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

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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·s⁻¹ in men and women. Ten women (mean ± s: age 23.0 ± 2.9 years, stature 1.61 ± 0.12 m, mass 63.3 ± 9.9 kg) and eight men (age 21.4 ± 3.0 years, stature 1.83 ± 0.11 m, mass 83.1 ± 15.2 kg) performed maximal voluntary concentric isokinetic leg extensions at 1.04 and 5.23 rad·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 < 0.05) from pre- to post-stretching at 1.04 and 5.23 rad·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;
Nelson et al., 2001b), dynamic constant external resistance (DCER) force (Fry, McLe1lan, 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 musculo-tendonous 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). 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, sensitivity (Avela et al., 1999; Behm et al., 2001, 2004; Cramer et al., 2004a, 2005; Fowles et al., 2000; Power et al., 2004).
Effects of stretching on the angle–torque relationship

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·s⁻¹ in men and women.

Methods

Ten women (mean ± s: age 23.0 ± 2.9 years, stature 1.61 ± 0.12 m, mass 63.3 ± 9.9 kg) and eight men (age 21.4 ± 3.0 years, stature 1.83 ± 0.11 m, mass 83.1 ± 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·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 repetition 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 exercises, the isokinetic test protocol was 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 r-tests. An alpha level of P ≤ 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
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 \(\cdot\) 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 \(\cdot\) 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 \(\cdot\) 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 \(\cdot\) 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 \(\cdot\) 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 times of 1.04 and 5.23 rad \(\cdot\) 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 \(\cdot\) s\(^{-1}\). There were no changes \((P > 0.05)\) in acceleration time from pre- to post-stretching.

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

<table>
<thead>
<tr>
<th></th>
<th>Pre-stretching</th>
<th>Post-stretching</th>
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<tbody>
<tr>
<td></td>
<td>1.04 rad (\cdot) s(^{-1})</td>
<td>5.23 rad (\cdot) s(^{-1})</td>
</tr>
<tr>
<td>Peak torque (N (\cdot) m)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men mean</td>
<td>239.6</td>
<td>234.1</td>
</tr>
<tr>
<td>(s_x)</td>
<td>20.7</td>
<td>17.5</td>
</tr>
<tr>
<td>Women mean</td>
<td>142.0</td>
<td>134.0</td>
</tr>
<tr>
<td>(s_x)</td>
<td>8.3</td>
<td>6.1</td>
</tr>
<tr>
<td>Work (J)</td>
<td></td>
<td></td>
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<tr>
<td>Men mean</td>
<td>270.2</td>
<td>261.4</td>
</tr>
<tr>
<td>(s_x)</td>
<td>20.4</td>
<td>24.1</td>
</tr>
<tr>
<td>Women mean</td>
<td>163.2</td>
<td>159.7</td>
</tr>
<tr>
<td>(s_x)</td>
<td>11.1</td>
<td>11.5</td>
</tr>
<tr>
<td>Joint angle at peak torque (°)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men mean</td>
<td>61.1</td>
<td>58.5</td>
</tr>
<tr>
<td>(s_x)</td>
<td>2.1</td>
<td>3.2</td>
</tr>
<tr>
<td>Women mean</td>
<td>66.5</td>
<td>64.2</td>
</tr>
<tr>
<td>(s_x)</td>
<td>2.9</td>
<td>3.1</td>
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<tr>
<td>Acceleration time (ms)</td>
<td></td>
<td></td>
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<tr>
<td>Men mean</td>
<td>63.8</td>
<td>37.5</td>
</tr>
<tr>
<td>(s_x)</td>
<td>15.3</td>
<td>6.5</td>
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<tr>
<td>Women mean</td>
<td>56.0</td>
<td>42.2</td>
</tr>
<tr>
<td>(s_x)</td>
<td>12.8</td>
<td>6.5</td>
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<tr>
<td>Range of motion (°)</td>
<td></td>
<td></td>
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<tr>
<td>Men mean</td>
<td>99.6</td>
<td>95.9</td>
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<tr>
<td>(s_x)</td>
<td>3.0</td>
<td>2.9</td>
</tr>
<tr>
<td>Women mean</td>
<td>102.2</td>
<td>101.7</td>
</tr>
<tr>
<td>(s_x)</td>
<td>2.8</td>
<td>2.7</td>
</tr>
<tr>
<td>MMG amplitude (mVrms)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men mean</td>
<td>45.6</td>
<td>51.0</td>
</tr>
<tr>
<td>(s_x)</td>
<td>6.0</td>
<td>7.6</td>
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<tr>
<td>Women mean</td>
<td>54.3</td>
<td>54.0</td>
</tr>
<tr>
<td>(s_x)</td>
<td>7.1</td>
<td>4.3</td>
</tr>
<tr>
<td>EMG amplitude (µVrms)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men mean</td>
<td>549.3</td>
<td>473.0</td>
</tr>
<tr>
<td>(s_x)</td>
<td>76.5</td>
<td>40.5</td>
</tr>
<tr>
<td>Women mean</td>
<td>422.0</td>
<td>401.9</td>
</tr>
<tr>
<td>(s_x)</td>
<td>57.8</td>
<td>54.3</td>
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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/s.

**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/s. 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/s (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/s), but not at the faster velocities (2.62, 3.66, or 4.72 rad/s) (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/s) and fast (5.23 rad/s) 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
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, 2005; Evetovich et al., 1992; Orizio, 1993; 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 at 1.04 and 4.72 rad · 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 · 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 · 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 · 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 · 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 · 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


Effects of stretching on the angle–torque relationship


