Isometric contractions reduce plantar flexor moment, Achilles tendon stiffness and neuromuscular activity but remove the subsequent effects of stretch.

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Contraction-induced force losses

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The effects of isometric contractions and passive stretching on muscle-tendon mechanics and muscle activity were studied in sixteen healthy human volunteers. First, peak concentric and passive ankle joint moment data were recorded on an isokinetic dynamometer with EMG monitoring of the triceps surae; real-time motion analysis of the lower leg and ultrasound imaging of the Achilles-medial gastrocnemius muscle-tendon junction were simultaneously conducted. The subjects then performed six 8-s maximal voluntary isometric contractions (MVICs) before repeating the passive and active trials. Although there was no decrease in isometric joint moment after MVICs, peak concentric moment was significantly reduced (11.5%; \( P < 0.01 \)). This was accompanied by, and correlated with \(( r = 0.90; P < 0.01 )\), significant reductions in peak triceps surae EMG amplitude (21.0%; \( P < 0.01 \)). Both Achilles tendon stiffness (10.9%; \( P < 0.01 \)) and passive joint moment (4.9%; \( P < 0.01 \)) were also significantly reduced. Subsequently, the subjects performed three 60-s static plantar flexor stretches before being re-tested 2 min and 30 min post-stretch. The stretch protocol caused no significant change in any measure. Thirty minutes after stretching, significant recovery in concentric moment and muscle activity was detected at dorsi flexed joint angles, while Achilles tendon stiffness and passive joint moment remained significantly reduced. These data show that the performance of maximal isometric contractions interrupts the normal stretch-induced losses in active and passive plantar flexor joint moment and neuromuscular activity, largely because concentric strength and tendon properties were already affected. Importantly, the decrease in Achilles tendon stiffness remained 30 min later, which may be an important etiological factor for muscle-tendon strain injury risk.

Keywords: Triceps surae, force deficits, tissue mechanics, electromyography
INTRODUCTION

Pre-performance warm-up routines are commonly promoted and are specifically designed to prepare an individual for high intensity physical activity and to reduce the risk of injury (9, 56). The routines typically include cardiovascular work, stretching and strong muscular contractions (with progressing intensity), which promotes increased peripheral blood flow to the working muscle, elevated intramuscular temperature, enhanced neural conduction velocity, increased range of motion (ROM) and decreased viscosity and stiffness of the MTC (1, 2, 9, 26, 27, 31, 32, 56). The stretching routines conducted within the warm-up protocol are employed primarily to increase functional range of motion (ROM) and reduce MTC stiffness in an attempt to reduce injury risk, although their effect in this regard is still debated (21, 49, 53, 55). The strong muscular isometric contractions have been shown to modify tendon stiffness (32) and potentiate muscle force (6, 23, 45), which may optimise neuromuscular recruitment and force production. Accordingly, some athletes use maximal isometric contractions within their warm-up protocol to potentiate neuromuscular recruitment for optimal performance.

Recently, significant reductions in force and power production have been reported immediately after passive muscle stretching (7, 12-16, 19, 26-29, 37, 38, 42-44, 48, 50, 52, 57). Although there are numerous possible mechanisms underpinning the decrease in force after stretching, two primary mechanisms include: 1) reduced neuromuscular activation (4, 5, 15, 19, 27) and 2) altered mechanical properties of muscle-tendon complex (MTC) (14, 16, 19, 26, 42, 43, 52). Stretch induced reductions in electromyographic activity (EMG) have been reported concurrently with force losses after 30 min (19) and 1 hour (4) of intermittent static stretching, plus stretch-induced force deficits within the plantar flexors have been
strongly correlated with the reductions in triceps surae EMG amplitude (~65% explained variance; \( P < 0.01 \)) (27). However, the changes in active joint moment cannot be fully explained by changes in neuromuscular activity so a separate mechanism must be partially responsible for the force deficits.

Post-stretch reductions in passive moment (26, 27, 31, 35, 36, 41), indicative of changes in the mechanical properties of the MTC, could impact force generating capacity of important muscle groups such as the triceps surae if there was a decrease in the series stiffness (and in particular a decrease in tendon stiffness), which would cause the muscle to operate at a shorter, weaker length (33, 34). Indeed, Kubo et al. (31) reported a significant decrease in Achilles tendon stiffness and muscle operating length after 10 min of static plantar flexor stretch, although, interestingly, this was not associated with a reduced isometric force output. In contrast, Morse et al. (41) and Kay & Blazevich (27) have reported that shorter duration stretches (< 5 min) did not affect Achilles tendon stiffness nor reduce muscle operating length (27). Interestingly, Kubo et al. (32) reported similar reductions in tendon stiffness following fifty 3-s isometric contractions. Collectively, these studies suggest that the duration and intensity of tissue strain imposed by either stretching or strong muscular contractions may determine whether changes in tendon stiffness occur. Identifying interventions that alter the mechanical properties of the tendon is important as reduced tendon stiffness may increase neuromechanical delay (17, 22, 30), reduce the rate of force development (11, 18, 32) and decrease the active muscle length (32), which could attenuate maximal force in the human plantar flexors according to its force-length relationship (33, 34). Altered mechanical properties have been hypothesized to contribute to post-stretch reductions in force production (14, 16, 19, 26, 42, 43, 52), and given that similar changes in tendon
stiffness may be induced by intense contractions (32), the greater intensity of tissue strain imposed during these contractions may mitigate or remove the effects of subsequent stretch.

Understanding the effects of maximal contractions combined with stretching is important for two reasons. First, some researchers (7, 13-15, 37, 47) but not others (35, 36, 41) have included maximal isometric contractions in the warm-up or as part of the experimental model prior to testing the effects of static stretch on MTC mechanics, neuromuscular activation and force production. The impact of their inclusion on subsequent stretch-induced force losses and the mechanical or neuromuscular mechanisms associated with these losses has not been directly measured. The inclusion of these contractions may modify MTC mechanics prior to the stretch intervention, which may reverse, mitigate or compound the effects of subsequent stretch. Second, warm-up protocols commonly include both maximal contractions and stretching so examining each activity in isolation does not allow estimation of their combined effects on tendon properties and force production.

To gain a more comprehensive understanding of the impact of both isometric contractions and static stretch on force production, it is necessary to quantify muscle activity and muscle (or tendon) length changes simultaneously, within a multi-intervention protocol. The present study aimed first to determine the effects of six 8-s ramped maximal voluntary isometric contractions (MVICs) on Achilles tendon stiffness, gastrocnemius medialis (GM) muscle operating length, active (concentric) and passive plantar flexor joint moment and neuromuscular activity (EMG) of the triceps surae. Second, additional effects of stretch on these measures were quantified 2 min and 30 min post-stretch to determine the influence of these contractions to the well-documented effects of stretch.
MATERIALS & METHODS

Subjects

Sixteen active participants (8 women and 8 men; age = 20.2 ± 2.6 y, mass = 65.5 ± 10.5 kg, height = 1.7 ± 0.1 m) with no recent history of lower limb injury or illness volunteered for the study after providing written and informed consent. The subjects were asked to avoid intense exercise, stretching and stimulant use for 48 hr prior to testing. Ethical approval was granted by the Ethics Committee’s of The School of Sport and Education at Brunel University and The School of Health at The University of Northampton and the study was conducted in accordance with the declaration of Helsinki.

Protocol

Overview

For a comprehensive review of the methods see Kay & Blazevich (2009). The subjects visited the laboratory on three occasions separated each time by one week. They were initially familiarized with the testing protocol one week prior to data collection and then visited the lab on two further occasions, once under control conditions (no stretch) and once under the experimental condition, in a randomized order. During the experimental sessions, the subjects performed a 5-min warm-up on a Monark cycle at 60 revolutions/min with a 1-kg resistance load producing a power output of 60 W. The subjects were then seated upright in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with knee fully extended (0°) to ensure the gastrocnemii were also placed under significant stretch and contributed significantly to plantar flexor joint moment (17, 25). The ankle was placed in neutral position (0°) with the sole of the foot perpendicular to the shank, and the lateral malleolus aligned to the centre of rotation of the dynamometer. The dynamometer footplate
initially rotated the subjects’ ankles to full plantar flexion (~30°), and then the subjects’ ankles were passively rotated to their maximum dorsiflexion range of motion (ROM) at 0.087 rad·s⁻¹ (5°·s⁻¹), whilst in this position the subjects performed a maximal concentric plantar flexor contraction at an angular velocity of 0.087 rad·s⁻¹. Subsequently, the subjects produced six 8-s ramped maximal voluntary isometric contractions (MVICs) with the ankle in neutral position (0°) before repeating the passive and active trials to determine any effects of isometric contractions. Three 60-s static plantar flexor stretches were then imposed by rotating the ankle to full dorsiflexion ROM, with 60 s of rest included between each stretch. The subjects subsequently repeated the passive and active trials at 2 min and 30 min post-stretch to determine the effects of stretch. The order and time of the experimental protocol including the passive and concentric trials and isometric and stretch interventions is shown in Fig. 1.

Passive ankle moment

While seated with a hip angle of 55° and their knee extended the subjects’ ankles were passively rotated through their full ROM at 0.087 rad·s⁻¹. They were instructed to volitionally terminate the rotation by pressing a hand held release button at the point of discomfort. Passive moment was recorded throughout the trial and then normalized (as a percentage) to the maximum pre-stretch passive joint moment (%Mₚₚ). To account for inter-individual differences in joint flexibility/ROM, moment data were analyzed at 50%, 70% and 90% of maximum ROM. Full ROM was calculated from the passive joint moment inflection point (mean angle = 0.8 ± 4.8° dorsiflexion), where a clear change in the slope of the passive moment curve occurred (27), to the volitional end of the ROM.
Whilst seated with an 85° hip angle and the knee extended, the dynamometer rotated the subjects’ ankles through their ROM at 0.087 rad·s⁻¹ until reaching the point of discomfort. The subjects then maximally contracted the plantar flexors until maximal isometric moment was attained (i.e. there was a visible plateau in the moment trace) before the footplate of the dynamometer was released at 0.087 rad·s⁻¹. This enabled the subjects to continue to maximally contract the plantar flexors through their full ROM (see Fig 2). Concentric plantar flexor moment was normalized as a percentage of the maximum plantar flexor moment measured during the maximum voluntary isometric contraction (%MVIC). Maximal concentric moment was recorded throughout the full ROM; data were analyzed only at 50% (mean angle = 7.7 ± 3.8° plantar flexion), 70% (mean angle = 0.7 ± 5.3° plantar flexion) and 90% (mean angle = 6.2 ± 6.8° dorsi flexion) of the full ROM, calculated between full plantar flexion (0%) and full dorsiflexion (100%), to remove inter-individual variations in flexibility. Analysis was not conducted at joint angles <50% of ROM as the slow concentric velocity (5°·s⁻¹) resulted in a total contraction period of approximately 12 s and incurred substantial fatigue. During testing, joint moment, joint angle and angular velocity data for both passive and active trials were directed from the dynamometer to a high level transducer (model HLT100C, Biopac, Goleta, CA) before analogue-to-digital conversion at a 2000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The data were then directed to a personal computer running AcqKnowledge software (v3.8.2, Biopac) and filtered with a double pass 6-Hz Butterworth low pass filter.

Maximal isometric contractions

Two minutes after completing the concentric trial, the subjects’ ankles were passively rotated from full plantar flexion at 0.087 rad·s⁻¹ until they reached the anatomical position (0°). The
subjects then produced a ramped maximal isometric plantar flexor contraction with maximal
moment reached ~3 s after contraction initiation (visible plateau in the moment curve);
subjects slowly reduced their force to zero (total contraction time ~8 s). The ramped
contractions allowed tendon deformation to be determined, which then enabled tendon
stiffness to be calculated. The subjects’ ankles were then returned to a plantarflexed position
(25°) and this process was repeated after a 30-s rest with subjects completing a total of six
contractions during the isometric trial. Two minutes after completing the six isometric
contractions, the subjects repeated the passive and active trials to determine any effects of the
isometric contractions (see Fig. 3).

Stretch protocol

Two minutes after completing the second concentric trial, the subjects’ ankles were passively
rotated at 0.087 rad·s⁻¹ through their full ROM until reaching the point of discomfort, a
position regularly used in stretch studies (7, 16, 26, 27, 47). The movement velocity was too
slow to elicit a significant myotatic stretch reflex response (37, 39, 40), which ensured that
full ROM was achieved and a substantial stress was applied to the MTC. The subjects’
ankles were held in the stretched position for 60 s and then released at 0.087 rad·s⁻¹, returning
the foot to a fully plantarflexed position. After 60 s of rest, the stretch protocol was repeated
(twice), giving a total stretch duration of 180 s.

Electromyographic (EMG) recording

Site preparation, electrode placement, EMG sampling, processing and normalization methods
were completed as previously described (27). Skin-mounted bi-polar double differentiated
active electrodes (model MP-2A, Linton, Norfolk, UK) constantly monitored the EMG
activity of the soleus (Sol), gastrocnemius medialis (GM), gastrocnemius lateralis (GL) and
tibialis anterior (TA). EMG signals were amplified (gain = 300, input impedance = 10 GΩ, CMRR = > 100 dB at 65 Hz) and directed to a high level transducer (model HLT100C, Biopac) before analogue-to-digital conversion at a 2000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The signals were then directed to a personal computer running AcqKnowledge software (v3.8.2, Biopac) where they were filtered using a 20- to 500-Hz band-pass filter. The filtered signal was then converted to root mean squared (RMS) EMG with a 250-ms sample window, and normalized as a percentage of the peak amplitude recorded during a maximal voluntary isometric contraction. The normalized EMG amplitude (%MVIC) was used as a measure of neuromuscular activity; the normalized EMG signals for GM, GL and Sol were then averaged to reflect the representative activity of the triceps surae (TS) muscle group (27). The antagonist tibialis anterior (TA) EMG data were processed and normalized using the same method.

Muscle and tendon length and stiffness

Motion analysis

Movement of the ankle in the dynamometer footplate was recorded using a real-time motion analysis system with three infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) operating Track Manager 3D (v.1. 8.226, Qualisys) software. Infrared reflective markers were placed over the insertion of the Achilles at the calcaneus (see Fig. 4; marker A), over the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (marker B), and over the GM-Achilles MTJ (marker C) with adhesive zinc-oxide hypoechoic tape placed on the skin aligned with this marker. Raw coordinate data were sampled at 100-Hz and smoothed using a 100-ms averaging window prior to the calculation of Achilles tendon and GM muscle lengths.
The GM-Achilles MTJ was identified (see Fig. 5) using real-time ultrasound video imaging (LOGIQ Book XP, General Electric, Bedford, UK) using a wide-band linear probe (8L-RS, General Electric) with a 39 mm wide field of view and coupling gel (Ultrasound gel, Dahlhausen, Cologne, Germany) between the probe and skin. The probe was then affixed perpendicular to the skin to maintain a constant position with zinc-oxide adhesive tape, which ensured consistent imaging of the MTJ and the hypoechoic tape throughout the trial. Ultrasound images were sampled at 28 Hz, and the position of the MTJ was manually digitized (Peak Motus, Englewood, CO) and smoothed using a 100-ms moving average.

Calculations

Motion analysis, ultrasound and dynamometer data were synchronized using a 5-V ascending transistor-transistor logic (TTL) pulse, which triggered the capture of ultrasound data (preceding 27 s of data) and simultaneously placed a marker on the ROM trace on the AcqKnowledge (v3.8.2, Biopac) software. GM muscle length was calculated as the distance between reflective markers B and C, plus the distance from actual MTJ position (determined with ultrasound; see Fig. 5). Tendon length was calculated as the distance between reflective markers A and C, minus the distance from actual MTJ position (27). Tendon stiffness was calculated by dividing tendon length change by the change in ankle moment.

Data Analysis

All data were analyzed using SPSS statistical software (v.11.5; LEAD Technologies, Chicago, IL); group data reported are means ± SE, change data reported are means ± SD. The study protocol included two interventions, isometric contractions (MVICs) and static stretches. Paired t-tests were used to test for differences in 1) peak isometric moment and
EMG, and 2) muscle and tendon length and stiffness between the first and sixth isometric contraction. Separate analyses of variance (ANOVA) with repeated measures were used to test for differences in 1) concentric and passive plantar flexor moment, 2) peak triceps surae amplitude (EMG), and 3) muscle and tendon length and stiffness post-MVICs. Pearson’s product moment correlation was used to determine relationship between post-MVICs reductions in moment and changes in EMG amplitude.

Following the static stretch intervention, separate analyses of variance (ANOVA) with repeated measures were used to test for differences in 1) peak concentric and passive plantar flexor moment, 2) peak triceps surae amplitude (EMG), and 3) muscle and tendon length and tendon stiffness. Post-hoc t-tests with Bonferroni correction were used to further examine changes in measures where statistical significance was reached. Statistical significance for all tests was accepted at $P < 0.05$.

Reliability

Previous test-retest reliability was determined from our laboratory (27) by calculating the intraclass correlation coefficients (ICC) for intra-tester reliability of the manual digitizing of ultrasound MTJ excursion from the hypoechoic tape ($n = 5$). ICCs ranged from 0.98 to 0.99; no significant difference was detected between mean values ($P > 0.05$). Coefficients of variation (expressed as a percentage of the mean) were also calculated and ranged from 0.3% to 0.4%. Test-retest reliability was calculated for concentric moment, passive moment, muscle and tendon length during the control condition ($n = 15$). The ICC ranged from 0.79 to 0.90, 0.83 to 0.99, and 0.99; and coefficients of variation ranged from 2.3% to 3.2%, 1.8% to 5.2%, 0.2% and 0.4% to 0.5% respectively.
RESULTS

There was no change in active or passive joint moment, EMG amplitude, tendon and muscle length or tendon stiffness after 5 min of rest within the control condition ($P > 0.05$). The current study protocol included two interventions: 1) 6 ramped maximal voluntary isometric contractions (MVICs) followed by 2) 3 min of passive static stretching. For clarity, the results have been separated into two sections in line with these interventions.

Isometric intervention

There was no significant difference in peak isometric joint moment between the first and sixth isometric contractions ($P > 0.05$) indicating that fatigue was not induced. A significant increase in Sol EMG amplitude was detected (15.5%; $P < 0.05$), however no change in EMG was detected in the other triceps surae muscles (GL, GM) or when EMG was averaged across the muscles (3.5%; $P > 0.05$). No change in TA EMG was observed, indicating that co-activity of the antagonist muscle was unchanged.

Significant reductions (mean = 11.5 ± 1.3%) were detected in concentric moment at 50% (12.9%; $P < 0.05$), 70% (11.4%; $P < 0.01$) and 90% (10.4%; $P < 0.01$) of ROM following the MVIC intervention (see Fig. 6). There was a significant decrease in peak TS EMG amplitude (21.0 ± 0.3%) observed after MVIC intervention at 50% (20.8%; $P < 0.01$), 70% (20.7%; $P < 0.01$) and 90% (21.3%; $P < 0.01$) of ROM during the concentric contractions (see Fig. 7). Similar reductions ($P < 0.01$) in all EMG amplitudes (GL mean = 23.5 ± 2.5%; GM mean 22.6 ± 3.0%; Sol mean = 16.3 ± 1.8%) were detected. A significant reduction in TA EMG amplitude (13.7 ± 1.8%; $P < 0.05$) was also detected, suggesting that the MVIC intervention also decreased muscular co-activity.
Pearson’s product moment correlations computed between reductions in triceps surae EMG and decreases in joint moment were significant at 50% ($r = 0.90; P < 0.01$), 70% ($r = 0.73; P < 0.01$) and 90% ($r = 0.74; P < 0.01$) of ROM (see Fig. 8), indicating that the subjects who had the greater reductions in EMG amplitude tended also to exhibit a greater loss of active joint moment. Significant correlations were also detected in individual muscle EMG amplitudes and joint moment at all joint angles ($P < 0.01$; data not shown).

Achilles tendon and GM muscle lengths were measured during the first and sixth contractions at 30%, 50%, 70% and 90% of MVIC (determined in the first contraction). Mean data revealed a trend towards a longer tendon (MVIC$_1 = 187 \pm 7$ mm, MVIC$_6 = 190 \pm 7$ mm) and shorter GM muscle length (MVIC$_1 = 243 \pm 5$ mm, MVIC$_6 = 241 \pm 5$ mm) at all MVIC percentages, which became significant (1.2%; $P < 0.01$) at 90%$_{MVIC}$, and resulted in the muscle operating at a shorter length during the sixth isometric contraction.

Following the MVIC intervention, no significant differences in muscle or tendon length existed at any ROM during the concentric trial ($P > 0.05$) indicating that the muscle operating length had not changed during this contraction mode. However, a significant reduction in tendon stiffness from $9.1 \pm 1.0$ Nm/mm pre-MVIC to $8.2 \pm 0.9$ Nm/mm post-MVIC ($10.9 \pm 1.0\%; P < 0.01$) was observed at 90% of ROM (see Fig. 9) during the concentric contraction as the magnitude of length change of the tendon (pre-MVIC $= 12.7 \pm 3.3$ mm; post-MVIC $= 12.9 \pm 4.1$ mm) was similar despite the lower muscle force.

Following the MVIC intervention, significant reductions in passive moment (4.9 $\pm$ 0.7%) were detected at 50% (5.6%; $P < 0.05$), 70% (4.8%; $P < 0.01$) and 90% (4.2%; $P < 0.01$) of
ROM (see Fig. 10). No significant differences were found in Achilles tendon or GM muscle lengths ($P > 0.05$), however there were consistent trends toward increased Achilles tendon length (under significantly less force) and decreased GM muscle length at all ROMs post-MVICs.

Stretch Intervention

There was no change ($P > 0.05$) in concentric joint moment or EMG from pre- to post-stretch, indicating that the stretch protocol did not impact upon force production or neuromuscular activity. Peak concentric moment increased significantly at 30 min post-stretch at 70% and 90% of ROM when compared to immediately post-stretch data (see Fig. 6); and was no longer significantly depressed relative to baseline (4.7 ± 2.4%). Peak EMG amplitudes in GM, GL, Sol and TS (see Fig. 7) increased significantly at 30 min post-stretch at 70% and 90% of ROM when compared to immediately post-stretch data, and were no longer significantly depressed relative to baseline (GM = 7.3 ± 3.0%; GL = 4.8 ± 4.4%; Sol = 2.3 ± 4.8; TS = 3.2 ± 1.1%). No significant recovery in moment or EMG was detected at 50% of ROM ($P > 0.05$).

Pearson’s product moment correlations computed between the recovery in triceps surae EMG and the recovery in concentric joint moment were significant at 70% ($r = 0.63; P < 0.05$) and 90% ($r = 0.73; P < 0.01$) of ROM, with the subjects who achieved greater recovery in EMG tending also to exhibit a greater recovery of active joint moment. Significant correlations were also detected in individual muscle EMG amplitudes and joint moment ($P < 0.05$; data not shown).
During the concentric trials, no significant change in GM muscle length, Achilles tendon length, or Achilles tendon stiffness (see Fig. 9) was detected post-stretch at any joint angle, indicating that muscle operating at these joint angles did not change post-stretch. Also there was no significant change in passive moment (see Fig. 10), Achilles tendon stiffness and length, or GM muscle length ($P < 0.05$) at any joint angle during the passive trials. Thus there was no significant recovery in these variables after 30 min.

DISCUSSION

The aims of the current research were twofold; first to determine the effects of six maximal voluntary isometric contractions (MVICs) on Achilles tendon stiffness, passive and concentric force production and neuromuscular activity (EMG), and second, to determine whether there were any additional changes in these measures following 3 min of static stretch. Following the MVIC intervention active (concentric) joint moment was significantly lower at all joint angles ($\text{mean} = 11.5 \pm 1.3\%; P < 0.01$) and this was accompanied by, and correlated with ($r = 0.90; P < 0.01$), reduced triceps surae EMG amplitude ($\text{mean} = 21.0 \pm 0.3\%; P < 0.01$). These changes could not be attributed to the initial concentric contraction as no change in any measure ($P > 0.05$) was detected under control conditions where the concentric protocol was employed followed by 5 min of rest before repeating the concentric trial. Also, metabolic muscle fatigue could not account for the force depression as there was no change in isometric joint moment between the first and sixth (last) isometric contractions.

The MVIC intervention also resulted in a significant decrease in passive joint moment ($\text{mean}$
indicating a decreased stiffness of the muscle-tendon complex (MTC) or joint capsule. The findings of a trend towards a longer tendon length during the passive joint rotation and a significantly reduced tendon stiffness measured during the maximal concentric contraction (mean = 10.7 ± 1.3%; \(P < 0.01\)) is suggestive that the reduced passive joint moment was attributable to a reduction in tendon stiffness. Importantly, the stretch intervention imposed after the MVIC intervention did not cause any further change in muscle-tendon properties, neuromuscular activity or force generating capacity of the triceps surae muscles \((P > 0.05)\). Finally, significant increases were detected in active joint moment and EMG amplitude in the most dorsiflexed positions after 30 min of passive recovery allowed after the stretch intervention, but passive moment and Achilles tendon stiffness remained depressed.

The present data indicate that active concentric plantar flexor moment decreases following the performance of maximal isometric contractions (MVICs), which are accompanied by, and highly correlated with, decreases in EMG amplitude. In the present study we found a linear relationship between the decreases in force and EMG amplitude such that over 81% of the variability in moment changes was explained by the changes in EMG amplitude. Although the strongest correlation was found when the EMG amplitude of the three triceps surae muscles were averaged \((r = 0.90; P < 0.01)\), significant correlations were also found for GM \((r = 0.63; P < 0.05)\), GL \((r = 0.68; P < 0.01)\) and Sol \((r = 0.85; P < 0.01)\) muscles individually. The reduction in EMG measured in the concentric contractions is intriguing because no decrease in force or EMG amplitude was seen during the isometric contractions. Typically reductions in EMG amplitude result from decreased central neural drive \((14, 15, 20)\) or peripheral inhibition or disfacilitation of the \(\alpha\)-motoneuron pool by associated muscle afferents \((4)\). The present data are suggestive of central rather than peripheral mechanisms
influencing EMG amplitude since peripheral alterations would be expected to influence EMG during both the isometric and concentric contractions. Furthermore, peripheral inhibition of the α motoneuron pool from increased golgi tendon organ (GTO) activity should increase co-
activity of the TA muscle. However, significant reductions were detected in TA EMG lending further support to central rather than peripheral mechanisms being responsible for the reductions in neuromuscular activity. We are unaware of other studies that have reported contraction mode dependent changes in muscle force and EMG, therefore further research is required as these results may have practical implications for muscle performances that rely on concentric force production.

Despite the strong relationship, the reduced active joint moment could not be completely explained by the decrease in EMG. Another possible mechanism of force reduction is a change in tendon stiffness, as this could result in a change in muscle operating length (32); the leftward shift in its force-length curve would cause the plantar flexors to operate further down their ascending limb (33, 34). This hypothesis appears to be supported by the significant reduction in tendon stiffness detected during the concentric trial post-MVICs in the present study (10.7%). However, ultrasound imaging of the MTJ revealed no change in tendon length (< 0.1 mm) or muscle operating length post-MVICs during the concentric trials, despite an 11.5% reduction in concentric moment. Thus, the decrease in force production combined with a greater tendon compliance allowed the muscle to work at the same length at a given joint angle so a leftward shift in the force-length curve was not present, and cannot explain the reduced muscle force. To ensure the present methods could detect a change in tendon length, a linear regression was employed to model tendon deformation during a ramped MVIC. This model calculated a projected 2 mm reduction in tendon deformation from the 11.5% reduction in joint moment seen in the present data; we
are confident that the present methods would be able to detect this small, but significant change.

Although altered force-length characteristics were clearly not a mechanism for the force depression, the decreased tendon stiffness might have functional consequences. Tendon structures account for 42.5 – 60% of total work done during the concentric phase (8, 10, 51) of stretch-shortening cycle (SSC) exercise, where the concentric action of the MTC is preceded by eccentric loading. The stiffness of tendinous structures has also been significantly correlated with rate of force development, maximal isometric force and vertical jump height (11, 18, 32, 54) and inversely correlated with neuromechanical delay (17, 22, 30). Therefore, a reduction in the stiffness of the tendon may impede fast force transmission through the tendon onto the bone, thus attenuating joint moment. However, a more compliant tendon would store more energy and transfer less force to the musculature during eccentric loading of the MTC, cited as a factor related to injury (24). Therefore, the decrease in tendon stiffness may provide a prophylactic effect and reduce the risk of muscle strain injury. It should also be stated that if a decrease in stiffness is reflective of a decreased tensile strength of the tissue, then there might be an increase in the risk of tendon strain injury. However, these two properties are not always functionally related, and strong muscular contractions are commonly performed in athletic populations and with a paucity of tendon rupture reported in the literature (in the healthy tendon), we believe this is unlikely. Nonetheless, the reduced maximal joint moment associated with this intervention may increase the injury risk, as strength has also been cited within the etiology of muscle strain injury (46). Thus, the impact of these contractions on MTC injury risk is unclear.
The isometric contraction protocol was chosen to ensure a consistent amount of work was completed between subjects and similar strain was applied to the tendinous tissues to enable valid post-intervention statistical analyses. However, a range of maximal contraction modes are commonly performed during athletic pre-performance routines, and rapid eccentric or concentric loading of the MTC may induce different effects to the ones observed following the maximal isometric contractions performed in the present study. Therefore, these data cannot be generalized to athletic populations where different pre-performance regimes may be used. Further research should be conducted to determine the effects of contraction mode on force production and the mechanical properties of the MTC.

Notwithstanding the possible differences in the modality of the pre-performance contractions employed in the present study, the examination of the effects of stretch after the performance of a series of intense muscle contractions is important given that many individuals perform a progressively intense warm-up in addition to stretching prior to maximal exercise bouts. Some research studies (7, 13-15, 37, 47) but not others (35, 36, 41) have included maximal contractions in the warm-up or as part of the experimental model prior to testing the effects of static stretch on joint moment. However, to our knowledge no studies have examined whether the inclusion of these contractions might mitigate or compound the effects of subsequent stretching. Clearly the MVIC intervention resulted in significant decreases in active (concentric) and passive joint moment. A novel finding of the present study was that no further reductions in concentric joint moment, EMG, passive moment or MTC stiffness were subsequently found post-stretch. This is despite using a stretch protocol that has previously resulted in substantial changes in these variables (27). Thus, the negative effects of stretch appear to be dependent on whether MVICs are performed before them. The impact of prior maximal isometric contractions not only has important methodological implications
for stretch-based studies but also has significant practical implications for athletes where
maximal concentric contractions are essential to performance. Significant improvements in
muscle performance (6, 23, 45) have been reported following the implementation of prior
maximal isometric contractions. Again, further research is required to determine whether
similar reductions are realized when other contraction modes (e.g. concentric) are used prior
to stretch and whether they also mitigate the subsequent effects of stretch.

Interestingly, after 30 min of rest there was a significant recovery of concentric joint moment
(64% recovery of the deficit) in the most dorsiflexed positions. This was accompanied by,
and correlated with ($r = 0.73; P < 0.01$) significant recovery in EMG amplitude (82%); such
that force and EMG were no longer significantly reduced compared to baseline levels.
However, tendon stiffness and passive joint moment remained significantly depressed.
Previously, stretch-based interventions (19, 27) have resulted in similar recovery patterns in
active joint moment and EMG amplitude, however passive joint moment tended to fully
recover to baseline (19, 27). These disparate results in the recovery of passive moment may
be explained by the different mechanical effects of these interventions to MTC tissues. The
present data clearly indicate that the reduction in passive joint moment after MVICs was
attributable to a decreased tendon stiffness, which remained apparent after 30 min. However,
moderate duration (< 5 min) stretch interventions (27, 41) have resulted in no change in
tendon stiffness, with decreases in passive moment suggested to originate from a decreased
muscle stiffness. Furthermore, the reduction in muscle stiffness appears to be transient,
dissipating after 30 min indicting only a temporary effect of stretch to muscle stiffness. The
continued depression of tendon stiffness after 30 min following MVICs, while concentric
force recovered, may have important prophylactic injury implications as tendon stiffness (24)
and muscle force (46) have been implicated with muscle strain injury risk. Further research
is required to determine the long-term effects of MVICs to force production, MTC mechanics and muscle strain injury risk.

In summary, the present study is the first to specifically examine the effects of pre-stretch maximal isometric contractions (MVICs) as part of a comprehensive warm-up in order to determine the subsequent effect of stretch on joint moment, neuromuscular activity and MTC mechanics. Significant reductions in tendon stiffness, active (concentric) and passive plantar flexor joint moment and EMG amplitude occurred following the MVIC intervention. This was unlikely to result from local muscle fatigue as there was no reduction in isometric force. Significant correlations were found between the reductions in joint moment and decreases in EMG amplitude, indicating that a substantial proportion of the reduction in force (~81%) could be attributed to a reduced neuromuscular activity. Although tendon stiffness was decreased, reductions in muscle operating length were clearly not a mechanism implicated with reductions in active force. An important finding of the present study was that no significant decrease in active or passive joint moment, EMG or MTC mechanics was evident when the MTC was then stretched passively for 3-min. This suggests that the use of MVICs in a warm-up routine might mitigate the widely reported negative effects of moderate duration stretch (< 3 min) within the plantar flexors but only because concentric force was already depressed. This finding has important implications both for research and warm-up intervention designs. The significant increase in active joint moment and EMG amplitude found 30 min after stretch suggests that physical tasks requiring high levels of plantar flexor muscle force are unlikely to be compromised at this time. However, decreased tendon stiffness and reduced passive joint moment remained, which may have important implications to injury risk for the triceps surae-Achilles muscle-tendon complex.
REFERENCES


FIGURE LEGENDS

Fig. 1. Timeline of the MVICs and stretch interventions. Five minutes after completing the warm-up, passive and active concentric trials were conducted, followed 2 min later by the MVIC intervention. After two minutes the passive and active trials were repeated to determine the effect of the MVIC intervention. The stretch intervention was initiated 2 min later with the passive and active trials repeated 2 min and 30 min post-stretch to determine the effects of the stretch intervention.

Fig. 2. Pre- and post-MVIC moment data from one subject’s trial. Joint moment was recorded during the concentric trials with a clear reduction in peak isometric joint moment apparent prior to the release of the dynamometer. This reduced joint moment remained throughout ankle ROM at all joint angles following the MVIC intervention.

Fig. 3. Concentric and isometric joint moment data from one subject’s trial. No change in isometric joint moment was apparent between the six isometric contractions indicating that fatigue had not been induced. Concentric joint moment was significantly reduced following the MVIC intervention but no change was apparent after the subsequent stretch intervention; concentric joint moment recovered 30 min later. #Significant to $P < 0.01$ compared to baseline.

Fig. 4. Reflective marker (motion analysis) and ultrasound probe positioning. Achilles tendon length was estimated from the distance between the reflective markers placed over the insertion of the Achilles on the calcaneus (marker A) and hypoechoic tape (marker C) placed over the GM-Achilles MTJ. Gastrocnemius medialis (GM) muscle length was estimated.
from the distance between the reflective markers placed over the origin of the GM muscle on the medial femoral epicondyle (marker B) and hypoechoic tape (marker C) placed over the GM-Achilles MTJ.

**Fig. 5.** Ultrasound image of the GM-Achilles MTJ. The position and displacement of the GM-Achilles MTJ was recorded using real-time ultrasound imaging. The MTJ was identified as the point where the deep and superficial GM aponeuroses merged with the Achilles tendon. Displacement of the MTJ from the hypoechoic area was synchronized with motion analysis data to calculate GM muscle and Achilles tendon lengths.

**Fig. 6.** Normalized joint moment during maximal concentric plantar flexion. Subjects performed a maximal concentric plantar flexion contraction through full ROM before and after six MVIC contractions; and 2 min and 30 min after three 60-s stretches. Significant reductions were seen post-MVICs at all joint angles and significant increases were seen after 30 min post-stretch. *Significant to $P < 0.05$, $^\#$Significant to $P < 0.01$ compared to baseline.

**Fig. 7.** TS EMG amplitude during maximal concentric plantar flexion. Neuromuscular activity (EMG) was examined before and after six MVIC contractions; and 2 min and 30 min after three 60 s stretches. Significant reductions were seen post-MVICs at all joint angles and significant increases were seen after 30 min post-stretch. *Significant to $P < 0.05$, $^\#$Significant to $P < 0.01$ compared to baseline.

**Fig. 8.** Correlation between post-MVICs reductions in moment and TS EMG at 50% of ROM). Significant correlations were detected between reductions in all EMG amplitudes and moment deficits ($r = 0.90; P < 0.01$) at all joint angles measured.
Fig. 9. Achilles tendon stiffness during maximal concentric plantar flexion. Achilles tendon stiffness was significantly reduced following the MVIC intervention at 90% of ROM (10.9 ± 1.0%). No significant change in tendon stiffness occurred post-stretch indicating that stretch did not impact on tendon stiffness and that no significant recovery occurred 30 min post-stretch. *Significant to $P < 0.05$, #Significant to $P < 0.01$ compared to baseline.

Fig. 10. Joint moment during passive dorsiflexion following the MVIC intervention. Significant reductions in joint moment were seen post-MVICs at all joint angles. No significant change in moment occurred post-stretch indicating that stretch did not impact on passive moment and that no significant recovery occurred 30 min post-stretch. *Significant to $P < 0.05$, #Significant to $P < 0.01$ compared to baseline.
STUDY PROTOCOL

Warm-up | Passive | Active | Passive | Active | Passive | Passive | Passive | Active
0 | 10 | 12 | 14 | 19 | 21 | 23 | 30 | 32 | 58 | 60

TIME (min)

- 6 × MVIC
- 3 × 60-s stretch
Joint Moment (Nm) vs. Time (min)

30 min stretch

30 min rest

MVICs
Reduction in Joint Moment (%MVIC) vs. Reduction in Triceps Surae EMG (%MVIC)

$R^2 = 0.81$
Achilles Tendon Stiffness (Nm/mm)

- Pre-MVIC
- Post-MVIC
- Post-stretch
- 30 min post-stretch

Legend:
- #