TITLE

Effects of contract-relax, static stretching, and isometric contractions on muscle-tendon mechanics

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ABSTRACT

Introduction: The loading characteristics of stretching techniques likely influence the specific mechanisms responsible for acute increases in range of motion (ROM). Therefore, the effects of a version of contract-relax proprioceptive neuromuscular facilitation (CR) stretching, static stretching (SS) and maximal isometric contraction (Iso) interventions were studied in 17 healthy human volunteers. **Methods:** Passive ankle moment was recorded on an isokinetic dynamometer with electromyographic (EMG) recording from the triceps surae, simultaneous real-time motion analysis, and ultrasound imaging recorded gastrocnemius medialis muscle and Achilles tendon elongation. The subjects then performed each intervention randomly on separate days before reassessment. **Results:** Significant increases in dorsiflexion ROM (2.5-5.3°; *P<0.01*) and reductions in whole muscle-tendon stiffness (10.1-21.0%; *P<0.01*) occurred in all conditions, with significantly greater changes detected following CR (*P<0.05*). Significant reductions in tendon stiffness were observed after CR and Iso (17.7-22.1%; *P<0.01*) but not after SS (*P>0.05*), while significant reductions in muscle stiffness occurred after CR and SS (16.0-20.5%; *P<0.01*) but not after Iso (*P>0.05*). Increases in peak passive moment (stretch tolerance) occurred after Iso (6.8%; *P<0.*05), CR (10.6%; *P=0.08*) and SS (5.2%; *P=0.08*); no difference in the changes between conditions was found (*P>0.05*). Significant correlations (r_s = 0.69-0.82; *P<0.01*) were observed between changes in peak passive moment and maximum ROM in all conditions. **Conclusion:** While similar ROM increases occurred after isometric contractions and static stretching, changes in muscle and tendon stiffness were distinct. Concomitant reductions in muscle and tendon stiffness after CR suggest a broader adaptive response that likely explains its superior efficacy to acutely increase ROM. While mechanical changes appear tissue-specific between interventions, similar increases in stretch tolerance after all interventions were strongly correlated with the changes in ROM.

Keywords: Proprioceptive neuromuscular facilitation, range of motion, tendon stiffness, stretch tolerance, ultrasonography.

INTRODUCTION

Both the maximum joint range of motion (ROM) and resistance to joint rotation within that range (i.e. resistance to stretch) are important physical characteristics influencing the capacity to perform activities of daily living and athletic tasks (34), and are affected considerably by aging (4) and disease (10). Nonetheless, although muscle stretching is commonly practiced, relatively little is known about the underlying mechanisms that influence ROM in particular or its change in response to acute and chronic muscle stretching training. Despite static stretching being the most commonly used stretching mode, proprioceptive neuromuscular facilitation (PNF) stretches are regularly reported as being more effective for increasing ROM (25, 27). The distinctive characteristic of PNF is that a brief (sometimes maximal) isometric contraction is performed while the muscle held on stretch (1). Two common methods of PNF stretching include the contract-relax (CR) and contract-relax agonist contract (CRAC) techniques (37). The CR method includes a static stretching phase followed by an intense isometric contraction of the stretched muscle, immediately followed by a further stretching phase, whereas the CRAC method requires an additional contraction of the agonist (i.e. opposing the muscle group being stretched) muscle during the stretch, prior to the subsequent additional stretch of the target muscle. However, despite these techniques being commonly employed in clinical environments to achieve rapid increases in ROM they are not commonly used in athletic warm-up routines, possibly because it normally requires an assisting partner, may be painful, and may pose a greater muscle strain injury risk compared with static stretching (5). Despite their efficacy, limited data exist describing the specific underlying mechanisms associated with

changes in ROM following these modes of stretch, which is problematic as determining mechanisms may allow researchers to determine a priori whether these interventions may be useful in different clinical populations, to understand why such stretch interventions elicit different responses in different individuals, and offer information that allows us to modify the technique to optimize/improve both acute and chronic responses to the stretching.

Two neuromuscular mechanisms have been traditionally theorized to underpin the significant improvements in ROM achieved through CR stretching: autogenic inhibition and gate control theory (13). Autogenic inhibition may occur during the contraction phase of CR as increased activity from type Ib muscle afferent fibers within the golgi tendon organs (GTO) act to hyperpolarize the dendritic ends of spinal α-motoneurons of the stretched muscle. This output could reduce the effectiveness of homonymous type Ia muscle afferent output during stretch, inhibiting the activation of the α-motoneuron pool, possibly enabling further increases in ROM (1, 36). Although intuitive that a reduction in α-motoneuron pool activity may enable further increases in ROM, there is no direct evidence of a causal relationship. However, GTO activity is substantially reduced or ceases once the contraction has terminated, with several studies reporting increased resting electromyographic (EMG) activity immediately following the contraction phase of a CR stretch (25, 32). Thus, autogenic inhibition is unlikely to be the primary underlying mechanism explaining either increases in ROM or the superiority of CR stretching in increasing ROM above other stretching modalities (37). While recent reviews have generated ambiguity over the involvement of autogenic inhibition (13, 37), other inhibitory neurological mechanisms may explain CR stretching's efficacy (38). Gate control theory suggests that an increased output from type III muscle afferents during the contraction phase of CR stretching could inhibit pain perception (28). Pressure receptors have larger myelinated neurons and connect to the same spinal interneurons within the spinal horn as un-

104 myelinated nociceptive fibers (type IV afferents) (31), therefore increased activity could
105 theoretically dampen pain perception and enable further increases in ROM (37). However,
106 increased peak passive torque at full volitional ROM, indicative of dampened pain perception
107 or increased stretch tolerance (i.e. the capacity to tolerate increased loading prior to terminating
108 the stretch), has also been commonly reported following static stretching (27, 39). Thus, an
109 increase in the ROM at which the stretch sensation, discomfort or pain perception is perceived
110 or tolerated (i.e. stretch tolerance) is a common characteristic across stretch modalities and may
111 not explain the superior ROM outcomes associated with CR stretching.
112
113 Acute increases in ROM following a single static (passive) muscle stretching session are
114 frequently reported with concomitant reductions in muscle-tendon complex (MTC) stiffness
115 (16, 17, 22, 24, 26, 33), a reduced neuromuscular reflex response (2, 3), and an increased stretch
116 tolerance (27, 39). Therefore, despite the relatively lower levels of tissue loading imposed
117 during static stretch compared with CR, mechanical changes in musculotendinous tissues are
118 notable and may underpin the increases in ROM, or at least influence receptor activity and/or
119 these afferent pathways. While dynamometry-based passive moment data are often used in the
120 quantification of MTC stiffness, ultrasonography provides the opportunity to examine the
121 influence of stretch on specific tissues, although relatively few studies have employed this
122 methodology *in vivo* during muscle stretching. During stretching, both muscular and tendinous
123 tissues experience deformation (i.e. strain), however moderate-duration static stretching (3-5
124 min) has been reported to reduce muscle stiffness without influencing Achilles tendon stiffness
125 (17, 33); which is indicative of a muscle-based response underpinning increases in ROM.
126 However, acute reductions in tendon stiffness have been reported following repeated maximal
127 isometric (18, 20) and concentric (19) contractions where relatively greater tissue loading
128 occurs within the tendon. Collectively, data from these studies are suggestive that the intensity

129 and location of tissue strain may influence the change in tendon stiffness, and thus the specific
130 site of mechanical changes in the MTC. It may be hypothesized, therefore, that CR stretching
131 might impose significant strain on both the muscle (because of the MTC stretch) and tendon
132 (during the muscle contraction phase), offering a unique stimulus for decreases in both muscle
133 and tendon stiffness. These may be directly (by reducing stiffness) or indirectly (through
134 alterations in afferent feedback) associated with the reductions in resistance to stretch and
135 increased ROM after CR stretching. Despite this possibility, testing of mechanical theories
136 associated with the increased ROM following CR have been limited to examinations of
137 viscoelastic stress relaxation and creep responses (13) with no studies examining the acute
138 effects of CR stretching on muscle and tendon stiffness.
139
140 The aims of the present study were to examine the influence of a version of CR stretching (i.e.
141 MTC stretch plus muscle contraction), static stretching (i.e. MTC stretch only), and maximal
142 isometric contractions (i.e. muscle contraction causing tendon stretch) on dorsiflexion ROM,
143 the slope of the passive joint moment curve (MTC stiffness), maximal passive joint moment at
144 full volitional ROM (stretch tolerance), gastrocnemius medialis (GM) muscle stiffness and
145 triceps surae EMG activity measured during a passive joint stretch. The acute effects of these
146 interventions on Achilles tendon stiffness, maximal isometric plantar flexor joint moment and
147 peak triceps surae EMG activity during a maximal isometric contraction were also measured.
148 We tested the hypothesis that CR stretching would produce significantly greater increases in
149 ROM and stretch tolerance whilst reducing muscle and tendon stiffness whereas static
150 stretching would influence only muscle stiffness and isometric contractions would influence
151 only tendon stiffness.
152
153 **MATERIALS & METHODS**

154 Subjects

155 Seventeen recreationally active participants (9 women, 8 men; age = 25.6 ± (SD) 8.8 yr, mass
156 = 74.8 ± 11.8 kg, height = 1.7 ± 0.1 m) with no recent history of lower limb injury or illness
157 volunteered for the study after providing written and informed consent. The subjects were
158 asked to avoid intense exercise, muscle stretching and stimulant use for 48 hr prior to testing.
159 Ethical approval was granted by The University of Northampton's Ethics Committee, and the
160 study was completed in accordance with the Declaration of Helsinki.

161

162 Protocol

163 *Overview*

164 The subjects were fully familiarized with the testing protocols one week prior to data collection
165 and they then visited the laboratory on three further occasions under experimental conditions,
166 with trials conducted in a randomized order separated by one week. During the experimental
167 trials, the subjects performed a warm-up for 5 min on a Monark cycle at 60 rev·min^{-1} with a 1
168 kg resistance load. The subjects were then seated in the chair of an isokinetic dynamometer
169 (Biodex System 3 Pro, IPRS, Suffolk, UK) with the knee fully extended (0°) to ensure all
170 plantar flexor components were influenced by the interventions and contributed significantly
171 to passive and active joint moments (15). The foot was then strapped to the dynamometer
172 footplate in the anatomical position (0°) with the sole of the foot perpendicular to the shank,
173 and with the lateral malleolus aligned with the center of rotation of the dynamometer. The
174 non-elastic Velcro strapping was used to minimize heel displacement from the dynamometer
175 footplate during passive and active trials to provide reliable and valid ROM and passive
176 moment data during the passive trials (33). To confirm that the degree of ankle fixation did
177 not substantially influence the passive moment data during the measurements, one highly
178 experienced analyst conducted all trials in order to remove inter-tester variability. To further

confirm the reliability of these methods, day-to-day reliability of passive moment was measured prior to each intervention (pre-test data); analysis of the data indicated very high reliability (ICC = 0.95, SE = 3.0%). Subjects then performed a maximal isometric plantar flexor contraction to determine maximal isometric joint moment and peak EMG activity (RMS amplitude, described later). This was followed two minutes later by three passive dorsiflexion rotations initiated from 20° plantar flexion through to full dorsiflexion at 0.087 rad·s^{-1} (5°·s^{-1}) to determine dorsiflexion range of motion (ROM) and peak passive moment at full ROM (stretch tolerance). Two minutes after completing the passive trials, the subjects performed one of three interventions (contract-relax [CR] stretches, static stretches [SS], or isometric contractions [Iso]; specific details provided below). Two minutes after completing the intervention, the subjects repeated the passive trials and active trials.

Dynamometry data

Subjects were seated in the dynamometer chair with the hip flexed to 55°, knee fully extended (0°), and ankle in the anatomical position (0°). The subjects then produced a ramped maximal isometric plantar flexor contraction with maximal joint moment reached ~3 s after contraction initiation and held for 2 s (i.e. there was a visible plateau in the moment trace), followed by an identical dorsiflexor contraction. The ramped plantar flexor contraction allowed maximum strength to be determined but also enabled tendon deformation to be captured using sonography, which allowed tendon stiffness to be calculated when combined with joint moment data (17-19). To confirm that the loading rate during the ramped contraction did not influence tendon stiffness, the subjects repeated the ramped contractions using visual feedback during the familiarization session until they reliably achieved a linear increase in joint moment reaching MVC after ~3 s. During the ramped contractions in the experimental trials, the time interval between 50-90%MVC (the range over which tendon stiffness was calculated) was

recorded in the pre- and post-intervention sessions. No significant difference (pre = 2.1 ± 0.1 s, post = 2.0 ± 0.1 s; $P > 0.05$) in the 50-90%MVC interval time (indicative of the tendon loading rate) was found. Two minutes after completing the isometric tests, the subjects' ankles were passively rotated through their full ROM at 0.087 $rad \cdot s^{-1}$ ($5° \cdot s^{-1}$) until they volitionally terminated the rotation by pressing a hand-held release button at the point of discomfort (6, 7). The passive rotations were performed three times with the slope of the passive moment curve (indicative of MTC stiffness), peak passive moment (stretch tolerance), and ROM data measured from the third trial to ensure muscular thixotropic properties did not influence the data. The slope of the passive moment curve was calculated as the change in plantar flexor moment through the final 10° of dorsiflexion (in the linear portion of the passive moment curve) in the pre-stretching trials; and these identical joint angles were used in post-stretching analysis. Joint moment and angle data were directed from the dynamometer to a high level transducer (model HLT100C, Biopac, Goleta, CA) before analog-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 (v4.1, Biopac) and filtered with a zero lag, 6-Hz Butterworth low-pass filter prior to maximum ROM and passive joint moment being determined. Peak passive moment was measured within a 250-ms epoch at full volitional ROM.

Electromyogram (EMG) recording

Electrode site preparation, electrode placement, and EMG sampling, processing and normalization methods were completed as described previously (17-19). EMG activity of gastrocnemius medialis (GM), gastrocnemius lateralis (GL), soleus (Sol) and tibialis anterior (TA) were monitored using skin-mounted bipolar double differential active electrodes (model MP-2A, Linton, Norfolk, UK). The EMG signals were pre-amplified by the electrode (gain =

300, input impedance = 10 GΩ, CMRR = > 100 dB at 65 Hz) and then directed to a high level transducer (model HLT100C, Biopac) before analog-to-digital conversion at a 2000-Hz sampling rate (model MP150 Data Acquisition, Biopac). The EMG signals were then directed to a personal computer running AcqKnowledge software (v4.1, Biopac), filtered using a 20- to 500-Hz band-pass filter, and then converted to root mean squared (RMS) EMG with a 250-ms sample window. The RMS EMG data were then normalized as a percentage of the peak amplitude recorded during the first maximal voluntary isometric contraction. The normalized EMG amplitude was used as a measure of neuromuscular activity during the active trials (volitional activity) and at the end of ROM during the passive trials (reflexive activity) with the antagonist tibialis anterior (TA) EMG data processed and normalized using the same method. During the active and passive trials, EMG activity was measured within a 250-ms epoch at peak joint moment and full volitional ROM, respectively.

Muscle and tendon stiffness and elongation

Motion analysis

Real-time motion analysis using four infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) and operating Track Manager 3D software (v.2.0, Qualisys) were used to record the movement of infrared reflective markers during the trials. Using methods previously described (17-19) to calculate Achilles tendon and GM muscle length and elongation, reflective markers were placed over the insertion of the Achilles at the calcaneus (see Figure 1; *marker A*) and on the distal edge of the ultrasound probe positioned over the GM-Achilles muscle-tendon junction (MTJ) (*marker B*). A third marker was placed over the origin of the medial head of the gastrocnemius at the medial femoral epicondyle (*marker C*). 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.

Ultrasound

Real-time ultrasound images (LOGIQ Book XP, General Electric, Bedford, UK) were recorded at 28 Hz 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 was used to image the GM-Achilles MTJ (see Figure 2). The probe was positioned perpendicular to the skin with zinc-oxide adhesive tape to ensure consistent imaging of the MTJ during the trials. The distance between the MTJ and distal edge of the ultrasound image was manually digitized (LOGIQ Book XP, General Electric).

Calculations

A 5-V ascending transistor-transistor logic (TTL) pulse triggered the capture of ultrasound data (preceding 15 s of data), ended the capture of motion analysis data and simultaneously placed a pulse trace on the AcqKnowledge (v4.1, Biopac) software to synchronize motion analysis, ultrasound and dynamometer data. Tendon length was calculated as the sum of the distance between reflective markers A and B (using motion analysis) and the distance from actual MTJ position to the distal border of the image (using ultrasound), in a method similar to that previously reported (17), where the MTJ distance was measured to a hypoechoic area (beneath tape affixed to the skin) in the image. Digitizing the position of the MTJ to the edge of the ultrasound image was employed as the ultrasound probe was permanently fixed to the skin for the duration of the test, thus reliable positioning was ensured. Furthermore, removal of the tape affixed to the skin eliminated the hypoechoic area in the ultrasound image enabling the MTJ to remain clearly visible throughout the recording, improving MTJ digitization. Tendon stiffness was calculated as the change in plantar flexor moment from 50-90%MVIC divided by the change in tendon length ($Nm \cdot mm^{-1}$). Muscle length was calculated as the distance between

279 reflective markers B and C (using motion analysis) minus the distance from actual MTJ
280 position to the distal border of the image. Muscle stiffness was calculated as the change in
281 plantar flexor moment through 10° of dorsiflexion (in the linear portion of the passive moment
282 curve) divided by the change in muscle length ($Nm \cdot mm^{-1}$).
283
284 *Interventions*
285 Two minutes after completing the passive ROM trials, the subjects performed one of three
286 interventions. During the static stretch condition (SS), the ankle was passively rotated at 0.087
287 $rad \cdot s^{-1}$ through to full ROM until reaching the point of discomfort, a position regularly used in
288 stretch studies (16, 17). The movement velocity was too slow to elicit a significant myotatic
289 stretch reflex response (29, 30), which ensured that full ROM was achieved and a substantial
290 stress was applied to the MTC. This ensured that the moment recorded was considered
291 reflective of the passive properties of the plantar flexors. The subject's ankle was held in the
292 stretched position for 15 s and then released, returning the foot to 20° plantar flexion. The
293 stretch protocol was then repeated three times with 15-s rests, giving a total stretch duration of
294 60 s. Such stretch durations are likely to be achievable in clinical and other contexts, and
295 previous research have shown that significant increases in ROM (35, 40) and decreases in MTC
296 stiffness (16, 40) result from stretches of equal and lesser duration. During subsequent
297 stretches the subject was encouraged to stretch to a greater joint angle to ensure that substantial
298 stress was imposed on the tissues and to more accurately reflect current stretching practices.
299 The contract-relax (CR) condition was performed using similar methods to SS with the
300 exception that the stretch was held passively for 10 s followed immediately by a 5-s ramped
301 maximal isometric contraction. While in traditional CR stretching the muscle would then be
302 immediately stretched to its new ROM before repeating the stretch and contraction phases, in
303 the present study the subject's limb was returned to the anatomical position to allow for similar

rest periods between stretches across interventions. After 15 s of rest, the protocol was repeated three times. During the isometric condition (Iso) the ankle was passively rotated at 0.087 rad·s^{-1} until reaching the anatomical position (0°) where the subject was held for 10 s before performing a 5-s ramped, maximal isometric contraction (i.e. identical to the contraction phase performed during the CR condition). After 15 s rest, the protocol was repeated three times. Two minutes after each intervention, the passive and active tests were repeated to determine the influence of the interventions on dorsiflexion ROM, the slope of the passive joint moment curve (MTC stiffness), maximal passive joint moment (stretch tolerance), Achilles tendon and GM muscle stiffness, and maximal isometric plantar flexor joint moment and triceps surae EMG activity.

Data analysis

All data were analyzed using SPSS statistical software (v.17.0; LEAD Technologies, Chicago, IL); condition data are reported as mean ± SE, and change data are reported as mean ± SD. The study protocol included three interventions; CR, SS and Iso. Normal distribution for pre- and post-group data in all variables was assessed using Kolmogorov-Smirnov and Shapiro-Wilk tests; no significant difference ($P > 0.05$) was detected in any variable indicating that all data sets were normally distributed. Separate repeated measures MANOVA's were used to test for differences between pre- and post-intervention data in: (1) peak isometric moment and EMG, and (2) ROM and peak passive moment (stretch tolerance), and (3) the slope of the passive joint moment curve (MTC stiffness), GM muscle and Achilles tendon stiffness. Where significant differences were detected, separate repeated measures ANOVAs were used to test for differences in absolute change score data between interventions. Post-hoc t-tests with Bonferroni correction were used to further examine changes in measures where statistical significance was reached. Normal distribution was also examined for change score data in all

variables using Kolmogorov-Smirnov and Shapiro-Wilk tests; a significant difference ($P < 0.05$) was detected for changes in ROM but no significant difference ($P > 0.05$) was detected in any other variable. Spearman's rank correlation coefficients (r_s) were computed to quantify the linear relationship between the change in ROM and changes in peak passive moment (stretch tolerance) and the slope of the passive joint moment curve (MTC stiffness) in each condition. Statistical significance for all tests was accepted at $P < 0.05$.

Reliability

Test-retest reliability was determined for peak isometric moment, peak passive moment (stretch tolerance), ROM, slope of the passive moment curve (MTC stiffness), muscle stiffness and tendon stiffness in the pre-test data across conditions. No significant difference was detected between mean values ($P > 0.05$) for any measure; intraclass correlation coefficients (ICC) were 0.89, 0.97, 0.97, 0.95, 0.80, and 0.96. Coefficients of variation and standard error (expressed as a percentage of the mean) were 9.5% (SE = 2.3%), 7.8% (SE = 1.9%), 4.4% (SE = 1.1%), 12.4% (SE = 3.0%), 11.1% (SE = 2.7%), and 4.4% (SE = 1.1%), respectively, for the above variables.

Sample size

Effect sizes (Cohen's D) were calculated from mean changes in variables (ROM, muscle and tendon stiffness, and peak passive moment) from previous studies employing similar methods (17, 18, 33). To ensure adequate statistical power for all analyses, power analysis was conducted for tendon stiffness (the variable with the smallest effect size) using the following parameters (variable = tendon stiffness, power = 0.80, alpha = 0.05, effect size = 0.95, attrition = 20%). The analysis revealed that the initial sample size required for statistical power was 15, thus 20 subjects were recruited to account for possible attrition. Three subjects withdrew

354 from the study with non-related injuries; statistical analyses were conducted on data sets for 17
355 subjects who completed the testing.
356
357 **RESULTS**
358 Range of motion
359 A significant increase in dorsiflexion ROM (see Figure 3) was found after CR (5.3 ± 4.6°; $P <$
360 0.01) and SS (2.6 ± 3.5°; $P < 0.01$) stretching as well as after Iso (2.5 ± 2.2°; $P < 0.01$). A
361 significant difference ($F = 4.3$; $P < 0.05$) was detected in the change in ROM across the three
362 interventions (see Figure 3). Post-hoc analysis revealed significantly greater increases in ROM
363 after CR compared with both SS and Iso ($P < 0.05$) but no difference between SS and Iso (P
364 > 0.05).
365
366 Stretch tolerance
367 A significant increase in peak passive moment (measured at full volitional ROM) was found
368 after Iso (6.8 ± 10.2%; $P < 0.05$) but the change after CR (10.6 ± 18.8%; $P = 0.08$) and SS
369 interventions (5.2 ± 16.8%; $P = 0.08$) did not reach statistical significance. Nonetheless, no
370 difference was found in the changes in peak passive moment between the three conditions (P
371 > 0.05). Significant correlations were observed (see Figure 4) between the changes in ROM
372 and changes in peak passive moment after CR ($r_s = 0.80$; $P < 0.01$), SS ($r_s = 0.82$; $P < 0.01$)
373 and Iso interventions ($r_s = 0.69$; $P < 0.01$) suggesting that changes in ROM were associated
374 with changes in the peak torque tolerated after each intervention.
375
376 MTC stiffness
377 Significant reductions were found in the slope of the passive joint moment curve after CR (21.0
378 ± 11.3%; $P < 0.01$), SS (10.1 ± 12.2%; $P < 0.01$) and Iso interventions (10.1 ± 11.8%; $P <$

379 *0.01*), indicating a significant reduction in MTC stiffness (see Figure 5). A significant
380 difference ($F = 4.9$; $P < 0.05$) was detected in the reductions in passive moment across the
381 three interventions. Similar to the ROM changes, post-hoc analysis revealed significantly
382 greater reductions in passive moment after CR compared with both SS and Iso ($P < 0.05$) but
383 no difference was found in the changes following static stretching and isometric contractions
384 ($P > 0.05$). As the mean changes in MTC stiffness after SS plus Iso were almost arithmetically
385 equal to changes following CR, we compared the changes in stiffness after CR with SS plus
386 Iso; where a significant correlation was detected ($r_s = 0.66$; $P < 0.01$). No significant
387 correlations were observed between reductions in MTC stiffness and increases in ROM after
388 CR, SS or Iso interventions ($P > 0.05$).
389
390 Achilles tendon stiffness and GM muscle stiffness
391 Significant reductions in tendon stiffness (see Figure 6. A) were found after CR (22.1 ± 24.1%;
392 $P < 0.01$) and Iso interventions (17.7 ± 20.8%; $P < 0.05$), but not after SS (1.7 ± 8.2%; $P >$
393 0.05). No difference in the reduction in tendon stiffness was found between CR and Iso
394 interventions ($P > 0.05$). Significant reductions in muscle stiffness (see Figure 6. B) were also
395 found after CR (20.5 ± 8.9%; $P < 0.01$) and SS (16.0 ± 12.3%; $P < 0.01$), but not after Iso
396 interventions (3.0 ± 7.0%; $P > 0.05$). No difference in the reduction in muscle stiffness was
397 found between CR and SS ($P > 0.05$).
398
399 Maximal isometric plantar flexor moment and EMG
400 No significant difference in maximal isometric plantar flexor moment or EMG activity (during
401 maximal contraction or passive rotation at full ROM) was found following any intervention (P
402 > 0.05), indicating that neuromuscular force generating capacity and reflexive muscle activity
403 was retained after all interventions.

DISCUSSION

Increases in ROM immediately following muscle stretching have been largely attributed to either increases in stretch tolerance (27, 38, 39) or changes in mechanical properties of the MTC (22, 24, 26, 33), although few studies have employed the requisite methodology to localize tissue-specific changes within the MTC. In the present study significantly greater increases in ROM and reductions in the passive moment measured at predetermined joint angles during a plantar flexor stretch were observed following acute CR stretching compared to static (passive) stretching or maximal isometric contractions, which is in agreement with our hypothesis. Additionally, moderate-to-strong correlations were observed between increases in ROM and increases in peak passive joint moment ($r_s = 0.80$: $P < 0.01$), which is considered an indication of greater 'stretch tolerance', after the CR stretching. Regarding mechanical changes, both muscle and tendon stiffness were reduced following CR stretching whereas static stretching influenced only muscle stiffness and isometric contractions influenced only tendon stiffness. In fact, the total decrease in MTC stiffness after CR stretching (~21%) was almost arithmetically equal to the changes in MTC stiffness after static stretching (~10%) plus the isometric contractions (~10%). The concomitant reductions in muscle and tendon stiffness after CR stretching are consistent with previous studies where reductions in muscle stiffness are reported after static stretching (17-19, 33) yet reductions in tendon stiffness are reported after maximal contractions (18-20), perhaps indicating that the separate effects of static stretching and isometric contractions were achieved by the singular imposition of CR stretching.

To determine whether the changes in MTC stiffness following CR could be explained by changes experienced following static stretching and isometric contractions, we compared the

changes in MTC stiffness after CR stretching with the summed changes in MTC stiffness following static stretching and isometric contractions, revealing a significant correlation (r_s = 0.66; $P < 0.01$). Despite the significant correlation, more than 50% of the changes in stiffness remain unexplained using this method, thus the separate loading strategies of static stretching and isometric contractions do not appear to fully explain the changes in stiffness following the CR stretching method included within the study. A more complex testing model, using a range of stretch and contraction intensities and durations to explore the relationship between the magnitude of separate and concurrent changes in muscle and tendon stiffness with changes in MTC stiffness, may provide a more comprehensive assessment of this relationship. The present methods used motion analysis in addition to ultrasonography to correct for possible ankle rotation during the ramped isometric contraction overestimating tendon length change measurements and compromising stiffness calculations. However, a possible limitation of this method is that linear 3D motion analysis model using 2 reflective markers was used to calculate Achilles tendon length. A curved path using multiple reflective markers may more accurately reflect Achilles tendon length (11), with substantial error introduced when measurements are taken over multiple, joint angles. However, error using the linear model is negligible in the anatomical position (0.8%; ref 11), thus any error in tendon length is likely minimal in the present study. Furthermore, acute changes in stiffness and test-retest reliability data were very high (ICC = 0.95) in the present study, thus we are confident that the present methods accurately captured changes in stiffness. These are the first data to confirm that CR stretching acutely influences both muscle and tendon stiffness, which is indicative of a broader adaptive response that offers a possible new mechanism for the reported superiority of CR stretching for acute ROM enhancement and reduction in resistance to stretch.

453 Historically, autogenic inhibition has been theorized as an important mechanism explaining the
454 superior effects of CR stretching for acute ROM enhancement (1, 36). Increased activity of
455 type Ib muscle afferents during the contraction phase was thought to hyperpolarize the dendritic
456 ends of spinal α-motoneurons of the stretched muscle, minimizing or removing the influence
457 of stretch-induced type Ia-mediated reflexive activity (29, 30). However, this mechanism is
458 unlikely in the present study as the low velocity of joint rotation during the stretching was
459 imposed in an attempt to minimize or remove Ia-mediated reflexive activity (30) and isolate
460 tissue mechanical responses as a possible underlying mechanism. The lack any substantial
461 EMG activity (< 5% MVC) at full ROM in both the pre- and post-intervention data, or any
462 significant pre-to-post change in EMG at full ROM, is indicative of minimal type Ia or Ib
463 reflexive involvement influencing maximal ROM or the post-stretch changes in ROM. These
464 data are similar to those reported in previous acute CR studies where EMG magnitude was
465 unchanged or even increased at full ROM (25, 32), thus autogenic inhibition is an unlikely
466 mechanism explaining either the increase in ROM following CR stretching or the significantly
467 greater increase in ROM compared with the other conditions. However, other neuromuscular
468 adaptations contributing to the gains in ROM cannot be discounted. In fact, a neurological
469 contribution is supported by the increase in peak passive moment (stretch tolerance) detected
470 after CR stretching (10.6%). However, increases in peak passive moment were also detected
471 after isometric contractions (6.8%) and static stretching (5.2%) and importantly, these increases
472 were not significantly different between conditions. Furthermore, moderate-to-strong
473 correlations (r_s = 0.69-0.82; $P < 0.01$) were observed between the changes in peak passive
474 moment and changes in ROM after each condition, indicative of altered type III or IV afferent
475 activity influencing pain perception and the magnitude of changes in ROM (27, 39). The
476 present changes in peak passive moment are strong evidence that stretch tolerance is an
477 important mechanism associated with acute increases in ROM regardless of the stretching

mode, however it is unlikely to explain CR stretching's efficacy to acutely increase ROM compared to other stretching modes as similar changes were observed between conditions.

Relatively low peak forces were applied to the MTC during the static stretching intervention (34.1 ± 4.2 Nm) compared to either the CR stretching (mean = 151.7 ± 13.2 Nm) or isometric contractions (123.3 ± 3.1 Nm). Despite this lower loading intensity, a significant increase in ROM and reduction in MTC stiffness was observed, although these changes were less than those elicited by CR stretching. Interestingly, a reduction in muscle stiffness of similar magnitude to that elicited by the CR protocol was observed, which is in accordance with the changes reported previously after static stretching (17-19, 33). While both the muscle and tendon deformed during the static stretch manoeuvre, the lower intensity of loading experienced during the static stretching was more likely to cause muscle rather than tendon stretch as the tendon is inherently stiffer than relaxed muscle tissue during plantar flexor stretches with the knee extended (6, 14, 33). The majority of studies have reported no change in tendon stiffness following plantar flexor static stretching (16, 33), with the few studies (9, 14, 22) reporting acute reductions in tendon stiffness using substantially longer stretch durations (5-20 min). Notwithstanding, no study using shorter (i.e. < 5 min), and potentially more practically/clinically relevant, durations of static stretch have reported a reduction in tendon stiffness. Thus, the duration of stretch may be a key determinant of the likelihood and location of stiffness change within the MTC. The intensity of stretching likely influences acute responses as greater changes in muscle stiffness are reported after constant torque versus constant angle stretching (12). However, our aim was to determine whether tendon loading (i.e. Iso and CR) contributed to the increase in ROM, thus identical stretching phases were performed across interventions (i.e. constant angle method). Continual ROM increases during the stretch phase (i.e. constant torque method) are difficult to control as some subjects may feel

503 unable to increase ROM further, introducing differing levels of strain between conditions and
504 compromising our ability to determine whether tendon loading influenced ROM. Furthermore,
505 constant torque stretches produce a more intense and sometimes painful stretch that may not
506 be suitable in sensitive populations (i.e. clinical or injured populations). However, to ensure
507 substantial stress was achieved during each stretch and to more closely reflect current practice
508 the subjects were encouraged to push each successive stretch to a greater joint angle as their
509 stretch tolerance increased. Collectively, the findings of the present and other studies point to
510 an acute muscle-based adaptive response following moderate-duration static stretching as an
511 important mechanism, either directly (by reduced muscle stiffness) or indirectly (by altered
512 afferent activity), underpinning the increases in ROM.
513
514 As the duration of stretch or tissue loading may influence the likelihood and location of changes
515 in tissue stiffness, an isometric contraction intervention was used to specifically impose an
516 isolated high-intensity loading to the tendon while reducing strain in the muscle. Substantially
517 higher forces are transmitted through the tendon during isometric contractions as compared to
518 static stretching (18), resulting in greater tendon deformation (6). We hypothesized that this
519 would increase the likelihood of changes in tendon stiffness. The isometric contraction
520 protocol was chosen in order to provide a similar level of tendinous tissue loading as the CR
521 intervention to determine the impact of tendinous stretch alone on the changes in ROM.
522 Significantly greater loading was observed during both isometric contractions and CR
523 stretching protocols compared with static stretching, however loading during CR was also
524 significantly greater than in the isometric protocol. This is likely a consequence of
525 gastrocnemii and soleus force-length properties, which would have operated on the ascending
526 limb of their force-length curve in the present experiments (23, 24). Despite the difference in
527 loading magnitude, a substantial reduction in tendon stiffness (~18%) was found after the

528 isometric contractions that was similar to the change found after CR stretching (~22%).
529 However, the reduction in tendon stiffness after the isometric contractions occurred without a
530 change in muscle stiffness and was associated with a similar and significant increase in ROM
531 (~3°) as the static stretch intervention. The increase in ROM achieved with a concomitant
532 reduction in tendon stiffness following the isometric contraction intervention is a novel finding
533 and provides further support for the concept that acute mechanical changes (from muscle
534 stretching or muscular contractions) influencing the increases in ROM. Despite similar
535 increases in ROM being observed following both static stretching and isometric contractions
536 the location of changes in tissue stiffness were clearly distinct, with increases in ROM post-
537 stretch being attributable to reductions in muscle stiffness but increases in ROM following the
538 isometric contractions being attributable to reductions in tendon stiffness. However, whether
539 these muscle- or tendon-based mechanical changes directly (reduced stiffness) or indirectly
540 (altered afferent feedback) influenced ROM remains to be established. Regardless, the present
541 findings have clear methodological implications as performing contractions in the anatomical
542 position results in similar mechanical changes in tendon stiffness as CR stretching. Thus,
543 modifying CR stretching to perform the contraction phase in the anatomical, rather than highly
544 stretched, position removes the need for partner assistance, decreases the likelihood of tissue
545 damage and muscle strain injury (5), and results in a simpler technique that may be more widely
546 used in clinical and athletic populations.
547
548 In summary, the present study is the first to examine the acute effects of muscle-dominant
549 versus tendon-dominant tissue loading using CR stretching, static (passive) stretching and
550 maximal isometric contractions on joint ROM, MTC stiffness, maximal passive joint moment
551 (stretch tolerance), muscle and tendon stiffness, and EMG activity. Although significant
552 increases in ankle joint ROM and reductions in MTC stiffness were evident after all

553 interventions, the increase in ROM after CR stretching was significantly greater. Furthermore,
554 reductions in stiffness were tissue specific and distinct between interventions, with static
555 stretching acutely reducing muscle stiffness, isometric contractions reducing tendon stiffness,
556 and CR stretching reducing both muscle and tendon stiffness. Clearly, the mode of tissue
557 loading is an important determinant of changes in tissue stiffness, with static stretching
558 sufficient to reduce muscle stiffness but isometric contractions and CR stretching influencing
559 the tendon. The present data provide clear evidence that tissue-specific imaging is essential to
560 determine the influence of such interventions on tissue-specific MTC properties and enable
561 possible underlying mechanisms associated with changes in ROM to be identified. The present
562 data provide novel and compelling evidence for a mechanical mechanism underpinning acute
563 changes in ROM after muscle stretching, with the greater efficacy of CR stretching to acutely
564 increase ROM likely attributable to concomitant reductions in muscle and tendon stiffness.
565 Notwithstanding the clear mechanical differences between conditions, significant correlations
566 between the change in peak passive moment and the change in ROM were also observed in all
567 conditions. This result is considered good evidence for a neurological adaptation (i.e. increased
568 stretch tolerance) also being important for the acute increases in ROM.
569

570 **ACKNOWLEDGMENTS**

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572

573 **CONFLICT OF INTEREST**

574 No conflicts of interest exist. The results of the present study do not constitute endorsement
575 by the American College of Sports Medicine.
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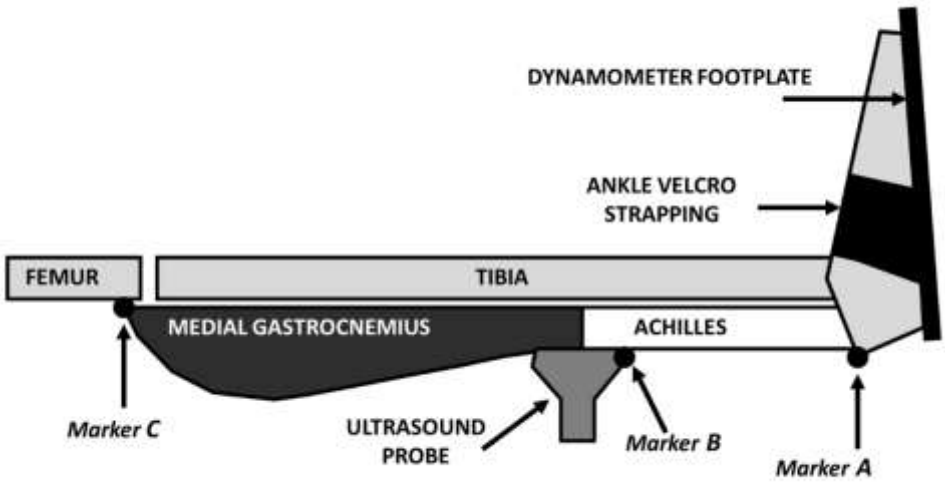
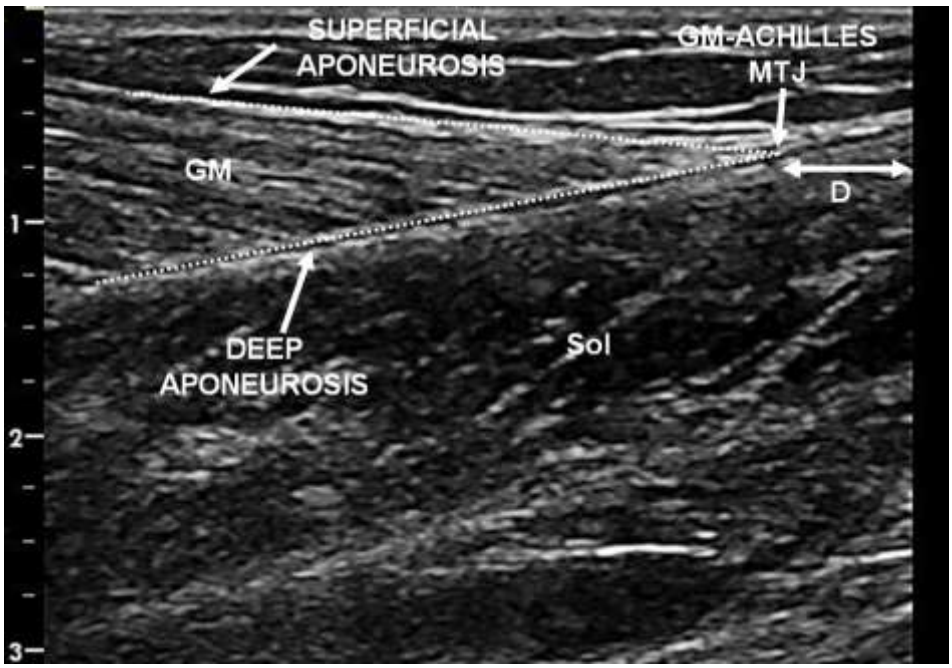
689

690 **FIGURE CAPTIONS**

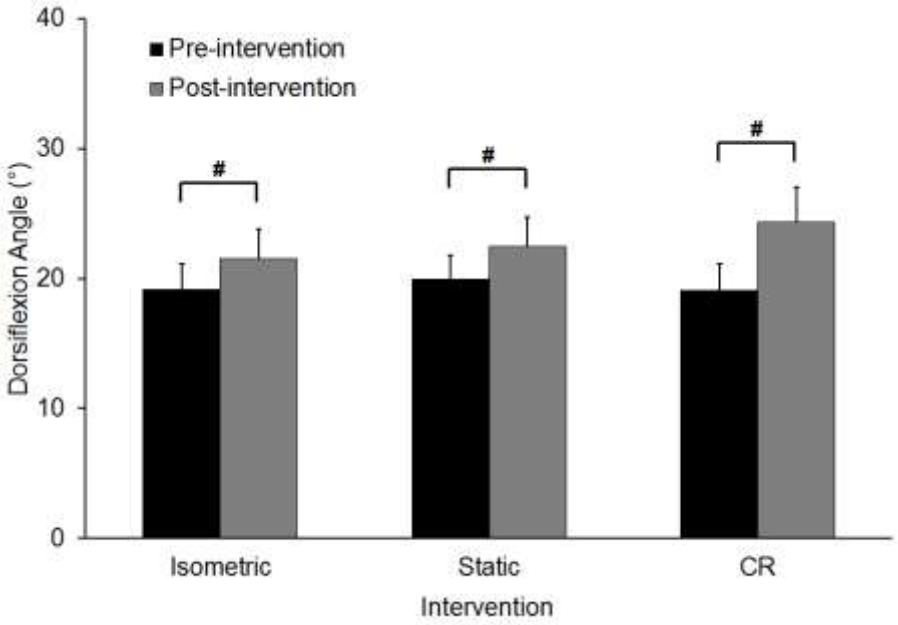


691

692 **Figure 1.** Schematic showing motion analysis reflective markers and ultrasound probe
693 positioning. Achilles tendon length was estimated from the distance between reflective
694 markers placed over the insertion of the Achilles on the calcaneus (*marker A*) and the distal
695 edge of the ultrasound probe (*marker B*) placed over the gastrocnemius medialis (GM)-Achilles
696 muscle-tendon junction (MTJ). GM muscle length was estimated from the distance between
697 the reflective markers placed over the origin of the GM muscle on the medial femoral
698 epicondyle (*marker C*) and the distal edge of the ultrasound probe (*marker B*) placed over the
699 GM-Achilles MTJ.
700



701
702 **Figure 2.** Ultrasound image of the GM-Achilles MTJ. The position and displacement of the
703 gastrocnemius medialis (GM)-Achilles muscle-tendon junction (MTJ) was recorded using real-
704 time ultrasound imaging. The MTJ was identified as the point where the deep GM and
705 superficial soleus (Sol) aponeuroses and superficial GM aponeurosis merged with the Achilles
706 tendon. Displacement of the MTJ from the distal edge of the image (D) was synchronized with
707 motion analysis data to calculate GM muscle and Achilles tendon lengths.
708



709

710 **Figure 3.** Mean dorsiflexion ROM pre- and post-intervention. Significant increases in
711 dorsiflexion range of motion (ROM) were found for contract-relax (CR) stretching (5.3°), static
712 stretching (2.6°) and isometric contractions (2.5°). Significantly greater increases in ROM
713 were found after CR stretching compared with both static stretching and isometric contractions
714 but no difference was found between the increases in ROM following static stretching and
715 isometric contractions. #Significant to $P < 0.01$.

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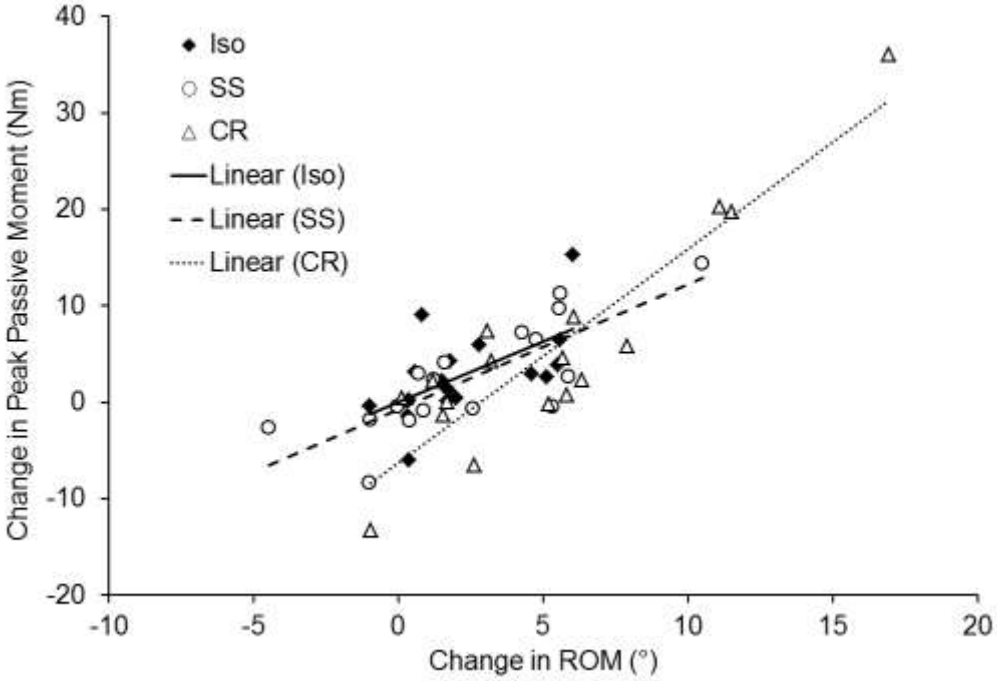
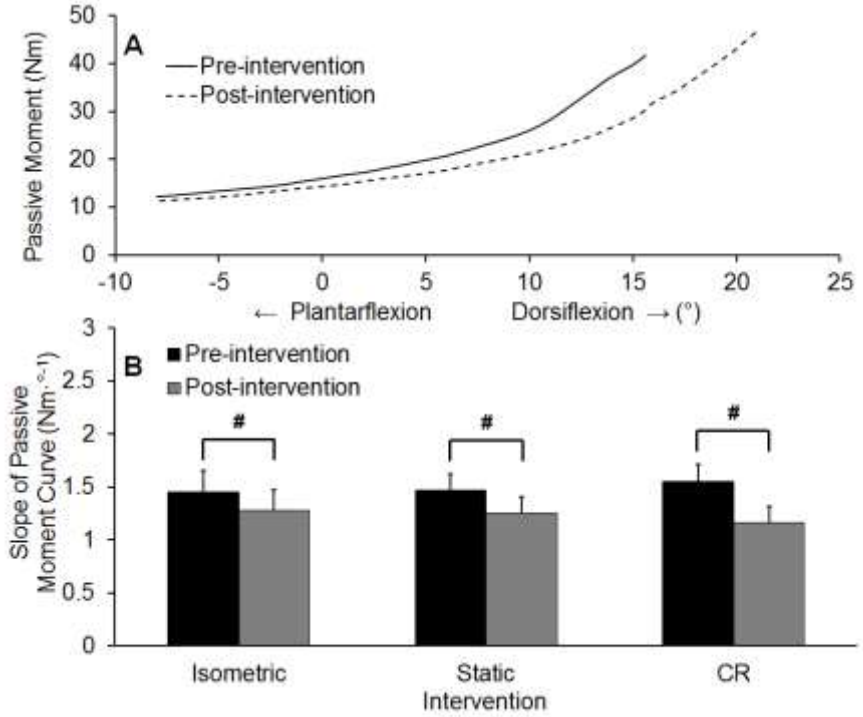


718

719 **Figure 4.** Correlation between changes in ROM (pre-to-post intervention) and peak passive
720 joint moment. Significant correlations were found between the changes in range of motion
721 (ROM) and peak passive moment (i.e. stretch tolerance) after contract-relax (CR) stretching (r_s
722 = 0.80; $P < 0.01$), static stretching (r_s = 0.82; $P < 0.01$) and isometric contractions (r_s = 0.69;
723 $P < 0.01$).

724



725

726 **Figure 5.** Passive plantar flexor moment (MTC stiffness) pre- and post-intervention. Passive
727 moment (A) was reduced post-intervention at all dorsiflexion angles along the joint moment-
728 angle curve (one subject's data depicted during a contract-relax [CR] trial). Significant
729 reductions (B) in the slope of the passive moment curve (indicative of muscle-tendon complex
730 [MTC] stiffness) were found after CR stretching (21.0%), static stretching (10.1%) and
731 isometric contractions (10.1%). Significantly greater reductions in passive moment were found
732 after CR stretching compared with both static stretching and isometric contractions, but no
733 difference was found between the changes in passive moment following static stretching or
734 isometric contractions. #Significant to $P < 0.01$.

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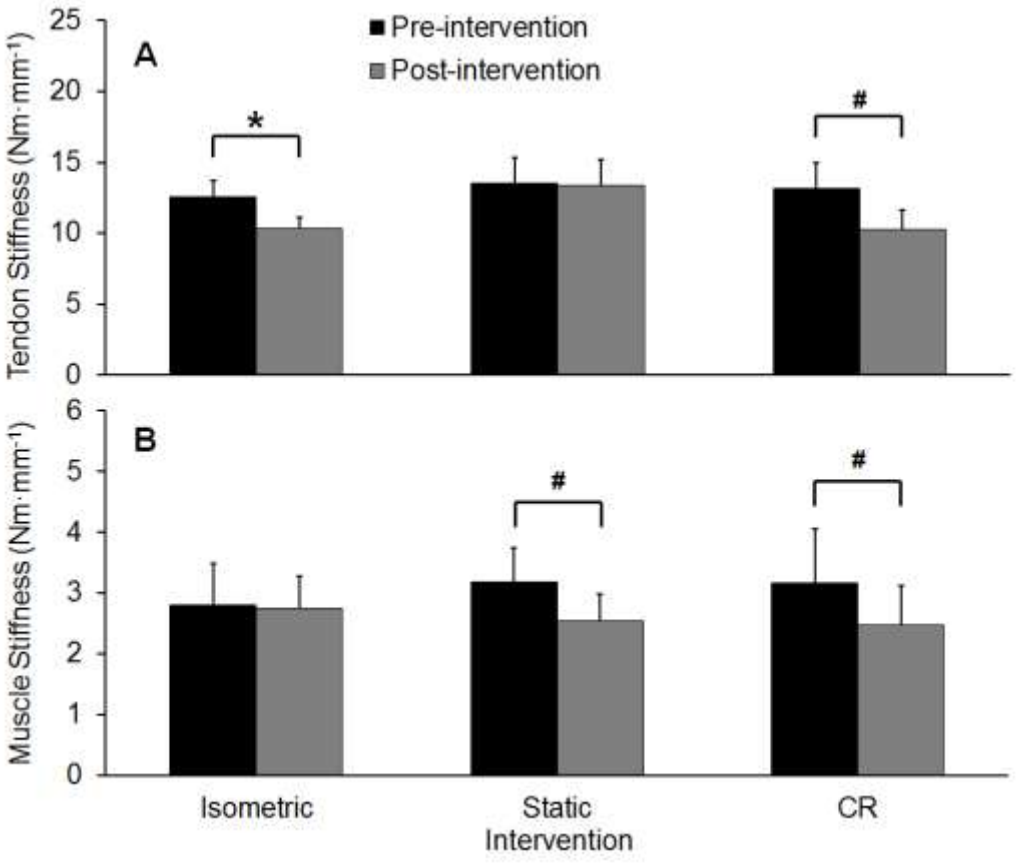

736

737 **Figure 6.** Achilles tendon stiffness and gastrocnemius medialis (GM) muscle stiffness pre-
738 and post-intervention. Significant reductions in tendon stiffness (A) were found after contract-
739 relax (CR) stretching (22.1%) and isometric contractions (17.7%), but not after static stretching
740 (1.7%). No difference in the reductions in tendon stiffness was found between CR stretching
741 and isometric contractions. Significant reductions in muscle stiffness (B) were found after CR
742 stretching (20.5%) and static stretching (16.0%), but not after isometric contractions (3.0%).
743 No difference in the reductions in muscle stiffness was found between CR stretching and static
744 stretching. *Significant to $P < 0.05$, #Significant to $P < 0.01$.

745