Journal of Sports Sciences, April 2007; 25(6): 687 – 698

Routledge
Taylor & Francis Group

Acute effects of static stretching on characteristics of the isokinetic angle – torque relationship, surface electromyography, and mechanomyography

JOEL T. CRAMER[1], TRAVIS W. BECK[2], TERRY J. HOUSH[2], LAURIE L. MASSEY[3], SARAH M. MAREK[3], SUZANNE DANGLEMEIER[3], SUSHMITA PURKAYASTHA[3], JULIE Y. CULBERTSON[4], KRISTI A. FITZ[3], & ALISON D. EGAN[1]

[1]*Department of Health and Exercise Science, University of Oklahoma, Norman, OK,* [2]*Department of Nutrition and Health Sciences, University of Nebraska-Lincoln, Lincoln, NE,* [3]*Department of Kinesiology, University of Texas at Arlington, Arlington, TX and* [4]*Baylor Institute for Rehabilitation, Baylor University Medical Center, Dallas, TX, USA*

(Accepted 28 April 2006)

Abstract
The aims of this study were to examine the acute effects of static stretching on peak torque, work, the joint angle at peak torque, acceleration time, isokinetic range of motion, mechanomyographic amplitude, and electromyographic amplitude of the rectus femoris during maximal concentric isokinetic leg extensions at 1.04 and 5.23 rad $\cdot$ s^{-1} in men and women. Ten women (mean $\pm$ *s*: age 23.0 $\pm$ 2.9 years, stature 1.61 $\pm$ 0.12 m, mass 63.3 $\pm$ 9.9 kg) and eight men (age 21.4 $\pm$ 3.0 years, stature 1.83 $\pm$ 0.11 m, mass 83.1 $\pm$ 15.2 kg) performed maximal voluntary concentric isokinetic leg extensions at 1.04 and 5.23 rad $\cdot$ s^{-1}. Following the initial isokinetic tests, the dominant leg extensors were stretched using four static stretching exercises. After the stretching, the isokinetic tests were repeated. Peak torque, acceleration time, and electromyographic amplitude decreased ($P \leq 0.05$) from pre- to post-stretching at 1.04 and 5.23 rad $\cdot$ s^{-1}; there were no changes ($P > 0.05$) in work, joint angle at peak torque, isokinetic range of motion, or mechanomyographic amplitude. These findings indicate no stretching-related changes in the area under the angle – torque curve (work), but a significant decrease in peak torque, which suggests that static stretching may cause a ''flattening'' of the angle – torque curve that reduces peak strength but allows for greater force production at other joint angles. These findings, in conjunction with the increased limb acceleration rates (decreased acceleration time) observed in the present study, provide tentative support for the hypothesis that static stretching alters the angle – torque relationship and/or sarcomere shortening velocity.

Keywords: *Electromyography, mechanomyography, peak torque, joint angle at peak torque, work, acceleration*

Introduction

Static stretching is often performed before exercise (deVries, 1963; Franklin, Whaley, Howley, & Balady, 2000) and athletic events (Beaulieu, 1981; Holcomb, 2000) in the belief that increasing flexibility (increasing joint range of motion) will enhance performance (Shellock & Prentice, 1985; Smith, 1994) and reduce the risk of injury (Bixler & Jones, 1992; Ekstrand, Gillquist, & Liljedahl, 1983a; Ekstrand, Gillquist, Moller, Oberg, & Liljedahl, 1983b; Garrett, 1990; Safran, Seaber, & Garrett, 1989). Recent systematic reviews (Shrier, 1999; Thacker, Gilchrist, Stroup, & Kimsey, 2004) and original studies (Behm, Button, & Butt, 2001; Cornwell, Nelson, & Sidaway, 2002; Cramer *et al.*, 2004a, 2005; Evetovich, Nauman, Conley, & Todd, 2003; Fowles, Sale, & MacDougall, 2000; Kokkonen, Nelson, & Cornwell, 1998; McNeal & Sands, 2003; Nelson, Allen, Cornwell, & Kokkonen, 2001a; Nelson, Guillory, Cornwell, & Kokkonen, 2001b; Nelson & Kokkonen, 2001; Power, Behm, Cahill, Carroll, & Young, 2004; Young & Behm, 2003; Young & Elliott, 2001), however, have suggested that pre-exercise stretching may temporarily compromise a muscle's ability to produce maximal force. This ''stretching-induced force deficit'' has been reported to affect isometric force production (Avela, Kyrolainen, & Komi, 1999; Behm *et al.*, 2001; Fowles *et al.*, 2000; Nelson *et al.*, 2001a; Power *et al.*, 2004), concentric isokinetic peak torque (Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003;

Correspondence: J. T. Cramer, Department of Health and Exercise Science, University of Oklahoma, 1401 Asp Avenue, Room 110, Norman, OK 73019-6081, USA. E-mail: jcramer@ou.edu

ISSN 0264-0414 print/ISSN 1466-447X online © 2007 Taylor & Francis
DOI: 10.1080/02640410600818416

Nelson *et al.*, 2001b), dynamic constant external resistance (DCER) force (Fry, McLellan, Weiss, & Rosato, 2003; Kokkonen *et al.*, 1998; Nelson & Kokkonen, 2001), vertical jumping performance (Church, Wiggins, Moode, & Crist, 2001; Cornwell *et al.*, 2002; McNeal and Sands, 2003; Young & Behm, 2003; Young and Elliott, 2001), and balance (Behm, Bambury, Cahill, & Power, 2004). Knudson, Bennett, Corn, Leick and Smith (2001), however, reported no kinetic or kinematic alterations in the vertical jump after static stretching.

Two main hypotheses have been proposed to explain the stretching-induced force deficit: (a) mechanical factors, such as decreases in musculotendonous stiffness that may affect the muscle's length–tension relationship and/or sarcomere shortening velocity (Cornwell *et al.*, 2002; Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003; Fowles *et al.*, 2000; Kokkonen *et al.*, 1998; Nelson *et al.*, 2001a, 2001b; Nelson & Kokkonen, 2001), and (b) neural factors, such as decreases in motor neuron pool excitability that may reduce peripheral muscle activation (Avela *et al.*, 1999; Behm *et al.*, 2001, 2004; Cramer *et al.*, 2004a, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004). Fowles *et al.* (2000) suggested that stretching-induced decreases in neural drive could only account for a percentage of the force deficit, and thus mechanical as well as neural factors may contribute to the stretching-induced force deficit. Indeed, preliminary evidence reported by Fowles *et al.* (2000) indicated that their stretching protocol increased fascicle length in at least one participant as measured by B-mode ultrasound. Nelson *et al.* (2001b) hypothesized that if there were stretching-induced increases in resting fascicle or sarcomere lengths, this would require the sarcomeres to shorten a greater distance in the same amount of time, which would increase sarcomere shortening velocity. At least during an isokinetic muscle action where the range of motion and velocity remain constant, an increase in sarcomere shortening velocity may decrease the number of sarcomeres that are capable of contributing to force production due to the force–velocity relationship. Since the angle–torque relationships produced during isokinetic (Brockett, Morgan, & Proske, 2001) and isometric (McHugh & Hogan, 2004; McHugh & Tetro, 2003) muscle actions have been used to globally examine the length–tension relationship for sarcomeres, the values that represent the isokinetic angle–torque relationship could provide indirect information about the mechanical factors underlying the stretching-induced force deficit.

It has also been hypothesized that stretching-induced decreases in force production are due to neural factors such as decreased motor unit activation, reduced firing frequency, and/or altered reflex sensitivity (Avela *et al.*, 1999; Behm *et al.*, 2001, 2004; Cramer *et al.*, 2004a, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004). Previous studies have demonstrated stretching-induced decreases in muscle activation through the use of surface (Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004) and fine-wire (Avela *et al.*, 1999) electromyography (EMG) in addition to twitch interpolation techniques (Behm *et al.*, 2001; Fowles *et al.*, 2000; Power *et al.*, 2004). Decreases in motor unit recruitment (EMG amplitude) and firing frequency (zero crossing rate) were observed after repeated passive stretches of the plantar flexors (Avela *et al.*, 1999). In addition, Fowles *et al.* (2000) reported that 60% of the stretching-induced decreases in force production of the triceps surae (up to 15 min after stretching) were due to neural factors. Behm *et al.* (2001) suggested that at least part of the stretching-induced decreases in maximal force production of the leg extensors was a result of decreases in muscle activation. There is also evidence to suggest that static stretching may affect the central nervous system. Avela *et al.* (1999) reported decreases in the Hoffman reflex amplitude, which is often used as a measure of motor neuron pool excitability (Enoka, 2002), recorded from the gastrocnemius and soleus muscles following a bout of passive stretching. Furthermore, Cramer *et al.* (2005) reported stretching-induced decreases in peak torque and surface EMG amplitude in both the stretched and unstretched (contralateral) leg extensor muscles and suggested that the decreases in force production and muscle activation that occur in response to static stretching may be due, in part, to an unidentified central nervous system inhibitory mechanism.

In addition to EMG, recent studies (Cramer *et al.*, 2005; Evetovich *et al.*, 2003) have also used mechanomyography (MMG) to examine the acute effects of static stretching on muscle force production. The MMG signal is generated by the low-frequency lateral oscillations of active skeletal muscle fibres (Orizio, 1993; Orizio, Gobbo, Diemont, Esposito, & Veicsteinas, 2003; Stokes, 1993; Stokes & Blythe, 2001), and it has been suggested that MMG reflects the mechanical counterpart of motor unit electrical activity, as measured by EMG (Gordon & Holbourn, 1948). The surface EMG signal, however, is a linear summation of the motor unit action potential trains that initiate muscle mechanical activity (Basmajian & De Luca, 1985). Together, the simultaneous measurements of MMG and EMG have been used to examine muscle function during isometric (Beck *et al.*, 2004a; Coburn *et al.*, 2004; Ebersole *et al.*, 1999) and isokinetic muscle actions (Beck *et al.*, 2004a, 2004b; Coburn *et al.*, 2004; Cramer *et al.*, 2000a, 2000b,

2002a, 2000b, 2000c, 2004b; Evetovich *et al.*, 1997, 1998), as well as maximal and submaximal cycle ergometry (Housh *et al.*, 2000; Perry, Housh, Johnson, Ebersole, & Bull, 2001a; Perry *et al.*, 2001b; Shinohara, Kouzaki, Yoshihisa, & Fukunaga, 1997; Stout, Housh, Johnson, Evetovich, & Smith, 1997). From these studies, it has been hypothesized that MMG amplitude may reflect muscle stiffness (Cramer *et al.*, 2000a, 2000b, 2002a, 2002b, 2002c, 2004b; Evetovich *et al.*, 1997), since a stiffer muscle would theoretically oscillate less than a more compliant muscle. By including measures of MMG amplitude in the present study, it may be possible to test this hypothesis, since stretching may decrease muscle stiffness, which could result in an increase in MMG amplitude.

In addition to peak torque, MMG amplitude, and EMG amplitude, it is possible that static stretching alters the joint angle at peak torque (Cramer *et al.*, 2004b; Fowles *et al.*, 2000; Nelson *et al.*, 2001a) and the rate of sarcomere shortening (Nelson *et al.*, 2001b), which could alter the isokinetic acceleration phase (time from initiation of velocity production to initiation of a constant angular velocity) (Brown, 2000; Brown and Whitehurst, 2003; Brown, Whitehurst, Gilbert, & Buchalter, 1995). Furthermore, the stretching-induced decrease in peak torque, with no change in mean power output reported by Cramer *et al.* (2005), suggested that stretching may reduce peak torque, but may not affect the area under the angle–torque curve, which is mathematically defined as force × distance, or work. Barring any changes in the joint range of motion, it is possible that if static stretching influences the angle–torque curve, then peak torque and work could respond differently. No previous studies, however, have examined the effects of static stretching on the factors that influence the angle–torque relationship, such as peak torque, work, joint angle at peak torque, acceleration time, and range of motion. The simultaneous examination of these parameters could provide indirect information about the potential contributions of mechanical and neural mechanisms to stretching-induced decreases in force production (Cramer *et al.*, 2005; Evetovich *et al.*, 2003). Therefore, the aims of this study were to examine the acute effects of static stretching on neuromuscular function (peak torque, work, joint angle at peak torque, acceleration time, range of motion, EMG amplitude, and MMG amplitude) during maximal concentric isokinetic leg extensions at 1.04 and 5.23 rad $\cdot$ s^{-1} in men and women.

Methods

Ten women (mean $\pm$ *s*: age 23.0 $\pm$ 2.9 years, stature 1.61 $\pm$ 0.12 m, mass 63.3 $\pm$ 9.9 kg) and eight men (age 21.4 $\pm$ 3.0 years, stature 1.83 $\pm$ 0.11 m, mass 83.1 $\pm$ 15.2 kg) volunteered to participate. The participants were healthy, recreationally active (non-athletes that were currently exercising between 1 and 5 h a week), and indicated no current or recent (within the past 6 months) hip-, knee- or ankle-related injuries. This study was approved by the University Institutional Review Board for Human Subjects, and all participants signed informed consent forms before testing began.

Each participant completed a 5-min warm-up at 50 W on a stationary cycle ergometer before the initial isokinetic test. Before and after the static stretching protocol, maximal concentric isokinetic peak torque for extension of the dominant leg (based on kicking preference) was measured 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 participants were seated with restraining straps over the upper thighs, trunk, and non-involved thigh, and gravity corrections for limb mass were performed before each isokinetic assessment in accordance with the manufacturer's instructions (Biodex Pro Manual, Applications/Operations, Biodex Medical Systems, Inc., Shirley, NY). The input axis of the dynamometer was aligned with the axis of the knee. Leg extension range of motion was maximized for each participant individually by placing mechanical stops at the beginning and end of their full active range of motion. Three submaximal warm-up trials preceded three maximal muscle actions at each velocity, and a 2-min rest was allowed between tests at each velocity. The repetion resulting in the greatest amount of work was selected for analysis. Peak torque, work, the joint angle at peak torque, acceleration time, and leg extension range of motion were provided by the dynamometer software (Biodex System 3 Advantage Software, Biodex Medical Systems, Inc., Shirley, NY). For the selected repetition, peak torque and joint angle at peak torque were reported as the maximum torque value and corresponding joint angle, work was calculated as the area under the angle–torque curve (torque × distance), acceleration time (ms) as the duration of time from the initiation of concentric velocity production to the initiation of a constant angular velocity (Brown, 2000; Brown & Whitehurst, 2003; Brown *et al.*, 1995), and leg extension range of motion as the range (maximum – minimum) of joint angle values during the isokinetic leg extension exercises, where full leg extension was set at 0° relative to each participant. One previous study (Perrin, 1986) interpreted the test–retest reliability coefficients for similar isokinetic variables as being "high" and that "clinicians can assume good reliability of instrumentation for assessment of peak torque, TAE (torque acceleration energy), average power, and total work" (p. 321).

Each participant performed an unassisted stretching exercise followed by three assisted stretching exercises. For the unassisted stretching exercise, the participant stood upright with one hand against a wall for balance. He or she then flexed the dominant leg to a knee joint angle of 90° before the ankle of the flexed leg was grasped by the ipsilateral hand, and the foot was raised so that the heel of the dominant foot approached the buttocks. Following the unassisted stretching exercise, the remaining stretching exercises were completed with the assistance of the primary investigator. The first assisted stretching exercise was performed with the participant lying prone on a padded table with the legs fully extended. The dominant leg was flexed at the knee joint and slowly pressed down so that the participant's heel approached the buttocks. If the heel was able to contact the buttocks, the knee was gently lifted off the supporting surface, causing a slight hyperextension at the hip joint, to complete the stretch. To perform the second assisted stretching exercise, the participant stood with their back to a table and rested the dorsal surface of their dominant foot on the table by flexing the leg at the knee joint. From this position, the dominant leg extensors were stretched by gently pushing back on both the knee of the flexed leg and the corresponding shoulder. The final assisted stretching exercise began with the participant lying supine along the edge of the padded table with the dominant leg hanging off of the table. The dominant leg was flexed at the knee and the thigh was slightly hyperextended at the hip by gently pressing down on the knee. Immediately after the stretching exercises, the isokinetic test protocol was repeated. The mean time that elapsed from the end of stretching to the beginning of isokinetic testing was 11.2 ($s = 1.5$) min.

Each participant underwent four static stretching exercises designed to stretch the leg extensor muscles of the dominant limb, according to the procedures of Cramer *et al.* (2004a, 2005) and Nelson *et al.* (2001b). Four repetitions of each stretching exercise were held for 30 s at a point of mild discomfort, but not pain, as acknowledged by the participant. Between each stretching repetition, the leg was returned to a neutral position for a 20-s rest period. The mean time of each stretching period was 15.6 ($s = 2.1$) min.

Bipolar surface electrode (Moore Medical, Ag–AgCl) arrangements were placed along the longitudinal axis of the rectus femoris at 50% of the distance from the anterior superior iliac spine to the superior border of the patella with a mean inter-electrode distance of 4.4 ($s = 0.2$) cm. Electrodes were placed in accordance with the recommendation of Hermens *et al.* (1999) to avoid overlap with the innervation zone and reduce the risk of cross-talk between muscles. Inter-electrode impedance was kept below 2000 Ω by careful skin abrasion. The EMG signals were pre-amplified (gain = 1000×) using a differential amplifier (EMG100C, Biopac Systems Inc., Santa Barbara, CA) with a bandwidth of 1–5000 Hz.

The MMG signal was detected using an active miniature accelerometer (EGAS-FS, Entran, Inc., Fairfield, NJ) that was pre-amplified (gain = 200×) with an in-line amplifier (Orizio, Liberati, Locatelli, De Grandis, & Veicsteinas, 1996; Watakabe, Itoh, Mita, & Akataki, 1998). The accelerometer was placed over the rectus femoris between the active EMG electrodes and affixed to the skin's surface using 3M double-sided foam tape and microporous surgical tape to ensure consistent contact pressure.

The MMG and EMG signals were sampled at a frequency of 1 kHz, stored on a personal computer, and expressed as root mean square (rms) amplitude values by software (AcqKnowledge III, Biopac Systems, Santa Barbara, CA). The MMG and EMG signals were bandpass filtered (second-order Butterworth filter) at 10–500 Hz and 5–100 Hz, respectively. The MMG and EMG amplitude values were calculated for a time period that corresponded to the full range of motion for each participant (mean range of motion = 99.0°, $s = 9.8°$) beginning with the onset of the EMG signal. This allowed for comparisons from before (pre-) to after (post-) stretching and between velocities based on a standardized range of motion.

Seven separate three-way mixed factorial analyses of variance (time [pre- vs. post-stretching] × velocity [1.04 vs. 5.23 rad · s^{-1}] × sex [male vs. female]) were used to analyse the data for peak torque, work, joint angle at peak torque, acceleration time, range of motion, MMG amplitude, and EMG amplitude. When appropriate, follow-up analysis included lower-order analyses of variance and paired sample *t*-tests. An alpha level of $P \leq 0.05$ was considered statistically significant for all comparisons. SPSS version 11.5 (SPSS, Inc., Chicago, IL) was used for all statistical analyses.

Results

Table I shows the absolute mean values (± standard errors of the mean) for the isokinetic measurements, MMG, and EMG amplitude values before and after stretching.

Peak torque

The statistical analysis for peak torque indicated no three-way interaction (time × velocity × sex; $P > 0.05$), no two-way interactions for time × velocity ($P > 0.05$) or time × sex ($P > 0.05$), but a

Table I. Isokinetic measurements, MMG and EMG amplitude values for the pre- and post-stretching assessments (mean ± s_x).

			Pre-stretching		Post-stretching	
			1.04 rad · s^{-1}	5.23 rad · s^{-1}	1.04 rad · s^{-1}	5.23 rad · s^{-1}
Peak torque (N · m)	Men	mean	239.6	143.4	234.1	143.5
		s_x	20.7	17.5	23.1	16.8
	Women	mean	142.0	86.7	134.0	81.2
		s_x	8.3	6.1	8.0	5.7
Work (J)	Men	mean	270.2	163.8	261.4	159.0
		s_x	20.4	22.2	24.1	22.7
	Women	mean	163.2	112.4	159.7	107.0
		s_x	11.1	11.4	11.5	10.8
Joint angle at peak torque (°)	Men	mean	61.1	50.1	58.5	47.8
		s_x	2.1	7.3	3.2	6.5
	Women	mean	66.5	54.6	64.2	55.8
		s_x	2.9	6.1	3.1	6.6
Acceleration time (ms)	Men	mean	63.8	80.0	37.5	77.5
		s_x	15.3	17.4	6.5	16.3
	Women	mean	56.0	92.0	42.2	84.0
		s_x	12.8	11.2	6.5	10.3
Range of motion (°)	Men	mean	99.6	95.9	95.9	94.3
		s_x	3.0	3.4	2.9	3.8
	Women	mean	102.2	101.3	101.7	99.1
		s_x	2.8	3.6	2.7	3.8
MMG amplitude (mVrms)	Men	mean	45.6	109.9	51.0	93.3
		s_x	6.0	21.6	7.6	25.2
	Women	mean	54.3	127.7	54.0	114.0
		s_x	7.1	15.1	4.3	19.9
EMG amplitude (μVrms)	Men	mean	549.3	505.6	473.0	431.1
		s_x	76.5	72.7	40.5	66.5
	Women	mean	422.0	427.4	401.9	387.4
		s_x	57.8	36.7	54.3	23.6

significant interaction for velocity × sex ($P \leq 0.05$) and a significant main effect for time ($P \leq 0.05$). The marginal mean for peak torque (collapsed across velocity and sex) decreased ($P \leq 0.05$) from pre- to post-stretching (Figure 1a). In addition, the marginal means for peak torque (collapsed across time) decreased ($P \leq 0.05$) from 1.04 to 5.23 rad · s^{-1} for the men and women, but the absolute values were greater ($P \leq 0.05$) for the men than the women at 1.04 and 5.23 rad · s^{-1}.

Work

The statistical analysis for work indicated no three-way interaction (time × velocity × sex; $P > 0.05$), no two-way interactions for time × velocity ($P > 0.05$) or time × sex ($P > 0.05$), no main effect for time ($P > 0.05$), but a significant interaction for velocity × sex ($P \leq 0.05$). The marginal means for work (collapsed across time) decreased ($P \leq 0.05$) from 1.04 to 5.23 rad · s^{-1} for the men and women, but the absolute values were greater ($P \leq 0.05$) for the men than the women at 1.04 and 5.23 rad · s^{-1}. There were no changes ($P > 0.05$), however, in work from pre- to post-stretching.

Joint angle at peak torque

The statistical analysis for joint angle at peak torque indicated no three-way interaction (time × velocity × sex; $P > 0.05$), no two-way interactions for time × velocity ($P > 0.05$), time × sex ($P > 0.05$), or velocity × sex ($P > 0.05$), and no main effects for time ($P > 0.05$) or sex ($P > 0.05$). There was, however, a significant main effect for velocity ($P \leq 0.05$). The marginal mean for joint angle at peak torque (collapsed across time and sex) decreased ($P \leq 0.05$) from 1.04 to 5.23 rad · s^{-1}. There were no changes ($P > 0.05$) in joint angle at peak torque from pre- to post-stretching.

Acceleration time

The statistical analysis for acceleration time indicated no three-way interaction (time × velocity × sex; $P > 0.05$), no two-way interactions for time × velocity ($P > 0.05$), time × sex ($P > 0.05$), or velocity × sex ($P > 0.05$), and no main effect for sex ($P > 0.05$). There were, however, significant main effects for time ($P \leq 0.05$) and velocity ($P \leq 0.05$). The marginal mean for acceleration time (collapsed across velocity

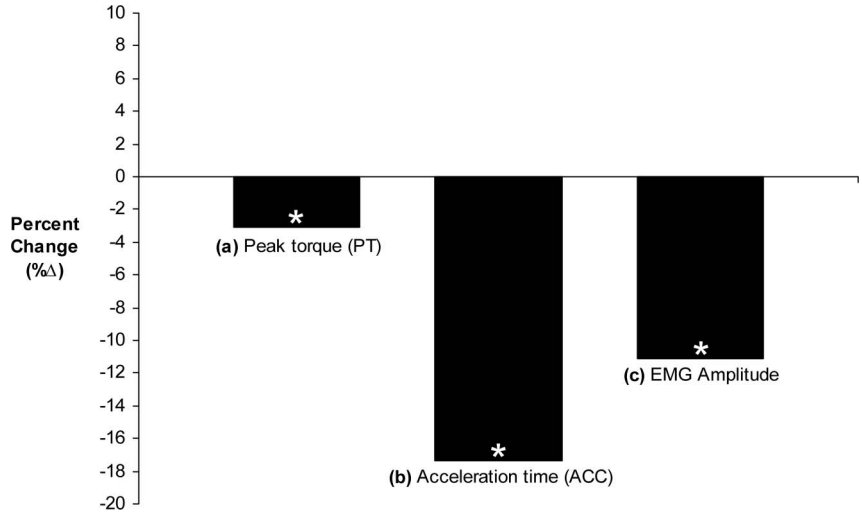

Figure 1. Percent changes (%Δ) from pre- to post-stretching for the marginal means (collapsed across velocity and sex) for (a) peak torque, (b) acceleration time, and (c) EMG amplitude. *Significant decrease ($P \leq 0.05$).

and sex) decreased ($P \leq 0.05$) from pre- to post-stretching (Figure 1b). The marginal mean for acceleration time (collapsed across time and sex) increased ($P \leq 0.05$) from 1.04 to 5.23 rad $\cdot$ s^{-1}.

Leg extension range of motion

The statistical analysis for leg extension range of motion indicated no three-way interaction (time $\times$ velocity $\times$ sex; $P > 0.05$), no two-way interactions for time $\times$ velocity ($P > 0.05$), time $\times$ sex ($P > 0.05$), or velocity $\times$ sex ($P > 0.05$), and no main effects for time ($P > 0.05$), velocity ($P > 0.05$), or sex ($P > 0.05$). Therefore, there were no changes ($P > 0.05$) in leg extension range of motion from pre- to post-stretching.

Mechanomyographic amplitude

The statistical analysis for MMG amplitude indicated no three-way interaction (time $\times$ velocity $\times$ sex; $P > 0.05$), no two-way interactions for time $\times$ velocity ($P > 0.05$), time $\times$ sex ($P > 0.05$), or velocity $\times$ sex ($P > 0.05$), and no main effects for time ($P > 0.05$) or sex ($P > 0.05$). There was, however, a significant main effect for velocity ($P \leq 0.05$). The marginal mean for MMG amplitude (collapsed across time and sex) increased ($P \leq 0.05$) from 1.04 to 5.23 rad $\cdot$ s^{-1}. There were no changes ($P > 0.05$) in MMG amplitude from pre- to post-stretching.

Electromyographic amplitude

The statistical analysis for EMG amplitude indicated no three-way interaction (time $\times$ velocity $\times$ sex; $P > 0.05$), no two-way interactions for time $\times$ velocity ($P > 0.05$), time $\times$ sex ($P > 0.05$), or velocity $\times$ sex ($P > 0.05$), and no main effects for velocity ($P > 0.05$) or sex ($P > 0.05$). The marginal mean for EMG amplitude (collapsed across velocity and sex) decreased ($P \leq 0.05$) from pre- to post-stretching (Figure 1c).

Discussion

Several studies (Avela *et al.*, 1999; Behm *et al.*, 2001; Church *et al.*, 2001; Cornwell *et al.*, 2002; Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003; Fowles *et al.*, 2000; Kokkonen *et al.*, 1998; McNeal & Sands, 2003; Nelson *et al.*, 2001a, 2001b; Nelson & Kokkonen, 2001; Power *et al.*, 2004; Young & Behm, 2003; Young & Elliott, 2001) have reported decreases in the force generating capacity of a muscle or muscle group following a bout of static stretching. The results of the present study support these previous findings and indicate a 3.4% decrease in peak torque at 1.04 and 5.23 rad $\cdot$ s^{-1} (Figure 1a) as a result of the static stretching. In a recent study, Nelson *et al.* (2001b) suggested that stretching-induced decreases in isokinetic peak torque are velocity-specific. That is, the stretching affected peak torque at the slower angular velocities (1.04 and 5.23 rad $\cdot$ s^{-1}), but not at the faster velocities (2.62, 3.66, or 4.72 rad $\cdot$ s^{-1}) (Nelson *et al.*, 2001b). The present findings, as well as those of previous studies (Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003), however, indicated stretching-induced decreases in peak torque at both slow (1.04 rad $\cdot$ s^{-1}) and fast (5.23 rad $\cdot$ s^{-1}) angular velocities and suggested that the stretching-induced decreases in peak torque might not be velocity-specific.

Two main mechanisms have been postulated to explain the stretching-induced decreases in force

production: (a) mechanical factors, such as decreases in musculotendinous stiffness that may affect the muscle's length–tension relationship and/or sarcomere shortening velocity (Cornwell *et al.*, 2002; Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003; Fowles *et al.*, 2000; Kokkonen *et al.*, 1998; Nelson *et al.*, 2001a, 2001b; Nelson & Kokkonen, 2001), and (b) neural factors, such as decreases in muscle activation (Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000). Fowles *et al.* (2000) reported that after 15 min of recovery from intense stretching, most of the decreases in muscular force-generating capacity were attributable to intrinsic mechanical properties of the musculotendinous unit, rather than neural factors. Specifically, Fowles *et al.* (2000) hypothesized that the stretching could have altered the length–tension relationship and/or the plastic deformation of connective tissues such that the maximal force-producing capabilities of the muscle could be limited. Nelson and co-workers (2001a, 2001b) have also suggested that the primary mechanism underlying the stretching-induced decreases in force production (after 10 min of recovery) is related to a decrease in musculotendinous stiffness that could alter the length–tension relationship of the muscle fibres. Unrelated previous studies have used the angle–torque relationship during maximal isometric (McHugh & Hogan, 2004; McHugh & Tetro, 2003) and isokinetic (Brockett *et al.*, 2001) muscle actions to examine the length–tension relationship in the active muscle fibres. Therefore, to test the hypotheses of Fowles *et al.* (2000) and Nelson *et al.* (2001a, 2001b) that the length–tension relationship is altered by stretching, changes in the values that represent the shape of the angle–torque relationship from pre- to post-stretching in the present study (Figure 2) were investigated. Our findings indicated that despite the stretching-induced decreases in peak torque, there were no changes in work as a result of the static stretching. In the present study, work was calculated as the area under the angle–torque curve, and thus a reduction in the peak of the angle–torque curve (peak torque) should theoretically have reduced the work done. It is possible, however, that the area lost by the reduction in peak torque could have been compensated for by increases in the area under the angle–torque curve at other joint angles. For example, the data for the participant shown in Figure 2 demonstrate increases in peak torque from pre- to post-stretching at joint angles ranging from approximately 40° to 0°, which may have compensated for the work lost due to the decreases in peak torque from 100° to 70°. Since there were no changes in leg extension range of motion from pre- to post-stretching, this "flattening" of the angle–torque relationship without a loss in area under the curve may have reflected stretching-induced alterations in the length–tension relationship. However, because peak torque was not examined at joint angles other than the angle at peak torque in the present study, this hypothesis cannot be confirmed. Therefore, this evidence provides only indirect and tentative support for the hypothesis that static stretching causes acute alterations in the length–tension relationship that may reduce the capacity for maximal force production by the stretched muscle fibres. Future studies are needed to examine specific, localized changes in the angle–torque relationship characterized by maximal isometric muscle actions at multiple joint angles (Brockett *et al.*, 2001; McHugh & Hogan, 2004; McHugh & Tetro, 2003).

The joint angle at peak torque is another measurement of the angle–torque relationship that has been used indirectly to investigate directional shifts in the length–tension relationship (Brockett *et al.*, 2001; McHugh & Hogan, 2004; McHugh & Tetro, 2003). It was hypothesized that changes in the angle at peak torque as a result of stretching could indicate that the sarcomeres are producing peak tension at a less-than-optimal position (Fowles *et al.*, 2000; Nelson *et al.*, 2001a). Previous studies have reported stretching-induced changes in the angle at peak torque, such that the angle occurred at longer muscle lengths during isometric (Fowles *et al.*, 2000; Nelson *et al.*, 2001a) and isokinetic (Cramer *et al.*, 2004a) muscle actions. Other studies, however, have reported no changes in the angle at peak torque as a result of stretching (Cramer *et al.*, 2005; Nelson *et al.*, 2001b). The results of the present study support those of Nelson *et al.* (2001b) and Cramer *et al.* (2005) and indicated no change in the angle at peak torque from pre- to post-stretching. This finding, in conjunction with the lack of change in work done and leg extension range of motion observed in the present study, suggests that the stretching-induced decreases in peak torque may have been due, in part, to a "flattening" of the distributional characteristics of the angle–torque relationship, rather than decreases in the area under the curve (work) or directional shifts in the curve (angle at peak torque) as a result of the static stretching.

One explanation as to why alterations in the angle–torque relationship might reduce the capacity for peak torque production could be related to a stretching-induced increase in the initial sarcomere shortening velocity (Nelson *et al.*, 2001b). Specifically, Nelson *et al.* (2001b) hypothesized that "a more compliant unit might initially allow the contractile component to shorten at a faster rate, and this would continue until the elastic components reached their limit of stretch ... It is tempting to suggest, therefore, that in the present study the

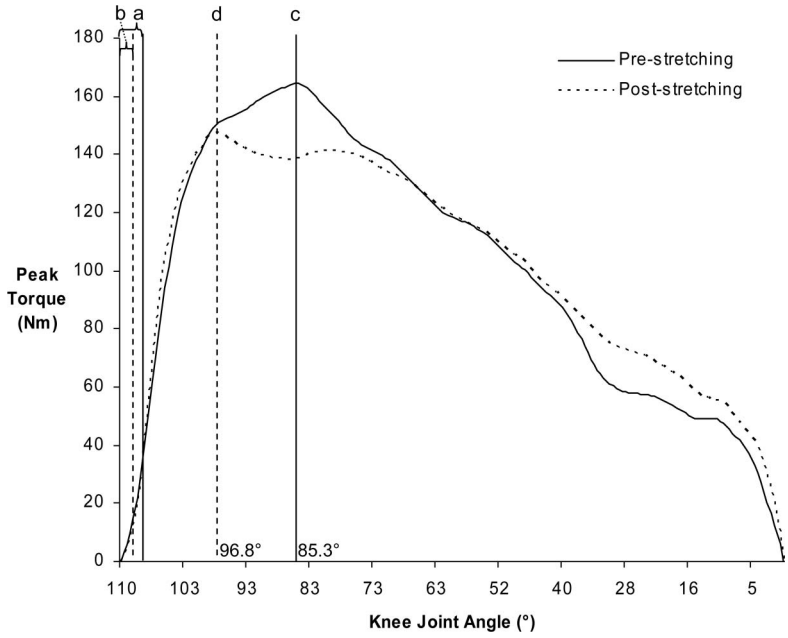

Figure 2. Examples of the angle–torque relationships for one participant during maximal concentric isokinetic leg extensions at 1.04 rad · s^{-1} for the pre-stretching (solid line) and post-stretching (dashed line) isokinetic assessments. (a) The acceleration time (ms, solid vertical line) from the onset of movement to the pre-selected velocity (1.04 rad · s^{-1}) during the pre-stretching isokinetic assessment. (b) The acceleration time (ms, dashed vertical line) during the post-stretching isokinetic assessment. (c) The joint angle at peak torque (degrees, solid vertical line) during the pre-stretching isokinetic assessment. (d) The joint angle at peak torque (degrees, dashed vertical line) during the post-stretching isokinetic assessment.

stretching protocol reduced the active musculotendinous stiffness sufficiently to allow the contractile component to shorten farther and at a faster rate, thus reducing force output'' (p. 244). An increase in the sarcomere shortening velocity in response to static stretching might be manifested through a more rapid acceleration phase of the limb from rest to the pre-set angular velocity during maximal concentric isokinetic muscle actions. The results of the present study indicated a decrease in acceleration time from pre- to post-stretching (Figure 1b). Acceleration time was defined as the time elapsing from the initiation of concentric velocity production to the initiation of a constant angular velocity (Brown, 2000; Brown & Whitehurst, 2003; Brown *et al.*, 1995). These results suggest, therefore, that the static stretching allowed the leg extensor muscles to accelerate the leg more rapidly from rest to the constant angular velocities (1.04 and 5.23 rad · s^{-1}). These findings provide tentative support for the hypothesis of Nelson *et al.* (2001b) that static stretching may increase the initial sarcomere shortening velocity, which would result in a decrease in force production due to the force–velocity relationship.

Several studies have reported stretching-induced decreases in muscle activation through the use of surface (Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004) and fine-wire (Avela *et al.*, 1999) EMG as well as twitch interpolation (Behm *et al.*, 2001; Fowles *et al.*, 2000; Power *et al.*, 2004). For example, Avela *et al.* (1999) reported decreases in motor unit recruitment (EMG amplitude) and firing frequency (zero crossing rate) after repeated passive stretches of the plantar flexors. Using the formula of Duchateau (1995), Fowles *et al.* (2000) reported that 60% of the stretching-induced decreases in force production of the triceps surae (up to 15 min post-stretching) were due to neural factors. Moreover, Behm *et al.* (2001) suggested that at least part of the stretching-induced decreases in maximal force production of the leg extensors was due to decreases in muscle activation. In addition, we recently reported decreases in EMG amplitude from pre- to post-stretching in the stretched and unstretched (contralateral) leg extensors, which suggests that the stretching-induced neural deficit could be related to a central nervous system inhibitory mechanism (Cramer *et al.*, 2004a, 2005). Evetovich *et al.* (2003), however, reported stretching-induced decreases in maximal concentric isokinetic peak torque, but no changes in surface EMG amplitude for the biceps brachii. The results of the present study are in line with those of previous studies (Avela *et al.*, 1999; Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000) and indicate decreases in EMG amplitude at 1.04 and 5.23 rad · s^{-1} for the rectus femoris as a result of the static stretching (Figure 1c). The differences between these results and those of Evetovich *et al.*

(2003) could be related to the architectural and/or anatomical differences between the muscle groups involved (i.e. rectus femoris vs. biceps brachii).

The amplitude of the EMG signal reflects muscle activation (both motor unit recruitment and firing rate) and is a reliable index of the efficiency of the neuromuscular system (deVries, 1968; Moritani, 1993). The amplitude of the MMG signal, however, is influenced by many factors, including the temperature and mass of the muscle, the viscosity of the intracellular and extracellular fluid media, and the number of active motor units and their firing rates (Marchetti, Felici, Bernardi, Minasi, & Di Filippo, 1992; Orizio, 1993; Orizio *et al.*, 2003; Orizio & Veicsteinas, 1992; Stokes, 1993; Stokes & Blythe, 2001). Muscle stiffness also affects MMG amplitude (Orizio, 1993), and it has been hypothesized that stretching-induced decreases in muscle stiffness may enhance the ability of the muscle fibres to oscillate, thereby increasing MMG amplitude (Cramer *et al.*, 2005; Evetovich *et al.*, 2003). The findings regarding this hypothesis, however, are inconclusive, since Evetovich *et al.* (2003) reported stretching-induced increases in MMG amplitude for the biceps brachii during maximal concentric isokinetic forearm flexion muscle actions at 1.04 and 4.72 rad $\cdot$ s^{-1}, while Cramer *et al.* (2005) found that static stretching resulted in no change in MMG amplitude for the rectus femoris and vastus lateralis muscles during maximal concentric isokinetic leg extensions at 1.04 and 5.23 rad $\cdot$ s^{-1}. Therefore, the MMG and EMG signals may provide useful information regarding the mechanical and neural hypotheses underlying the stretching-induced force deficit.

It has been suggested that MMG amplitude is inversely related to muscle stiffness (Barry & Cole, 1988; Cramer *et al.*, 2000a, 2000b, 2002a, 2002b, 2002c, 2004b; Evetovich *et al.*, 1997; Orizio, 1993; Orizio, Peirini, & Veicsteinas, 1989). That is, as muscle stiffness decreases, the active muscle fibres are allowed to oscillate to a greater extent, which causes an increase in the amplitude of the MMG signal. Data reported by Fowles *et al.* (2000) and Halar, Stolov, Venkatesh, Borozovich and Harley (1978) suggested that prolonged stretching may increase the resting length of the contractile components within a muscle, rather than the tendon. Thus, decreases in ''musculotendinous stiffness'' as a result of static stretching may be manifested through decreases in ''muscle stiffness'' as well as ''tendinous stiffness''. Therefore, based on the inverse relationship between MMG amplitude and muscle stiffness (Barry & Cole, 1988; Cramer *et al.*, 2000a, 2000b, 2002a, 2002b, 2002c, 2004b; Evetovich *et al.*, 1997; Orizio, 1993; Orizio *et al.*, 1989) and data from Fowles *et al.* (2000) and Halar *et al.* (1978), we hypothesized that MMG amplitude would increase as a result of the static stretching. It has been demonstrated, however, that MMG amplitude is directly related to muscle activation (EMG amplitude) during submaximal to maximal muscle actions (Beck *et al.*, 2004a, 2004b; Coburn *et al.*, 2004; Maton, Petitjean, & Cnockaert, 1990; Zwarts & Keidel, 1991). That is, as muscle activation increases with increasing submaximal force production, MMG amplitude also increases, which could reflect a greater quantity of muscle fibres that are oscillating from the additional motor units being recruited. In the present study, we observed a decrease in peak torque and EMG amplitude, but no change in MMG amplitude from pre- to post-stretching. It is possible that any increases in MMG amplitude as a result of the stretching-induced decreases in muscle stiffness were counteracted by the decreases in muscle activation. A decrease in muscle activation (EMG amplitude) as a result of the static stretching may have decreased the number of oscillating muscle fibres that contributed to the MMG signal. This hypothesis is supported by Evetovich *et al.* (2003), who reported no change in EMG amplitude but a significant increase in MMG amplitude as a result of static stretching for the biceps brachii muscle. Future studies should examine the effects of stretching on MMG amplitude and the competing influences of muscle stiffness and motor unit activation on the MMG signal.

There were no sex-related differences for the decreases in peak torque, acceleration time, range of motion, or EMG amplitude from pre- to post-stretching. The only differences between the sexes were that the absolute values for peak torque and work were higher for the men than the women at both 1.04 and 5.23 rad $\cdot$ s^{-1}. These results suggest that men and women respond in the same way to static stretching. In addition, there were velocity-related decreases in the joint angle at peak torque from 1.04 to 5.23 rad $\cdot$ s^{-1}, which is consistent with previous findings that this angle is velocity-dependent (Knapik, Wright, Mawdsley, & Braun, 1983). The results also indicated velocity-related increases in acceleration time and MMG amplitude from 1.04 to 5.23 rad $\cdot$ s^{-1}. These findings are consistent with those of Brown *et al.* (1995), who reported increases in acceleration range of motion from 1.04 to 5.23 rad $\cdot$ s^{-1}. Furthermore, results from our laboratory have consistently shown velocity-related increases in MMG amplitude (Cramer *et al.*, 2000a, 2000b, 2002a, 2002b, 2002c, 2004b) similar to those of the present study, which could be related to the physical principles governing the vibrational characteristics of systems that increase power production (Bodor, 1999).

In conclusion, the results of the present study indicated decreases in peak torque and EMG

amplitude and improvements in acceleration time from pre- to post-stretching at 1.04 and 5.23 rad $\cdot$ s^{-1}, but there were no stretching-induced changes for work, joint angle at peak torque, range of motion, or MMG amplitude. These findings are consistent with previous studies (Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004) that have shown decreases in muscle strength (peak torque) and muscle activation (EMG amplitude) as a result of stretching. Two hypotheses have been proposed to explain the stretching-induced decreases in strength: (a) mechanical factors involving alterations in the sarcomere shortening velocity and (b) neurological factors involving decreases in muscle activation. With the purpose of testing the ''mechanical'' hypothesis (Cornwell *et al.*, 2002; Cramer *et al.*, 2004a, 2005; Evetovich *et al.*, 2003; Fowles *et al.*, 2000; Kokkonen *et al.*, 1998; Nelson *et al.*, 2001a, 2001b; Nelson & Kokkonen, 2001), our results indicated no stretching-related changes in the area under the angle–torque curve (work), no directional shifts (joint angle at peak torque) in the angle–torque relationship, but a significant decrease in peak torque. Therefore, since there were also no changes in the leg extension range of motion, these findings indirectly and tentatively suggest that static stretching may cause a ''flattening'' of the angle–torque relationship that reduces peak strength, but allows for greater force production at other joint angles, thereby maintaining the amount of work performed by the repetition. Furthermore, the increased limb acceleration rates (decreased acceleration time) in the present study supported the hypothesis of Nelson *et al.* (2001b) that static stretching may increase the initial sarcomere shortening velocity, thereby reducing peak strength due to the force–velocity relationship. However, further studies employing more sophisticated characterizations of the angle–torque curve (i.e. isometric muscle actions at multiple joint angles) are needed to determine the effects of static stretching on specific, localized areas of the angle–torque curve. In addition, due to the potential for mechanomyography as a non-invasive mechanism for providing unique information about changes in the mechanical and neural properties of muscle, future studies should examine the competing influences of muscle stiffness and muscle activation on the MMG signal.

Regarding the ''neurological'' hypothesis (Avela *et al.*, 1999; Behm *et al.*, 2001; Cramer *et al.*, 2004a, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004), our results indicated a decrease in muscle activation (EMG amplitude) as a result of the static stretching, which was consistent with several previous studies (Behm *et al.*, 2001; Cramer *et al.*, 2005; Fowles *et al.*, 2000; Power *et al.*, 2004). Therefore, these findings suggest that acute decreases in strength after a bout of static stretching may be due to both the mechanical and neurological factors, involving stretching-induced increases in sarcomere shortening velocity as well as decreases in muscle activation. Although specific recommendations regarding stretching before performance must await further evidence, static stretching appears to affect muscle strength at slow and fast speeds, and thus may affect all types of athletes. Future studies should determine the volume of stretching necessary to safely increase joint range of motion before performance, but not elicit detrimental changes in muscle force production that could adversely affect performance.

References

Avela, J., Kyrolainen, H., & Komi, P. V. (1999). Altered reflex sensitivity after repeated and prolonged passive muscle stretching. *Journal of Applied Physiology*, *86*, 1283–1291.

Barry, D. T., & Cole, N. M. (1988). Fluid mechanics of muscle vibrations. *Biophysics Journal*, *53*, 899–905.

Basmajian, J. V., & De Luca, C. J. (1985). *Muscles alive: Their functions revealed by electromyography.* Baltimore, MD: Williams & Wilkins.

Beaulieu, J. E. (1981). Developing a stretching program. *Physician and Sportsmedicine*, *9*, 59–66.

Beck, T. W., Housh, T. J., Johnson, G. O., Weir, J. P., Cramer, J. T., Coburn, J. W. *et al.* (2004a). Mechanomyographic amplitude and mean power frequency versus torque relationships during isokinetic and isometric muscle actions of the biceps brachii. *Journal of Electromyography and Kinesiology*, *14*, 555–564.

Beck, T. W., Housh, T. J., Johnson, G. O., Weir, J. P., Cramer, J. T., Coburn, J. W. *et al.* (2004b). Mechanomyographic and electromyographic time and frequency domain responses during submaximal to maximal isokinetic muscle actions of the biceps brachii. *European Journal of Applied Physiology*, *92*, 352–359.

Behm, D. G., Bambury, A., Cahill, F., & Power, K. (2004). Effect of acute static stretching on force, balance, reaction time, and movement time. *Medicine and Science in Sports and Exercise*, *36*, 1397–1402.

Behm, D. G., Button, D. C., & Butt, J. C. (2001). Factors affecting force loss with prolonged stretching. *Canadian Journal of Applied Physiology*, *26*, 261–272.

Bixler, B., & Jones, R. L. (1992). High-school football injuries: Effects of a post-halftime warm-up and stretching routine. *Family Practice Research Journal*, *12*, 131–139.

Bodor, M. (1999). Mechanomyographic and electromyographic muscle responses are related to power. *Muscle and Nerve*, *22*, 649–650.

Brockett, C. L., Morgan, D. L., & Proske, U. (2001). Human hamstring muscles adapt to eccentric exercise by changing optimum length. *Medicine and Science in Sports and Exercise*, *33*, 783–790.

Brown, L. E. (Ed.) (2000). *Isokinetics in human performance.* Champaign, IL: Human Kinetics.

Brown, L. E., & Whitehurst, M. (2003). The effect of short-term isokinetic training on force and rate of velocity development. *Journal of Strength and Conditioning Research*, *17*, 88–94.

Brown, L. E., Whitehurst, M., Gilbert, R., & Buchalter, D. N. (1995). The effect of velocity and gender on load range during knee extension and flexion exercise on an isokinetic device. *Journal of Orthopedic and Sports Physical Therapy*, *21*, 107–112.

Church, J. B., Wiggins, M. S., Moode, F. M., & Crist, R. (2001). Effect of warm-up and flexibility treatments on vertical jump performance. *Journal of Strength and Conditioning Research*, *15*, 332–336.

Coburn, J. W., Housh, T. J., Cramer, J. T., Weir, J. P., Miller, J. M., Beck, T. W. *et al.* (2004). Mechanomyographic time and frequency domain responses of the vastus medialis muscle during submaximal to maximal isometric and isokinetic muscle actions. *Electromyography and Clinical Neurophysiology*, *44*, 247–255.

Cornwell, A., Nelson, A. G., & Sidaway, B. (2002). Acute effects of stretching on the neuromechanical properties of the triceps surae muscle complex. *European Journal of Applied Physiology*, *86*, 428–434.

Cramer, J. T., Housh, T. J., Evetovich, T. K., Johnson, G. O., Ebersole, K. T., Perry, S. R. *et al.* (2002a). The relationships among peak torque, mean power output, mechanomyography, and electromyography in men and women during maximal, eccentric isokinetic muscle actions. *European Journal of Applied Physiology*, *86*, 226–232.

Cramer, J. T., Housh, T. J., Johnson, G. O., Ebersole, K. T., Perry, S. R., & Bull, A. J. (2000a). Mechanomyographic amplitude and mean power output during maximal, concentric, isokinetic muscle actions. *Muscle and Nerve*, *23*, 1826–1831.

Cramer, J. T., Housh, T. J., Johnson, G. O., Ebersole, K. T., Perry, S. R., & Bull, A. J. (2000b). Mechanomyographic and electromyographic responses of the superficial muscles of the quadriceps femoris during maximal, concentric isokinetic muscle actions. *Isokinetics and Exercise Science*, *8*, 109–117.

Cramer, J. T., Housh, T. J., Johnson, G. O., Miller, J. M., Coburn, J. W., & Beck, T. W. (2004a). The acute effects of static stretching on peak torque in women. *Journal of Strength and Conditioning Research*, *18*, 176–182.

Cramer, J. T., Housh, T. J., Weir, J. P., Johnson, G. O., Berning, J. M., Perry, S. R. *et al.* (2002b). Mechanomyographic and electromyographic amplitude and frequency responses from the superficial quadriceps femoris muscles during maximal, eccentric isokinetic muscle actions. *Electromyography and Clinical Neurophysiology*, *42*, 337–346.

Cramer, J. T., Housh, T. J., Weir, J. P., Johnson, G. O., Berning, J. M., Perry, S. R. *et al.* (2004b). Gender, muscle, and velocity comparisons of mechanomyographic and electromyographic responses during isokinetic muscle actions. *Scandinavian Journal of Medicine and Science in Sports*, *14*, 116–127.

Cramer, J. T., Housh, T. J., Weir, J. P., Johnson, G. O., Coburn, J. W., & Beck, T. W. (2005). The acute effects of static stretching on peak torque, mean power output, electromyography, and mechanomyography. *European Journal of Applied Physiology*, *93*, 530–539.

Cramer, J. T., Housh, T. J., Weir, J. P., Johnson, G. O., Ebersole, K. T., Perry, S. R. *et al.* (2002c). Power output, mechanomyographic, and electromyographic responses to maximal, concentric, isokinetic muscle actions in men and women. *Journal of Strength and Conditioning Research*, *16*, 399–408.

deVries, H. A. (1963). The "looseness" factor in speed and O_2 consumption of an anaerobic 100-yard dash. *Research Quarterly for Exercise and Sport*, *34*, 305–313.

deVries, H. A. (1968). "Efficiency of electrical activity" as a physiological measure of the functional state of muscle tissue. *American Journal of Physical Medicine*, *47*, 10–22.

Duchateau, J. (1995). Bed rest induces neural and contractile adaptations in triceps surae. *Medicine and Science in Sports and Exercise*, *27*, 1581–1589.

Ebersole, K. T., Housh, T. J., Johnson, G. O., Evetovich, T. K., Smith, D. B., & Perry, S. R. (1999). MMG and EMG responses of the superficial quadriceps femoris muscles. *Journal of Electromyography and Kinesiology*, *9*, 219–227.

Ekstrand, J., Gillquist, J., & Liljedahl, S. O. (1983a). Prevention of soccer injuries: Supervision by doctor and physiotherapist. *American Journal of Sports Medicine*, *11*, 116–120.

Ekstrand, J., Gillquist, J., Moller, M., Oberg, B., & Liljedahl, S. O. (1983b). Incidence of soccer injuries and their relation to training and team success. *American Journal of Sports Medicine*, *11*, 63–67.

Enoka, R. M. (2002). Activation order of motor axons in electrically evoked contractions. *Muscle and Nerve*, *25*, 763–764.

Evetovich, T. K., Housh, T. J., Johnson, G. O., Smith, D. B., Ebersole, K. T., & Perry, S. R. (1998). Gender comparisons of the mechanomyographic responses to maximal concentric and eccentric isokinetic muscle actions. *Medicine and Science in Sports and Exercise*, *30*, 1697–1702.

Evetovich, T. K., Housh, T. J., Stout, J. R., Johnson, G. O., Smith, D. B., & Ebersole, K. T. (1997). Mechanomyographic responses to concentric isokinetic muscle contractions. *European Journal of Applied Physiology and Occupational Physiology*, *75*, 166–169.

Evetovich, T. K., Nauman, N. J., Conley, D. S., & Todd, J. B. (2003). Effect of static stretching of the biceps brachii on torque, electromyography, and mechanomyography during concentric isokinetic muscle actions. *Journal of Strength and Conditioning Research*, *17*, 484–488.

Fowles, J. R., Sale, D. G., & MacDougall, J. D. (2000). Reduced strength after passive stretch of the human plantarflexors. *Journal of Applied Physiology*, *89*, 1179–1188.

Franklin, B. A., Whaley, M. H., Howley, E. T., & Balady, G. J. (2000). *ACSM's guidelines for exercise testing and prescription.* Philadelphia, PA: Lippincott Williams & Wilkins.

Fry, A. C., McLellan, E., Weiss, L. W., & Rosato, F. D. (2003). The effects of static stretching on power and velocity during the bench press exercise. *Medicine and Science in Sports and Exercise*, *35*, (suppl. 5), S264.

Garrett, W. E., Jr. (1990). Muscle strain injuries: Clinical and basic aspects. *Medicine and Science in Sports and Exercise*, *22*, 436–443.

Gordon, G., & Holbourn, H. (1948). The sounds from single motor units in a contracting muscle. *Journal of Physiology*, *107*, 456–464.

Halar, E. M., Stolov, W. C., Venkatesh, B., Brozovich, F. V., & Harley, J. D. (1978). Gastrocnemius muscle belly and tendon length in stroke patients and able-bodied persons. *Archives of Physical Medicine and Rehabilitation*, *59*, 476–484.

Hermens, H. J., Freriks, B., Merletti, R., Stegeman, D., Blok, J., Rau, G., Disselhorst-Klug, C., & Hegg, G. (1999). *European Recommendations for Surface ElectroMyography: Results of the SENIAM Project (SENIAM8)*. Enschede, The Netherlands: Roessingh Research and Development B.V.

Holcomb, W. R. (2000). Stretching and warm-up. In T. R. Baechle & R. W. Earle (Eds.), *Essentials of strength training and conditioning* (pp. 321–342). Champaign, IL: Human Kinetics.

Housh, T. J., Perry, S. R., Bull, A. J., Johnson, G. O., Ebersole, K. T., Housh, D. J. *et al.* (2000). Mechanomyographic and electromyographic responses during submaximal cycle ergometry. *European Journal of Applied Physiology*, *83*, 381–387.

Knapik, J. J., Wright, J. E., Mawdsley, R. H., & Braun, J. (1983). Isometric, isotonic, and isokinetic torque variations in four muscle groups through a range of joint motion. *Physical Therapy*, *63*, 938–947.

Knudson, D., Bennett, K., Corn, R., Leick, D., & Smith, C. (2001). Acute effects of stretching are not evident in the kinematics of the vertical jump. *Journal of Strength and Conditioning Research*, *15*, 98–101.

Kokkonen, J., Nelson, A. G., & Cornwell, A. (1998). Acute muscle stretching inhibits maximal strength performance. *Research Quarterly for Exercise and Sport*, *69*, 411–415.

Marchetti, M., Felici, F., Bernardi, M., Minasi, P., & Di Filippo, L. (1992). Can evoked phonomyography be used to recognize fast and slow muscle in man? *International Journal of Sports Medicine*, *13*, 65–68.

Maton, B., Petitjean, M., & Cnockaert, J. C. (1990). Phonomyogram and electromyogram relationships with isometric force reinvestigated in man. *European Journal of Applied Physiology and Occupational Physiology*, *60*, 194–201.

McHugh, M. P., & Hogan, D. E. (2004). Effect of knee flexion angle on active joint stiffness. *Acta Physiologica Scandinavica*, *180*, 249–254.

McHugh, M. P., & Tetro, D. T. (2003). Changes in the relationship between joint angle and torque production associated with the repeated bout effect. *Journal of Sports Sciences*, *21*, 927–932.

McNeal, J. R., & Sands, W. A. (2003). Acute static stretching reduces lower extremity power in trained children. *Pediatric Exercise Science*, *15*, 139–145.

Moritani, T. (1993). Neuromuscular adaptations during the acquisition of muscle strength, power and motor tasks. *Journal of Biomechanics*, *26* (suppl. 1), 95–107.

Nelson, A. G., Allen, J. D., Cornwell, A., & Kokkonen, J. (2001a). Inhibition of maximal voluntary isometric torque production by acute stretching is joint-angle specific. *Research Quarterly for Exercise and Sport*, *72*, 68–70.

Nelson, A. G., Guillory, I. K., Cornwell, C., & Kokkonen, J. (2001b). Inhibition of maximal voluntary isokinetic torque production following stretching is velocity-specific. *Journal of Strength and Conditioning Research*, *15*, 241–246.

Nelson, A. G., & Kokkonen, J. (2001). Acute ballistic muscle stretching inhibits maximal strength performance. *Research Quarterly for Exercise and Sport*, *72*, 415–419.

Orizio, C. (1993). Muscle sound: Bases for the introduction of a mechanomyographic signal in muscle studies. *Critical Reviews in Biomedical Engineering*, *21*, 201–243.

Orizio, C., Gobbo, M., Diemont, B., Esposito, F., & Veicsteinas, A. (2003). The surface mechanomyogram as a tool to describe the influence of fatigue on biceps brachii motor unit activation strategy: Historical basis and novel evidence. *European Journal of Applied Physiology*, *90*, 326–336.

Orizio, C., Liberati, D., Locatelli, C., De Grandis, D., & Veicsteinas, A. (1996). Surface mechanomyogram reflects muscle fibres twitches summation. *Journal of Biomechanics*, *29*, 475–481.

Orizio, C., Perini, R., & Veicsteinas, A. (1989). Changes of muscular sound during sustained isometric contraction up to exhaustion. *Journal of Applied Physiology*, *66*, 1593–1598.

Orizio, C., & Veicsteinas, A. (1992). Soundmyogram analysis during sustained maximal voluntary contraction in sprinters and long distance runners. *International Journal of Sports Medicine*, *13*, 594–599.

Perrin, D. (1986). Reliability of isokinetic measures. *Athletic Training*, *21*, 319–321.

Perry, S. R., Housh, T. J., Johnson, G. O., Ebersole, K. T., & Bull, A. J. (2001a). Mechanomyographic responses to continuous, constant power output cycle ergometry. *Electromyography and Clinical Neurophysiology*, *41*, 137–144.

Perry, S. R., Housh, T. J., Johnson, G. O., Ebersole, K. T., Bull, A. J., Evetovich, T. K. *et al.* (2001b). Mechanomyography, electromyography, heart rate, and ratings of perceived exertion during incremental cycle ergometry. *Journal of Sports Medicine and Physical Fitness*, *41*, 183–188.

Power, K., Behm, D., Cahill, F., Carroll, M., & Young, W. (2004). An acute bout of static stretching: Effects on force and jumping performance. *Medicine and Science in Sports and Exercise*, *36*, 1389–1396.

Safran, M. R., Seaber, A. V., & Garrett, W. E., Jr. (1989). Warm-up and muscular injury prevention: An update. *Sports Medicine*, *8*, 239–249.

Shellock, F. G., & Prentice, W. E. (1985). Warming-up and stretching for improved physical performance and prevention of sports-related injuries. *Sports Medicine*, *2*, 267–278.

Shinohara, M., Kouzaki, M., Yoshihisa, T., & Fukunaga, T. (1997). Mechanomyography of the human quadriceps muscle during incremental cycle ergometry. *European Journal of Applied Physiology and Occupational Physiology*, *76*, 314–319.

Shrier, I. (1999). Stretching before exercise does not reduce the risk of local muscle injury: A critical review of the clinical and basic science literature. *Clinical Journal of Sports Medicine*, *9*, 221–227.

Smith, C. A. (1994). The warm-up procedure: To stretch or not to stretch. A brief review. *Journal of Orthopedic and Sports Physical Therapy*, *19*, 12–17.

Stokes, M. J. (1993). Acoustic myography: Applications and considerations in measuring muscle performance. *Isokinetics and Exercise Science*, *3*, 4–15.

Stokes, M., & Blythe, M. (2001). *Muscle sounds in physiology, sports science and clinical investigation: Applications and history of mechanomyography.* Oxford: Medintel Medical Intelligence Oxford Ltd.

Stout, J. R., Housh, T. J., Johnson, G. O., Evetovich, T. K., & Smith, D. B. (1997). Mechanomyography and oxygen consumption during incremental cycle ergometry. *European Journal of Applied Physiology and Occupational Physiology*, *76*, 363–367.

Thacker, S. B., Gilchrist, J., Stroup, D. F., & Kimsey, C. D., Jr. (2004). The impact of stretching on sports injury risk: A systematic review of the literature. *Medicine and Science in Sports and Exercise*, *36*, 371–378.

Watakabe, M., Itoh, Y., Mita, K., & Akataki, K. (1998). Technical aspects of mechnomyography recording with piezoelectric contact sensor. *Medical & Biological Engineering and Computing*, *36*, 557–561.

Young, W. B., & Behm, D. G. (2003). Effects of running, static stretching and practice jumps on explosive force production and jumping performance. *Journal of Sports Medicine and Physical Fitness*, *43*, 21–27.

Young, W., & Elliott, S. (2001). Acute effects of static stretching, proprioceptive neuromuscular facilitation stretching, and maximum voluntary contractions on explosive force production and jumping performance. *Research Quarterly for Exercise and Sport*, *72*, 273–279.

Zwarts, M. J., & Keidel, M. (1991). Relationship between electrical and vibratory output of muscle during voluntary contraction and fatigue. *Muscle and Nerve*, *14*, 756–761.