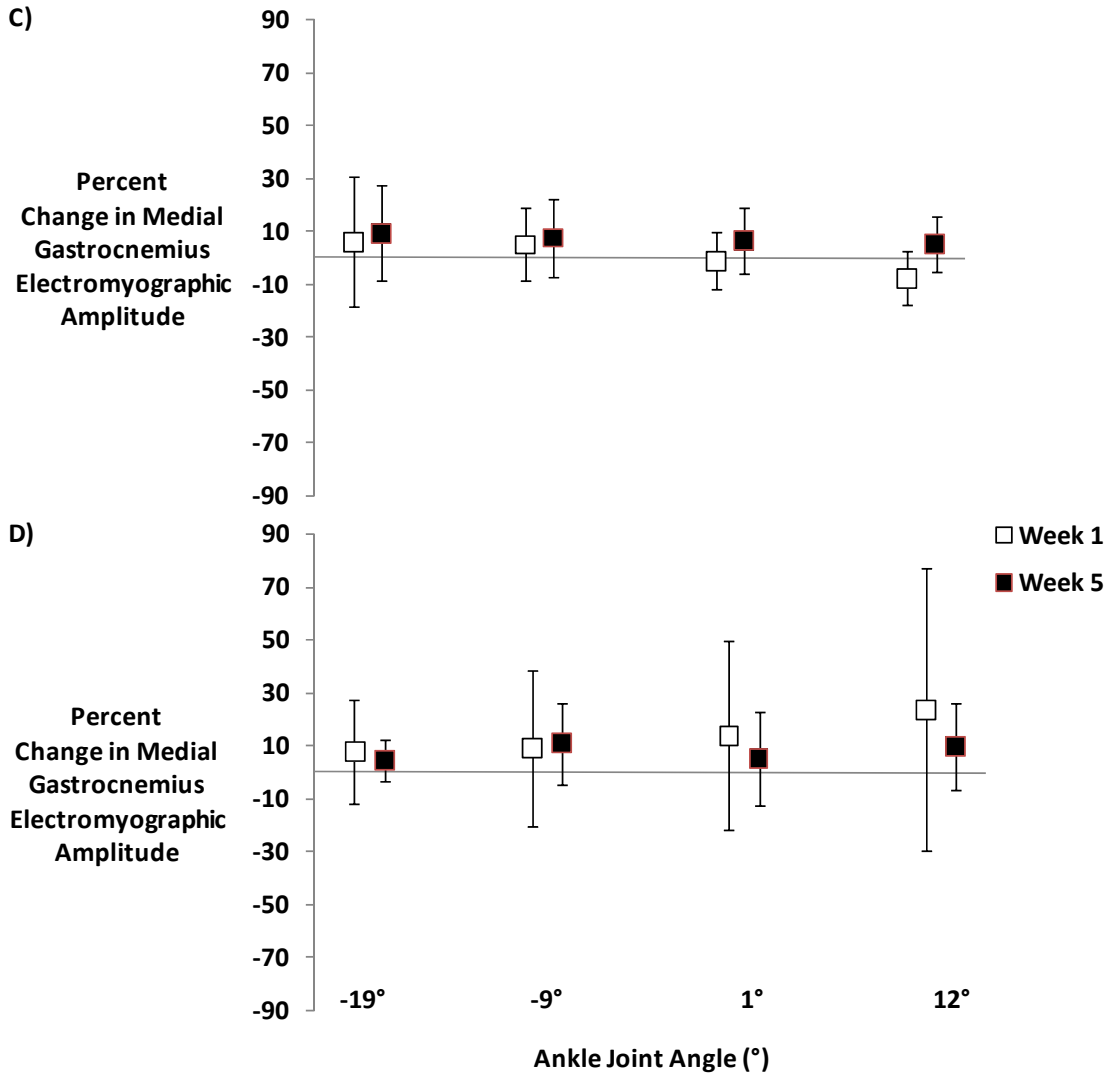
C)
90
70
50
30
10
-10
-30
-50
-70
-90
Percent Change in Medial Gastrocnemius Electromyographic Amplitude
D)
90
70
50
30
10
-10
-30
-50
-70
-90
Percent Change in Medial Gastrocnemius Electromyographic Amplitude
Week 1
Week 5
-19°
-9°
1°
12°
Ankle Joint Angle (°)

Figure 9.

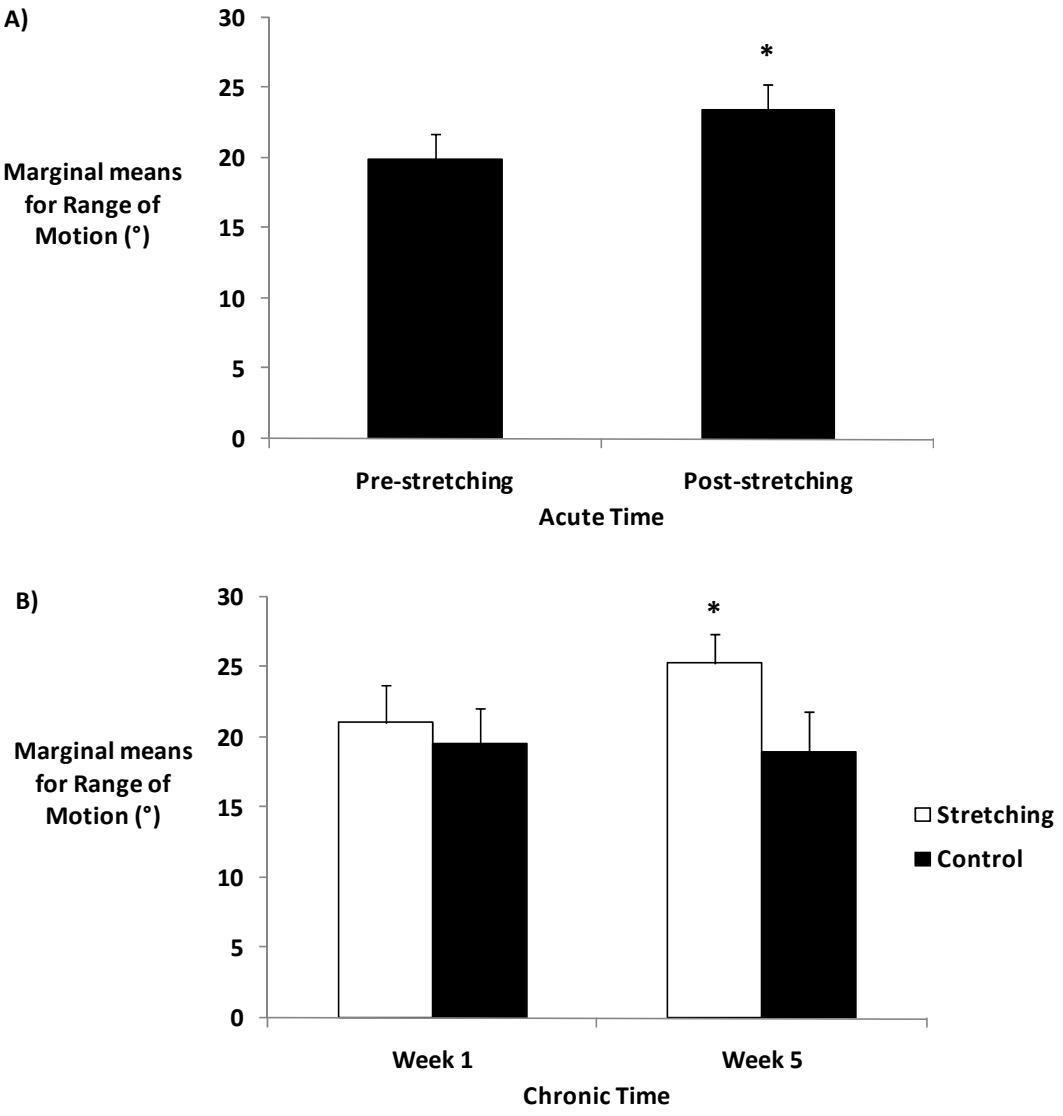
A)
Marginal means for Range of Motion (°)
30
25
20
15
10
5
0
*
Pre-stretching
Post-stretching
Acute Time
B)
Marginal means for Range of Motion (°)
30
25
20
15
10
5
0
*
Week 1
Week 5
Chronic Time
Stretching
Control

Figure 9.

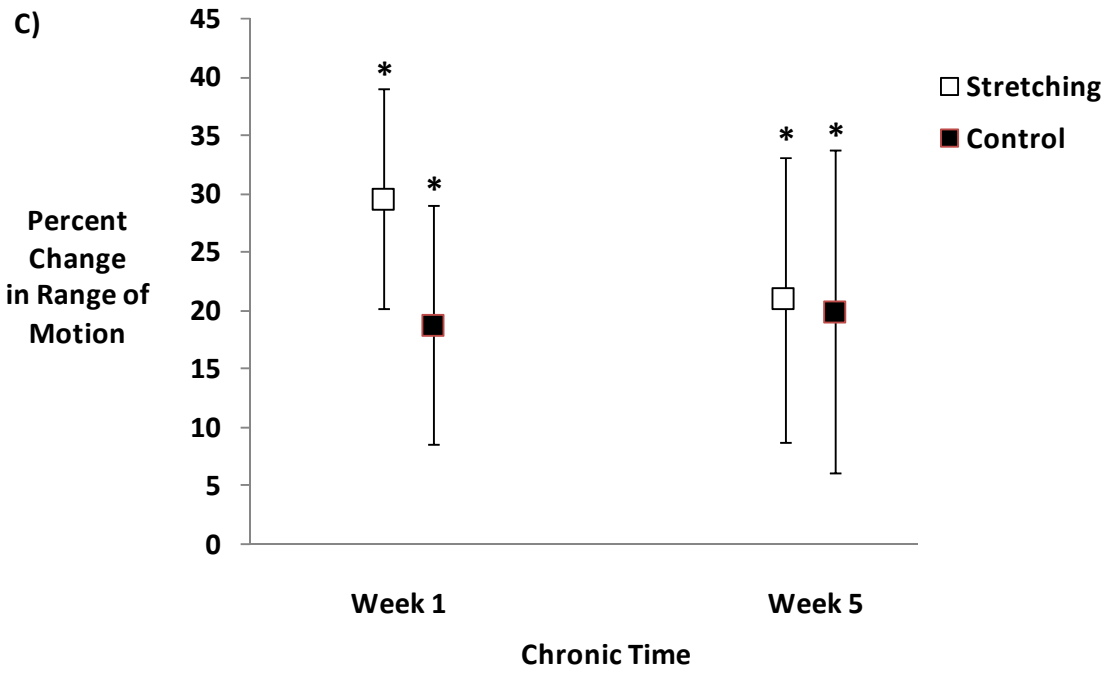
C)
45
40
35
30
25
20
15
10
5
0
Percent Change in Range of Motion
*
*
*
*
Stretching
Control
Week 1
Week 5
Chronic Time

Figure 10.

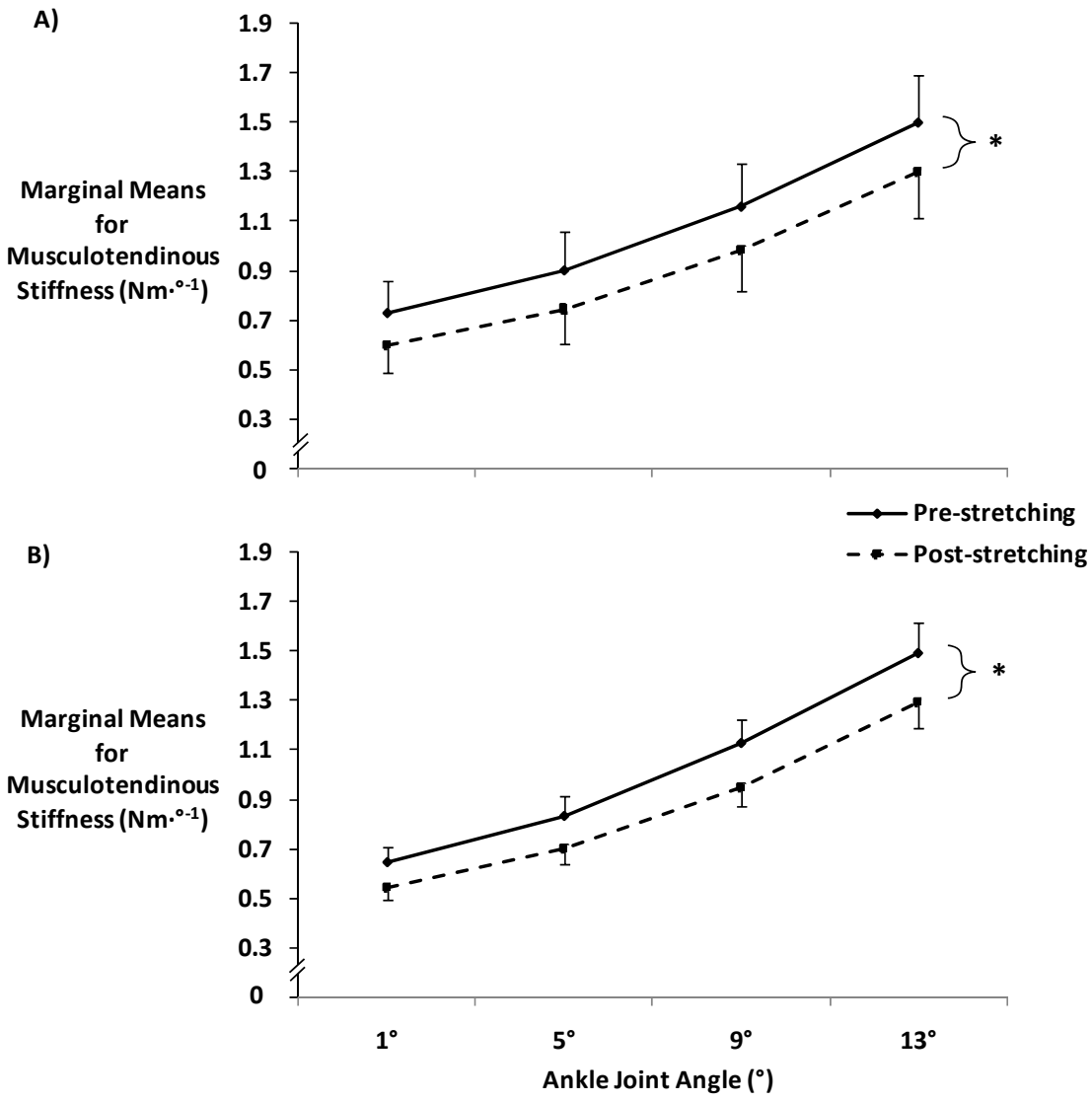
A)
1.9
1.7
1.5
1.3
1.1
0.9
0.7
0.5
0.3
0
Marginal Means for Musculotendinous Stiffness (Nm·°-1)
*
Pre-stretching
Post-stretching
B)
1.9
1.7
1.5
1.3
1.1
0.9
0.7
0.5
0.3
0
Marginal Means for Musculotendinous Stiffness (Nm·°-1)
*
1°
5°
9°
13°
Ankle Joint Angle (°)

Figure 10.

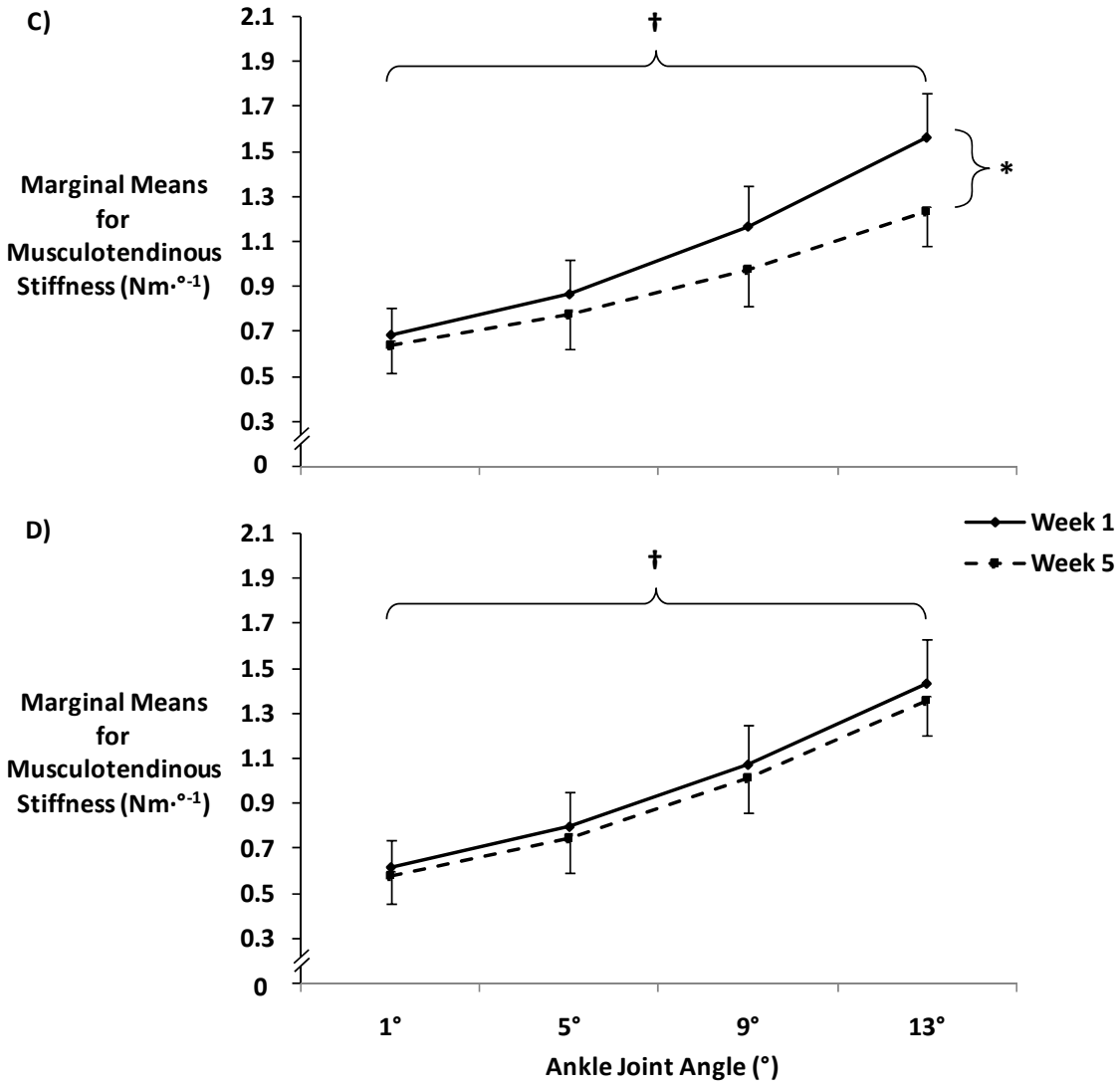
C)
D)
Marginal Means for Musculotendinous Stiffness (Nm·°-1)
2.1
1.9
1.7
1.5
1.3
1.1
0.9
0.7
0.5
0.3
0
†
*
Week 1
Week 5
1°
5°
9°
13°
Ankle Joint Angle (°)

Figure 10.

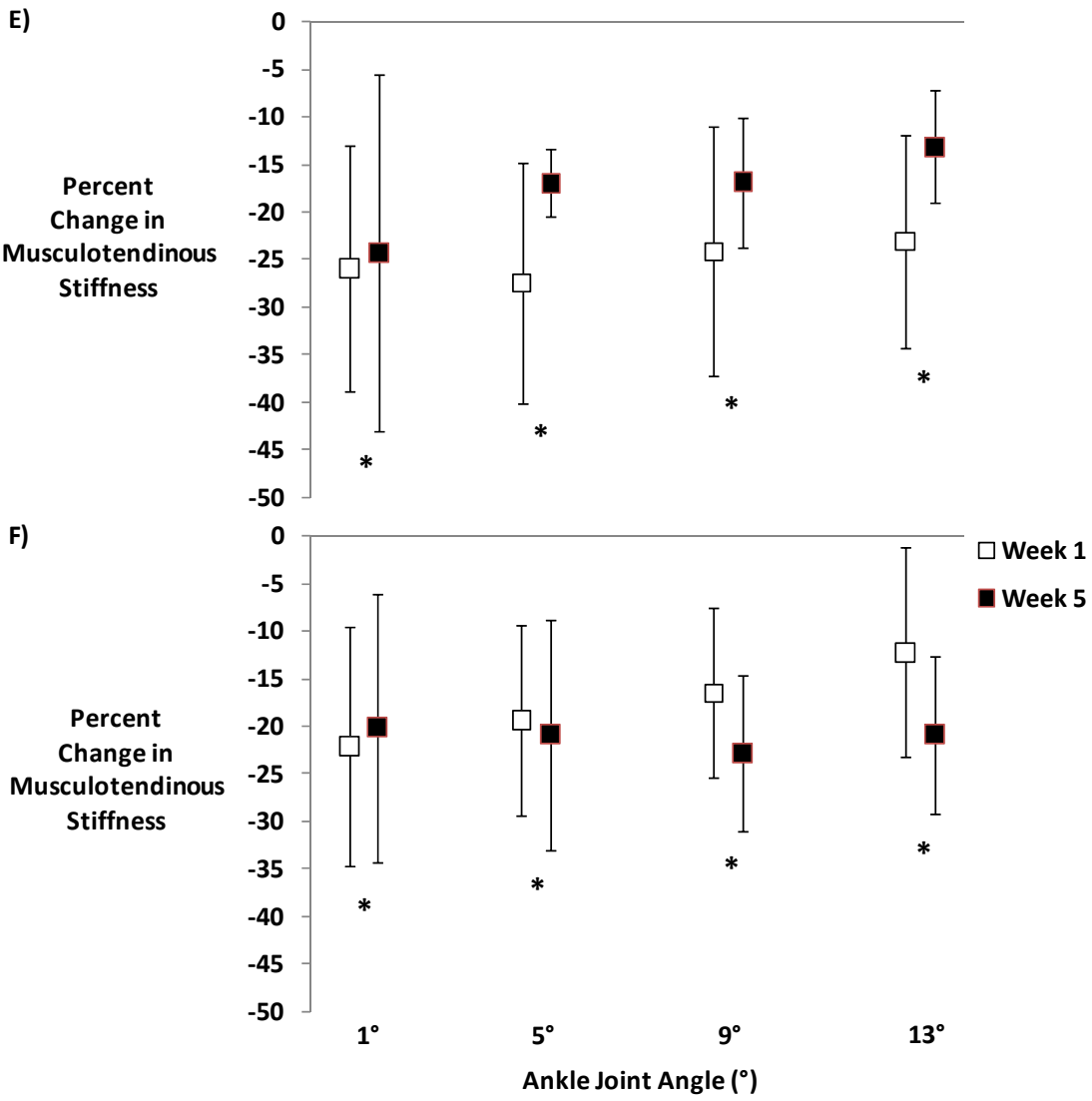
E)
F)
Percent Change in Musculotendinous Stiffness
Percent Change in Musculotendinous Stiffness
0
-5
-10
-15
-20
-25
-30
-35
-40
-45
-50
*
Week 1
Week 5
1°
5°
9°
13°
Ankle Joint Angle (°)

Table 1. Participant demographics by group (mean ± SD).

	n	Age (yrs)	Height (cm)	Mass (kg)	Corrected Calf Girth (cm)	MVC at -19° (N)	MVC at -9° (N)	MVC at 1° (N)	MVC at 12° (N)	ROM (degrees)
STR	15	22 ± 2	175 ± 8	74 ± 12	34 ± 2	273 ± 78	338 ± 95	359 ± 97	328 ± 99	20 ± 9
CON	11	21 ± 2	176 ± 7	76 ± 11	35 ± 2	320 ± 92	391 ± 118	401 ± 125	372 ± 141	18 ± 9

There were no differences ($P > 0.05$) between groups for any of the variables listed.

Appendix C.

FIGURE LEGENDS

Figure 1. Study design flow chart.

Figure 2. Experimental setup.

A) An example of the subjects positioning in the modified McComas Boot attached to the Biodex isokinetic dynaomometer.

B) Picture of foot placement in the modified McComas Boot.

Figure 3. Marginal means for isometric maximal voluntary contraction (MVC) force for (A) pre- to post-stretching [collapsed across group, chronic, and angle], (B) week 1 to week 5 for the stretching (open) and control (shaded) groups [collapsed across acute and angle], and (C) -19°, -9°, 1°, and 12° joint angles [collapsed across group, chronic, and acute]. Mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (D) stretching and (E) control groups.

(A) * indicates a significant decrease from pre- to post-stretching ($P<0.05$). Values are marginal means ± SEM.

(B) * indicates a significant increase from week 1 to week 5 for the STR group only ($P<0.05$). Values are marginal means ± SEM.

(C) * indicates -19° values are significantly less than 9°, 1°, and 12° values ($P<0.05$). † indicates 1° values are significantly greater ($P<0.05$) than 9° and 12°. Values are marginal means ± SEM.

(D) * indicates the percent change for MVC was significantly less than zero ($P<0.05$). Values are means ± 95% confidence intervals.

(E) * indicates the percent change for MVC was significantly less than zero ($P<0.05$). Values are means ± 95% confidence intervals.

Figure 4. Marginal means for peak single twitch force for (A) pre- to post-stretching [collapsed across group and angle] at week 1 and 5 and (B) -19°, -9°, 1°, and 12° joint angles [collapsed across acute and group] at week 1 and 5. Mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (C) stretching and (D) control group.

(A) * indicates week 5 pre-stretching values are significantly greater than week 5 post-stretching values and week 1 pre- and post-stretching values ($P<0.05$). † indicates week 5 post-stretching values are significantly greater than week 1 post-stretching values ($P<0.05$). Values are marginal means ± SEM.

(B) * indicates -19° values are significantly less than -9°, 1°, and 12° values at both week 1 and 5 ($P<0.05$). † indicates -9° values are significantly less than 1° and 12° values at both week 1 and 5 ($P<0.05$). ** indicates week 5 values are significantly greater than week 1 values at all joint angles ($P<0.05$). Values are marginal means ± SEM.

(C) * indicates the percent change for single twitch force was significantly less than zero ($P<0.05$). Values are means ± 95% confidence intervals.

(D) * indicates the percent change for single twitch force was significantly less than zero ($P<0.05$). Values are means ± 95% confidence intervals.

Figure 5. Marginal means for peak tetanic twitch force for (A) pre- to post-stretching [collapsed across chronic, angle, and group] and (B) -19°, -9°, 1°, and 12° joint angles [collapsed across acute and group] at week 1 and 5. Mean percent change scores from

pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (C) stretching and (D) control group.

(A) * indicates a significant decrease from pre- to post-stretching ($P<0.05$). Values are marginal means ± SEM.

(B) * indicates -19° values are significantly less than -9°, 1°, and 12° values at week 1 ($P<0.05$). † indicates 1° values are significantly greater than 12° values at week 1 ($P<0.05$). ** -19° values are significantly less than -9°, 1°, and 12° values at week 5 ($P<0.05$). †† indicates 1° values are significantly greater than-9° and 12° values at week 5 ($P<0.05$). $^{\beta}$ indicates week 5 values are significantly greater than week 1 values at all joint angles ($P<0.05$). Values are marginal means ± SEM.

(E) The percent change for tetanic twitch force is not different from zero ($P>0.05$). Values are means ± 95% confidence intervals.

(F) * indicates the percent change for tetanic twitch force was significantly less than zero ($P<0.05$). Values are means ± 95% confidence intervals.

Figure 6. Marginal means for percent voluntary activation for (A) -19°, -9°, 1°, and 12° joint angles [collapsed across acute, chronic, and group]. Mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (B) stretching and (C) control group.

(A) * indicates 12° values are significantly less than -19° -9° and 1° values ($P<0.05$). † indicates 1° values are significantly less than -19° values ($P<0.05$). Values are marginal means ± SEM.

(B) The percent change for percent voluntary activation is not different from zero (P>0.05). Values are means ± 95% confidence intervals.

(C) The percent change for percent voluntary activation is not different from zero (P>0.05). Values are means ± 95% confidence intervals.

Figure 7. Soleus electromyographic amplitude mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (A) stretching and (B) control group.

(A) The percent change for percent voluntary activation is not different from zero (P>0.05). Values are means ± 95% confidence intervals.

(B) * indicates the percent change for soleus electromyographic amplitude is significantly greater than zero (P<0.05). Values are means ± 95% confidence intervals.

Figure 8. Marginal means for medial gastrocnemius electromyographic amplitude for (A) week 1 to week 5 [collapsed across acute, angle, and group] and (B) -19°, -9°, 1°, and 12° joint angles [collapsed across acute, chronic, and group]. Mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (C) stretching and (D) control group.

(A) * indicates week 5 values are significantly greater than week 1 values (P<0.05). Values are marginal means ± SEM.

(B) * indicates -19° values are significantly greater than -9°, 1°, and 12° values (P<0.05). Values are marginal means ± SEM.

(C) The percent change for medial gastrocnemius electromyographic amplitude is not different from zero (P>0.05). Values are means ± 95% confidence intervals.

(D) The percent change for medial gastrocnemius electromyographic amplitude is not different from zero (P>0.05). Values are means ± 95% confidence intervals.

Figure 9. Marginal means in range of motion for (A) pre- to post-stretching [collapsed across chronic and group] and (B) week 1 to week 5 for both the stretching (open) and control (shaded) groups [collapsed across acute]. Mean percent change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (C) stretching and (D) control group.

(A) * indicates post-stretching values are significantly greater than pre-stretching values (P<0.05). Values are marginal means ± SEM.

(B) * indicates week 5 values are significantly greater than week 1 for the stretching group only (P<0.05). Values are marginal means ± SEM.

(C) * indicates the percent change for range of motion is significantly greater than zero (P<0.05). Values are means ± 95% confidence intervals.

(D) * indicates the percent change for range of motion is significantly greater than zero (P<0.05). Values are means ± 95% confidence intervals.

Figure 10. Marginal means for musculotendinous stiffness across 1°, 5°, 9°, and 13° joint angles for the (A) stretching and (B) control group from pre- (solid) to post- (dashed) stretching [collapsed across chronic] and for the (C) stretching and (D) control group from week 1(solid) to week 5 (dashed) [collapsed across acute]. Mean percent

change scores from pre- to post-stretching with 95% confidence intervals at week 1 (open) and week 5 (shaded) for the (E) stretching and (F) control group.

(A) * indicates pre- values are significantly greater than post-stretching values across all joint angles ($P<0.05$). Values are marginal means ± SEM.

(B) * indicates pre- values are significantly greater than post-stretching values across all joint angles ($P<0.05$). Values are marginal means ± SEM.

(C) * indicates week 1 values are significantly greater than week 5 values across all joint angles ($P<0.05$). † indicates values increase across joint angles ($1° < 5° < 9° < 13°$). Values are marginal means ± SEM.

(D) There are no differences from week 1 to week 5 across all joint angles ($P>0.05$). † indicates values increase across joint angles ($1° < 5° < 9° < 13°$). Values are marginal means ± SEM.

(E) * indicates the percent change for musculotendinous stiffness is significantly less than zero at both week 1 and 5 ($P<0.05$). Values are means ± 95% confidence intervals.

(F) * indicates the percent change for musculotendinous stiffness is significantly less than zero at both week 1 and 5 ($P<0.05$). Values are means ± 95% confidence intervals.

Appendix D.

University of Oklahoma
Institutional Review Board
Informed Consent to Participate in a Research Study

Project Title:	The acute and chronic effects of passive stretching on neuromuscular function and flexibility in the plantar flexor muscles.
Principal Investigator:	Eric D. Ryan, M.S.
Department:	Health and Exercise Science

You are being asked to volunteer for this research study. This study is being conducted at Biophysics Laboratory in the Department of Health and Exercise Science. You were selected as a possible participant because you are a healthy man who is able to exercise between the age of 18-30.

Please read this form and ask any questions that you may have before agreeing to take part in this study.

Purpose of the Research Study

The purpose of this study is: to examine the acute and chronic effects of passive stretching of the plantar flexor (calf) muscles on muscle function and flexibility.

Number of Participants

About 40 people will take part in this study, with 20 participants randomly assigned to a stretching group and 20 participants assigned to a non-stretching control group.

Procedures

If you agree to be in this study, you will be asked to do the following:

- Fill out a Pre-exercise testing health & exercise status questionnaire, which may determine your ability to participate in this study.
- Sign and date the Authorization to Use or Disclose Protected Health Information for Research form regarding your private health information.
- Sign and date an Informed Consent document, indicating that you understand all procedures and your rights as a research participant.

- Set a schedule for 3 laboratory visits on 3 separate days around the same time of day. The second visit will occur 2-5 days after the first visit and the third visit will occur 4 weeks after the second visit.

- After the second visit you will be randomly assigned to a stretching or non-stretching group for the following 4 weeks.

- In the stretching group, you will visit the laboratory 3 times a week and stretch your calf muscles.

- Wear sensors that will be taped to your skin over areas that have been shaved and cleaned with alcohol during the second and third laboratory visit.

- Allow your foot to be passively moved slowly through a range of motion from a position of your toes pointing forward to a position of your toes pointing backwards.

- Perform maximal isometric (no movement) muscle contractions of the lower leg. During the maximal effort muscle contraction, electrical stimulation will be applied to the lower leg.

- Electrical stimulation will also be applied to the leg at rest prior to each maximal muscle contraction.

Length of Participation

Each visit will take around 1-1:30 hours, for a total of 3-4.5 hours for each laboratory visit. If assigned to the stretching group, you will visit the laboratory 3 times a week for 4 weeks, with each session lasting approximately 10 min (30 min total each week).

This study has the following risks:

Possible risks include muscle soreness, skin abrasions due to shaving and cleansing the skin with alcohol, and temporary blood pressure elevation due to muscle contractions. Medical records will only be used during the screening process.

Benefits of being in the study are

The benefits to participation are helping to determine how acute and chronic stretching influence muscle strength and flexibility.

Injury

In case of injury or illness resulting from this study, emergency medical treatment is available. However, you or your insurance company may be expected to pay the usual charge from this treatment. The University of Oklahoma Norman Campus has set aside no funds to compensate you in the event of injury.

Confidentiality

In published reports, there will be no information included that will make it possible to identify you without your permission. Research records will be stored securely and only approved researchers will have access to the records.

There are organizations that may inspect and/or copy your research records for quality assurance and data analysis, which include the OU Institutional Review Board.

Compensation

You will be reimbursed for you time and participation in this study. Participants who complete the study will receive a stipend in the amount of $200 if you are randomly assigned to the stretch training group or $100 if you are randomly assigned to the control group. Participants will receive the full stipend upon the completion of the study; otherwise a prorated amount will be awarded.

Voluntary Nature of the Study

Participation in this study is voluntary. If you withdraw or decline participation, you will not be penalized or lose benefits or services unrelated to the study. If you decide to participate, you may decline to answer any question and may choose to withdraw at any time.

Contacts and Questions

If you have concerns or complaints about the research, the researcher(s) conducting this study can be contacted at 405-325-5211 or via email: Eric Ryan, eryan@ou.edu and Dr. Joel Cramer, jcramer@ou.edu.
Contact the researcher(s) if you have questions or if you have experienced a research-related injury.

If you have any questions about your rights as a research participant, concerns, or complaints about the research and wish to talk to someone other than individuals on the research team or if you cannot reach the research team, you may contact the University of Oklahoma – Norman Campus Institutional Review Board (OU-NC IRB) at 405-325-8110 or irb@ou.edu.

You will be given a copy of this information to keep for your records. If you are not given a copy of this consent form, please request one.

Statement of Consent

I have read the above information. I have asked questions and have received satisfactory answers. I consent to participate in the study.

Signature Date

Appendix E.

PRE-EXERCISE TESTING HEALTH & EXERCISE STATUS QUESTIONNAIRE

The University of Oklahoma
DEPARTMENT OF HEALTH AND EXERCISE SCIENCE

Name _____ Date_____

Home Address _____
We will send your payment for completing this study to this address (print neatly).

Work Phone _____ Home Phone _____

Person to contact in case of emergency _____

Emergency Contact Phone _____ Birthday mm/dd/yy)____/_____/_____

Student Identification #:_____ Social Security #:_____
NOTE: These numbers will only be used to process your payment for completing this study. They will be held strictly confidential and will be kept in a locked cabinet only accessible to the investigators.

Gender_____ Age _____(yrs) Height _____(ft)_____(in)
Weight_____(lbs)

Blood Pressure_____/_____ Heart Rate _____ (bpm)
Does the above weight indicate: a gain____ a loss____ no change____ in the past year?
If a change, how many pounds?_____(lbs)

A. JOINT-MUSCLE STATUS (✓Check areas where you currently have problems)

Joint Areas
( ) Wrists
( ) Elbows
( ) Shoulders
( ) Upper Spine & Neck
( ) Lower Spine
( ) Hips
( ) Knees
( ) Ankles
( ) Feet
( ) Other_____

Muscle Areas
( ) Arms
( ) Shoulders
( ) Chest
( ) Upper Back & Neck
( ) Abdominal Regions
( ) Lower Back
( ) Buttocks
( ) Thighs
( ) Lower Leg
( ) Feet
( ) Other_____

B. HEALTH STATUS (✓Check if you currently have any of the following conditions)

( ) High Blood Pressure
( ) Heart Disease or Dysfunction
( ) Peripheral Circulatory Disorder
( ) Lung Disease or Dysfunction
( ) Arthritis or Gout
( ) Edema
( ) Epilepsy
( ) Multiple Sclerosis
( ) High Blood Cholesterol or Triglyceride Levels
( ) Allergic reactions to rubbing alcohol
( ) Acute Infection
( ) Diabetes/Blood Sugar Level Abnormality
( ) Anemia
( ) Hernias
( ) Thyroid Dysfunction
( ) Pancreas Dysfunction
( ) Liver Dysfunction
( ) Kidney Dysfunction
( ) Phenylketonuria (PKU)
( ) Loss of Consciousness

* *NOTE: If any of these conditions are checked, then a physician's health clearance will required.*C. PHYSICAL EXAMINATION HISTORY

Approximate date of your last physical examination_____

Physical problems noted at that time_____

Has a physician ever made any recommendations relative to limiting your level of physical exertion? _____YES _____NO
If YES, what limitations were recommended?_____
_____

D. CURRENT MEDICATION USAGE (List the drug name and the condition being managed)

MEDICATION	***CONDITION***
_____	_____
_____	_____
_____	_____

E. PHYSICAL PERCEPTIONS (Indicate any unusual sensations or perceptions. ✓Check if you have recently experienced any of the following during or soon after *physical activity* (PA); or during *sedentary periods* (SED))

PA	SED		PA	SED	
( )	( )	Chest Pain	( )	( )	Nausea
( )	( )	Heart Palpitations	( )	( )	Light Headedness
( )	( )	Unusually Rapid Breathing	( )	( )	Loss of Consciousness
( )	( )	Overheating	( )	( )	Loss of Balance
( )	( )	Muscle Cramping	( )	( )	Loss of Coordination
( )	( )	Muscle Pain	( )	( )	Extreme Weakness
( )	( )	Joint Pain	( )	( )	Numbness
( )	( )	Other_____	( )	( )	Mental Confusion

F. FAMILY HISTORY (✓Check if any of your blood relatives . . . parents, brothers, sisters, aunts, uncles, and/or grandparents . . . have or had any of the following)

( ) Heart Disease
( ) Heart Attacks or Strokes (prior to age 50)
( ) Elevated Blood Cholesterol or Triglyceride Levels
( ) High Blood Pressure
( ) Diabetes
( ) Sudden Death (other than accidental)

G. EXERCISE STATUS

Do you regularly engage in aerobic forms of exercise (i.e., jogging, cycling, walking, etc.)?

YES NO

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours

Do you regularly lift weights?

YES NO

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours

Do you regularly play recreational sports (i.e., basketball, racquetball, volleyball, etc.)?

YES NO

How long have you engaged in this form of exercise? _____ years _____ months

How many hours per week do you spend for this type of exercise? _____ hours