## ORIGINAL ARTICLE



# Acute effects of contract-relax (CR) stretch versus a modified CR technique

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#### **Abstract**

Purpose Contract–relax (CR) stretching increases range of motion (ROM) substantively, however its use in athletic environments is limited as the contractions performed in a highly stretched position require partner assistance, are often painful, and may induce muscle damage. Therefore, the acute effects of performing the contractions 'off stretch' in the anatomical position [stretch–return–contract (SRC)] were compared with traditional CR stretching in 14 healthy human volunteers.

Methods Passive ankle joint moment and dorsiflexion ROM were recorded on an isokinetic dynamometer with electromyographic monitoring of the triceps surae, whilst simultaneous real-time motion analysis and ultrasound imaging recorded gastrocnemius medialis muscle and Achilles tendon elongation. The subjects then performed CR or SRC stretches ( $4 \times 10$ -s stretches and 5-s contractions) randomly on separate days before reassessment.

Results Significant increases in dorsiflexion ROM  $(4.1^{\circ}-4.0^{\circ}; P < 0.01)$  and peak passive moment (10.9-15.1 %; P < 0.05) and decreases in the slope of the passive moment curve (19.1-13.3 %; P < 0.05), muscle stiffness

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(21.7–21.3 %; P < 0.01) and tendon stiffness (20.4–15.7 %; P < 0.01) were observed in CR and SRC, respectively. No between-condition differences were found in any measure (P > 0.05).

Conclusions Similar mechanical and neurological changes were observed between conditions, indicating that identical mechanisms underpin the ROM improvements. These data have important practical implications for the use of this stretching mode in athletic environments as performing the contractions 'off stretch' eliminates the pain response, reduces the risk of inducing muscle damage, and removes the need for partner assistance. Thus, it represents an equally effective, simpler, and yet potentially safer, stretching paradigm.

 $\begin{tabular}{ll} \textbf{Keywords} & Proprioceptive neuromuscular facilitation} \cdot \\ Range of motion \cdot Tendon stiffness \cdot Ultrasound \\ \end{tabular}$ 

## **Abbreviations**

CI Confidence intervals
CR Contract—relax
EMG Electromyography
GL Gastrocnemius lateralis
GM Gastrocnemius medialis

ICC Intraclass correlation coefficient

MTC Muscle-tendon complex MTJ Muscle-tendon junction

MVC Maximal voluntary contraction

PNF Proprioceptive neuromuscular facilitation

ROM Range of motion

Sol Soleus

SE Standard error

SRC Stretch-return-contract

TA Tibialis anterior

TTL Transistor-transistor logic



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#### Introduction

Both the maximal joint range of motion (ROM) and resistance to stretch during rotation (indicative of tissue stiffness) are important functional parameters that may affect muscle strain injury risk (Witvrouw et al. 2003), influence the capacity to perform activities of daily living (Mulholland and Wyss 2001), and are compromised with aging (Bassey et al. 1989) and disease (Duffin et al. 1999). Static muscle stretching is a commonly used technique to acutely improve ROM with these improvements thought to be attributable to several mechanisms, including reductions in tissue stiffness (Kay et al. 2015; Morse et al. 2008), altered peripheral (afferent) output (Avela et al. 1999, 2004), and dampened pain, pressure or stretch perception increasing stretch tolerance (i.e., the capacity to tolerate increased loading prior to terminating the stretch; Magnusson et al. 1996; Mitchell et al. 2007; Weppler and Magnusson 2010). Despite the popularity of static stretching, proprioceptive neuromuscular facilitation stretching (PNF) is regularly reported as being the most effective stretching technique for acute and chronic improvements in ROM (Funk et al. 2003; Hindle et al. 2012). A common method of PNF stretching is the contract-relax (CR) technique (Sharman et al. 2006), which includes a static stretching phase for a prescribed duration, followed immediately by an intense, often maximal, isometric contraction performed in a fully stretched position. Upon completion of the contraction the joint is rotated further to again stretch the target muscle, with stretch intensity normally to the point of discomfort. While CR stretching is highly effective and often used in clinical environments to achieve rapid increases in ROM, it is not commonly used in athletic warm-up routines possibly because it normally requires an assisting partner, may be painful, and is thought to pose a greater muscle strain injury risk compared with static stretching (Beaulieu 1981).

Few studies have examined the underlying mechanisms associated with increases in ROM following CR stretching (Hindle et al. 2012; Kay et al. 2015), consequently these mechanisms remain essentially theoretical and poorly understood. Two neuromuscular mechanisms (autogenic inhibition, gate control theory) have been theorized (for review see Hindle et al. 2012). Regarding autogenic inhibition, a neuromuscular inhibition was thought to occur as the loading of the tendon during the contraction phase of CR activated/stimulated type Ib muscle afferent output from the golgi tendon organs, stimulating inhibitory spinal synapses and hyperpolarizing the dendritic ends of spinal α-motoneurons of the stretched muscle. The Ib activity would likely diminish the influence of homonymous Ia muscle afferents on the  $\alpha$ -motoneuron pool of the stretched muscle, with the diminished reflex activity thought to allow

further increases in ROM (Prentice 1983). However, several original studies have previously reported no change in electromyographic (EMG) magnitude at full ROM (Kay et al. 2015; Mitchell et al. 2009; Osternig et al. 1990), with reviews concluding autogenic inhibition was unlikely to be an important mechanism underpinning the increase in ROM following CR stretching (Hindle et al. 2012; Sharman et al. 2006). Gate control theory posits that pressure receptors (type III afferents) activated during the contraction phase could inhibit pain perception (Mazzullo 1978), as pressure receptors are associated with larger myelinated neurons that connect to the same spinal interneurons as unmyelinated nociceptive fibers (type IV afferents) within the spinal horn (Melzack 1993). The increased activity of pressure receptors would theoretically diminish the influence of homonymous IV afferent output and pain perception, thus enabling further increases in ROM. While these neuromuscular pathways are theoretical, increased stretch tolerance (dampened pain perception) is commonly reported following CR stretching (Kay et al. 2015; Mitchell et al. 2009). Thus although autogenic inhibition has largely been discounted, a neurological contribution to the increased ROM following CR stretching is at least partly supported.

The distinct muscle-tendon (and joint) loading characteristics of various stretching methods likely result in different mechanical responses, with a key distinction between CR and other stretching techniques being the inclusion of an intense, often maximal, isometric contraction performed following the stretching phase and performed with the muscle remaining in a highly stretched position. During passive ankle dorsiflexion stretches, more flexible subjects demonstrate greater tendon elongation with no detectable differences in the onset or magnitude of muscle activity toward the end of rotation or near full ROM (Blazevich et al. 2014), therefore tendon properties may, at least partly, influence maximum ROM. While muscular and tendinous tissues experience deformation during stretching (Blazevich et al. 2014; Morse et al. 2008), studies employing ultrasonography techniques have found muscle stiffness to be reduced after an acute bout of static stretching, whereas tendon stiffness remained unaltered (Kay and Blazevich 2009a; Morse et al. 2008). However, a recent study revealed that CR stretching acutely reduced both muscle and tendon stiffness and elicited significantly greater increases in ROM compared with a similar volume of static stretching after which only a reduction in muscle stiffness was induced (Kay et al. 2015). This broader acute adaptive response, where both muscle and tendon stiffness are influenced concurrently, offers a potentially important mechanism underpinning the superior efficacy of CR stretching for acutely increasing ROM when compared to other stretching techniques.



CR stretching is implemented to the aim of increasing ROM, often in an attempt to reduce muscle strain injury risk. However, paradoxically, performing intense muscular contractions in a highly stretched position, where the muscle is vulnerable to injury, increases the risk of inducing tissue damage (Beaulieu 1981; Butterfield and Herzog 2006; Whitehead et al. 2003). Thus, the question should be asked whether the performance of isometric contractions in a non-stretched position between each passive static stretching cycle is as effective as performing the contractions during each passive static stretching cycle (i.e., contractions performed with the muscle in a highly stretched position). Interestingly, several studies have reported acute reductions in tendon stiffness following maximal isometric contractions performed in the anatomical position (i.e., with the muscle off stretch; Kay and Blazevich 2009b; Kay et al. 2015; Kubo et al. 2002). Furthermore, a recent study reported concomitant increases in ROM and reductions in tendon stiffness following isometric contractions performed in the anatomical position (Kay et al. 2015), with the acute increase in ROM being similar to that observed following static stretching. Collectively, these findings suggest that substantial tendon loading, regardless of muscle length, should influence tendon stiffness and ROM. Consequently, modification of the CR stretching technique to perform the muscle contraction phase with the muscle 'off stretch' may provide a similar stimulus whilst reducing injury risk. Therefore, the aims of the present study were to examine the influence of an acute bout of CR stretching versus a modified CR technique [stretch-return-contract (SRC); where the contractions are performed 'off stretch'] on dorsiflexion ROM, maximal passive joint moment at full volitional ROM (stretch tolerance), the slope of the passive moment curve [indicative of whole muscle-tendon complex (MTC) stiffness], gastrocnemius medialis (GM) muscle stiffness and triceps surae EMG activity (measured during a passive joint stretch). The acute effects of these interventions on Achilles tendon stiffness, maximal isometric plantar flexor joint moment and peak triceps surae EMG activity during a maximal isometric contraction were then measured. We tested the hypothesis that CR and SRC stretching techniques would produce similar increases in ROM and stretch tolerance whilst reducing muscle and tendon stiffness.

#### **Methods**

## **Subjects**

Fourteen recreationally active participants (8 women, 6 men; age =  $26.1 \pm (SD)$  9.6 years, height =  $1.7 \pm 0.1$  m, and mass =  $75.6 \pm 13.3$  kg) with no recent history of lower

limb musculoskeletal injury or neurological deficit volunteered for the study after completing a pre-test medical questionnaire and giving written and informed consent. The subjects were asked to avoid any flexibility training, intense exercise and stimulant use for 48 h prior to testing. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee, and the study was completed in accordance with the Declaration of Helsinki.

#### **Protocol**

Overview

The subjects were familiarized with the testing protocol 1 week prior to data collection and then visited the laboratory on two further occasions under experimental conditions, with trials counterbalanced and separated by 1 week. During the experimental trials, the subjects performed a 5-min warm-up on a Monark cycle at 60 rpm with a 1-kg resistance load. The subjects were then seated in the chair of an isokinetic dynamometer (Biodex System 3 Pro, IPRS, Suffolk, UK) with the hip flexed to 55° and knee fully extended (0°) to ensure all plantar flexor muscles could be strongly activated and were at an appropriate length to contribute strongly to the total passive and active joint moments (Cresswell et al. 1995; Kawakami et al. 1998). The ankle was then placed in the dynamometer footplate in the anatomical position (0°) with the lateral malleolus aligned to the centre of rotation of the dynamometer. Nonelastic Velcro strapping was used to minimize heel displacement from the dynamometer footplate to provide reliable and valid ROM and passive moment data during the passive trials (Morse et al. 2008). To ensure that the degree of ankle fixation did not substantially influence the passive moment or ROM data during the pre- and post-intervention measurements, one highly experienced analyst conducted all trials to remove inter-tester variability (Fig. 1).

Skin-mounted bipolar double differential active electrodes (model MP-2A, Linton, Norfolk, UK) were placed over the soleus (Sol), gastrocnemius medialis (GM), gastrocnemius lateralis (GL) and tibialis anterior (TA) muscles. Site preparation, electrode placement, EMG sampling, processing and normalization methods were completed as previously described (Kay and Blazevich 2009a). EMG amplitude was constantly monitored during the passive and active trials to quantify muscle activity (described later). As ankle rotation can occur during 'isometric' plantar flexion contractions performed in a dynamometer (Karamanidis et al. 2005), measurement of calcaneal movement (i.e., distal aspect of the Achilles tendon) using motion analysis was used to correct for any error induced by this joint rotation (Manal et al. 2013), which would otherwise have resulted



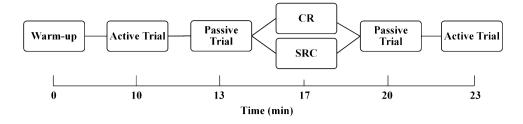
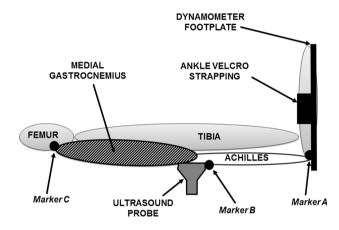


Fig. 1 Timeline of the contract-relax (CR) and stretch-return-contract (SRC) stretching protocols. At 5 min after completion of the warm-up, active and passive trials were conducted, and after 2 min

further rest either the CR or SRC stretching intervention was carried out. Two minutes later, passive and active trials were repeated to determine the effects of each intervention



**Fig. 2** Infrared reflective motion analysis marker and ultrasound probe positioning. Achilles tendon length was estimated as the distance between the reflective markers placed on the distal edge of the ultrasound probe (*marker B*) located over the gastrocnemius medialis (GM)-Achilles muscle–tendon junction (MTJ) and the insertion of the Achilles on the calcaneus (*marker A*). GM muscle length was estimated from the distance between the reflective markers placed on the distal edge of the ultrasound probe (*marker B*) and the origin of the GM muscle on the medial femoral epicondyle (*marker C*)

in an overestimation of tendon length change. Four infrared digital cameras (ProReflex, Qualisys, Gothenburg, Sweden) enabled real-time motion analysis to record the movement of infrared reflective markers placed over the insertion of the Achilles at the calcaneus (see Fig. 2; 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). Real-time ultrasound imaging (LOGIQ Book XP, General Electric, Bedford, UK) was performed with a sampling frequency 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) to image the position (and excursion) of the GM-Achilles MTJ (see Fig. 3). The probe was positioned with the proximal end towards the origin of the

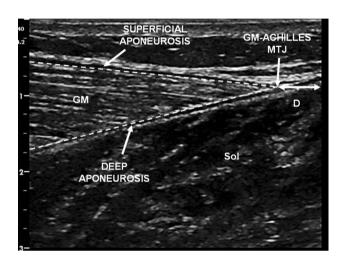


Fig. 3 Ultrasound image of the gastrocnemius medialis (GM)-Achilles muscle-tendon junction (MTJ). Real-time ultrasound imaging was used to record the position (and displacement) of the GM-Achilles MTJ. The MTJ was identified as the point where the deep GM and superficial soleus (*Sol*) aponeuroses and superficial GM aponeurosis merged with the Achilles tendon. Displacement of the MTJ from the distal edge of the image (*D*) was synchronized with motion analysis data to calculate GM muscle and Achilles tendon lengths

medial head and the distal end towards the insertion of the Achilles tendon. The probe was then manipulated until the superficial and deep GM aponeuroses could be visualized to enable triangulation of the GM-Achilles tendon MTJ. The probe was then fixed to the skin with zinc-oxide adhesive tape to ensure consistent and accurate imaging of the MTJ during the experimental trials.

#### Active and passive trials

During the active trial the subjects were instructed to perform a 5-s ramped maximal isometric plantar flexor contraction to determine maximal isometric strength, EMG activity, and tendon stiffness (described later). To confirm that loading rate did not influence tendon stiffness, during the familiarization session, visual feedback of the plantar



flexor joint moment was provided during the ramped contractions until the subjects reliably achieved a linear increase in moment reaching maximal voluntary contraction (MVC) after ~3 s. Furthermore, during the experimental trials the time taken by the subjects to increase plantar flexor moment from 50 to 90 % MVC (the range that tendon stiffness was calculated) was recorded in the pre- and post-intervention sessions; no significant difference (pre =  $2.1 \pm 0.2$  s, post =  $2.0 \pm 0.2$  s; P > 0.05) was found. Two minutes later the subjects performed three passive dorsiflexion rotations initiated from 20° plantar flexion through to full dorsiflexion at 0.087 rad s<sup>-1</sup> (5° s<sup>-1</sup>) until the subject volitionally terminated the rotation by pressing a hand held release button at the point of discomfort.

#### Stretching interventions

Two minutes after completing the passive ROM trials the subjects performed either the CR or SRC stretching intervention. During the CR condition the ankle was passively rotated at 0.087 rad s<sup>-1</sup> until reaching the point of discomfort, a position regularly used in stretch studies (Blazevich et al. 2012; Kay and Blazevich 2008, 2009a). The movement velocity was too slow to elicit a significant myotatic stretch reflex response, which ensured that full ROM was achieved and substantial stress was applied to the MTC (McNair et al. 2001). Furthermore, this ensured that moment data were reflective of the passive properties of the MTC. The subjects' ankles were held in the stretched position for 10 s and followed immediately with a 5-s ramped maximal isometric contraction (peak joint moment was obtained after ~3 s from contraction initiation and held for ~2 s) performed with the muscle at full stretch (i.e., point of discomfort). Upon contraction cessation, the ankle was then immediately rotated again at 0.087 rad s<sup>-1</sup> until reaching the point of discomfort with the protocol repeated three further times giving a total duration of 60 s (i.e.,  $4 \times$ 10-s stretches and  $4 \times 5$ -s contractions). The constant angle stretching method was chosen during the static stretching phase of the CR stretching technique as increasing ROM during the stretches (constant torque) may introduce differing levels of strain between conditions (Herda et al. 2014), which would have compromised our ability to determine whether muscle length during the contraction phase influenced ROM. Furthermore, the static stretch phase duration (10 s) was considered too short to enable further meaningful or reliable increases in ROM prior to the contraction phase. During the SRC protocol the static stretch phase was performed identically, however after the 10 s of stretching the ankle was returned to the anatomical position where the 5-s ramped maximal isometric contraction was performed. The ankle was rotated again 0.087 rad s<sup>-1</sup> until reaching the point of discomfort with the protocol repeated three times giving a total duration of 60 s. Two minutes later the subjects repeated the passive and active trials (see Fig. 1).

#### Measures

Plantar flexor moment and ROM

Maximal isometric plantar flexor moment was recorded pre- and post-intervention during the active trial to determine the influence of CR and SRC stretching on isometric strength. Peak isometric plantar flexor moment was also recorded during the four contractions performed during the CR and SRC interventions to determine the average peak loading during each intervention. Passive moment data were recorded from the third passive ROM trial to ensure thixotropic properties of the skeletal muscles did not influence the joint moment data (Proske and Morgan 1999). The passive rotation enabled ROM, peak passive moment (stretch tolerance), and the slope of the passive moment curve (indicative of MTC stiffness) to be recorded. The slope of the passive moment curve represents joint stiffness (i.e., all joint structures contribute to moment), however at the ankle the triceps surae contribute >70 % to the total ankle joint moment (Murray et al. 1976), thus the slope may be considered primarily indicative of MTC stiffness. Peak passive moment was measured within a 250-ms epoch at full volitional ROM, with the slope of the passive moment curve 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; identical joint angles were used in the post-stretching trial. Joint moment and dorsiflexion angle data were directed from the dynamometer to a high level transducer (model HLT100C, Biopac, Goleta, CA) before analogueto-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.

## Electromyographic (EMG) activity

Raw EMG signals from the Sol, GM, GL (i.e., the triceps surae muscle group) and TA (antagonistic muscle) were amplified (gain = 300, input impedance = 10 G $\Omega$ , common mode rejection ratio  $\geq$ 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) and stored on a personal computer running AcqKnowledge software (v4.1, Biopac). EMG signals collected during maximal volitional contractions as well as during muscle stretches were then processed using a 20- to 500-Hz



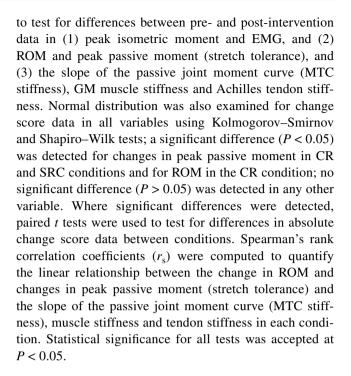
band-pass filter and converted to root-mean-squared EMG with a moving symmetrical 250-ms averaging window. Additionally, a 10-ms averaging window was used on EMG data collected during muscle stretches in order to check for reflexive short-burst EMG activity that may have resulted from velocity-dependent stretch receptors (i.e., type Ia). The EMG data were then normalized as a percentage of the peak amplitude recorded during the first MVC in the preintervention active trial. The normalized EMG amplitude (%MVC) was used as a measure of neuromuscular activity quantified within a 250-ms epoch at peak joint moment during the active trials and at full volitional ROM during the passive trials.

## Muscle and tendon length and stiffness

Ultrasound, motion analysis and dynamometry data were electronically synchronized using a 5-V ascending transistor-transistor logic (TTL) pulse. The TTL pulse simultaneously placed a marker on the AcqKnowledge (v4.1, Biopac) software while ending the capture of motion analysis and ultrasound data. Motion analysis data were then directed to and stored on a personal computer operating Track Manager 3D software (v.2.0, Qualisys). 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. Tendon length was calculated as the distance between reflective markers A and B (using motion analysis), plus the distance from the actual MTJ position to the distal edge of the image (using ultrasound) in a method identical to that previously reported (Kay et al. 2015). Tendon stiffness was calculated as the change in plantar flexor moment from 50 to 90 %MVC divided by the change in tendon length (Nm mm<sup>-1</sup>). Muscle length was calculated as the distance between reflective markers B and C (using motion analysis), minus the distance from actual MTJ position to the distal border of the image. Muscle stiffness was calculated as the change in plantar flexor moment through 10° of dorsiflexion (in the linear portion of the passive moment curve) divided by the change in muscle length (Nm mm<sup>-1</sup>).

#### Statistical analysis

All data were analyzed using SPSS statistical software (v.20; LEAD Technologies Inc., USA) and are reported as means and 95 % confidence intervals (CI). Normal distribution was assessed for pre- and post-group data 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. The effects of time were examined using separate multiple analyses of variance (MANOVA) with repeated measures



#### Reliability

Test–retest reliability was determined for peak isometric moment, peak passive moment, ROM, the slope of the passive moment curve (MTC stiffness) and muscle and tendon stiffness in the pre-test data in both conditions. No significant difference was detected between mean values (P > 0.05) for any measure; ICC's were 0.84, 0.93, 0.96, 0.97, 0.99, and 0.78. Coefficients of variation and SE (expressed as a percentage of the mean) were 13.4 % (SE = 3.6 %), 14.5 % (SE = 3.9 %), 5.1 % (SE = 1.4 %), 14.6 % (SE = 3.9 %), 11.1 % (SE = 3.0 %), and 11.6 % (SE = 3.1 %), 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 (Kay and Blazevich 2009b; Kay et al. 2015; Kubo et al. 2002; Magnusson et al. 1996). To ensure an adequate sample size was recruited for the study, power analyses were conducted using the following parameters (power = 0.80, alpha = 0.05, effect size = 1.0, attrition = 20 %). The analysis revealed that the initial sample size required to reach statistical power was 14, thus 18 subjects were recruited to account for possible attrition or data loss. Two subjects withdrew from the study with non-related injuries and two failed to complete both interventions; statistical analyses were conducted on data sets for 14 subjects who completed the testing.



#### Results

## Range of motion and stretch tolerance

A significant increase in dorsiflexion ROM (see Fig. 4) was found after CR [4.1° (CI = 2.6, 5.6); P < 0.01] and SRC [ $4.0^{\circ}$  (CI = 2.0, 6.0); P < 0.01] stretching conditions. No difference in the increase in ROM was found between conditions (P > 0.05) indicating that both techniques were equally effective at increasing dorsiflexion ROM. When peak passive moment (stretch tolerance) was examined at full ROM pre- and post-intervention, a significant increase was found after CR [10.9 % (CI = 4.4, 17.4); P < 0.05] and SRC [15.1 % (CI = 1.3, 28.9); P < 0.05] stretching; no difference in the increase in stretch tolerance was found between conditions (P > 0.05). Significant correlations were observed between the changes in ROM and peak passive moment (stretch tolerance) in CR ( $r_s = 0.63$ ; P < 0.05) and SRC conditions ( $r_s = 0.71$ ; P < 0.05) indicating that changes in ROM were associated with changes in stretch tolerance after both interventions.

## MTC, muscle and tendon stiffness

When the slope of the passive moment curve (indicative of MTC stiffness) was examined pre- and post-intervention, significant reductions (see Fig. 5b) were found after both CR [19.1 % (CI = 9.7, 28.5); P < 0.05] and SRC [13.3 % (CI = 3.8, 22.8); P < 0.05] stretching. No difference in the reduction in MTC stiffness was found between conditions (P > 0.05), indicating a similar response after each condition. To determine the influence on specific tissues, changes in muscle and tendon stiffness were also estimated separately. Significant reductions were found in tendon stiffness (see Fig. 6a) after CR [20.4 % (CI = 15.2, 25.6); P < 0.01] and SRC [15.7 % (CI = 9.6, 21.8); P < 0.01] stretching. Significant reductions were also found in GM muscle stiffness (see Fig. 6b) after CR [21.7 % (CI = 17.4, 26.0); P < 0.01] and SRC [21.3 % (CI = 10.6, 32.0); P < 0.05] stretching. No differences (P > 0.05) in the reduction in muscle or tendon stiffness were found between conditions, indicating a similar adaptive mechanical response. No significant correlations (P > 0.05) were found between the changes in ROM and changes in MTC stiffness, muscle stiffness or tendon stiffness in CR and SRC conditions, respectively.

## Isometric plantar flexor moment and EMG

No significant difference was found in maximal isometric plantar flexor moment [-0.8% (CI = -9.9, 8.3), P > 0.05; 1.7% (CI = -6.6, 10.1), P > 0.05] or triceps surae EMG activity (average of Sol, GM and GL activity reported)

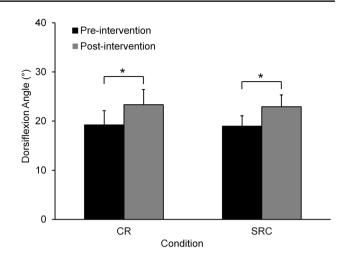


Fig. 4 Mean ( $\pm$ SE) dorsiflexion range of motion (ROM) before and after stretching. Significant increases in dorsiflexion ROM were found after contract-relax (CR 4.1°) and stretch-return-contract (SRC 4.0°) stretching. No difference was found in the changes in ROM between conditions. *Asterisk* significant to P < 0.01

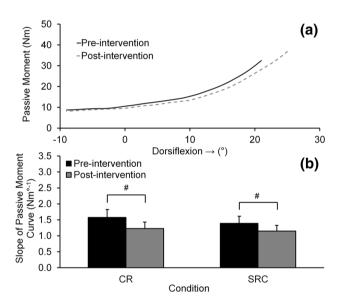
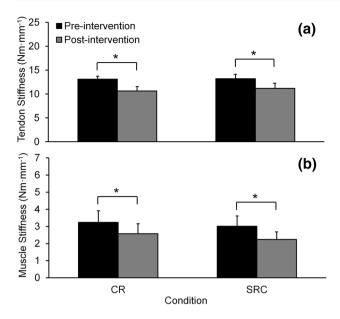


Fig. 5 Mean ( $\pm$ SE) passive plantar flexor moment before and after stretching. Passive moment (**a**) was reduced after stretching at all dorsiflexion angles along the joint moment–angle curve (one subject's data depicted during a contract-relax trial). Significant reductions in the slope of the passive moment curve (**b**) were found after contract-relax (CR 19.1 %) and stretch-return-contract (SRC 13.3 %) stretching. No difference was found in the changes in passive moment between conditions (P > 0.05). Hash symbol Significant to P < 0.05

during MVC [-11.5% (CI = -29.0, 6.0), P > 0.05; 3.0 % (CI = -8.4, 14.5), P > 0.05] or EMG activity at full ROM during the passive trial [8.4% (CI = -7.1, 23.8), P > 0.05; -4.9% (CI = -17.0, 7.1), P > 0.05] following the CR and SRC conditions, respectively. These data are indicative that neuromuscular force generating capacity and reflexive





**Fig. 6** Mean ( $\pm$ SE) Achilles tendon stiffness and gastrocnemius medialis (GM) muscle stiffness before and after stretching. Significant reductions in Achilles tendon stiffness (**a**) were observed after contract-relax (CR 20.4 %) and stretch-return-contract (SRC 15.7 %) stretching. Significant reductions in GM muscle stiffness (**b**) were found after CR (21.7 %) and SRC (21.3 %) stretching. No difference in the reductions in muscle and tendon stiffness was found between conditions (P > 0.05). *Asterisk* significant to P < 0.01

muscle activity were neither inhibited nor potentiated after either condition. However, the mean voluntary isometric plantar flexor moment during the four MVCs generated at full stretch during the CR condition (147.2  $\pm$  12.7 Nm) was significantly greater [10.6 % (CI = 7.1, 14.1); P < 0.05] than the moment produced in the anatomical position during the SRC condition (131.4  $\pm$  7.6 Nm), suggesting a greater tensile loading of the tendon during CR stretching compared with SRC stretching.

## Discussion

Contract-relax (CR) stretching has been commonly cited as the optimal stretching mode for achieving acute increases in ROM (Funk et al. 2003; Hindle et al. 2012), although the underlying mechanisms responsible for the efficacy of CR stretching to increase ROM remain to be established. Despite the efficacy of CR to acutely increase ROM, there is some concern that performing CR stretching can be painful and also increase muscle strain injury risk (Beaulieu 1981), which may partly explain why CR stretching is not as commonly used as static stretching for improving ROM. During muscular contractions sarcomere lengths are heterogeneous within single fibres and in different regions of the muscle, and this heterogeneity is exacerbated at long

muscle lengths where the muscle operates on the descending limb of the force-length curve (Macpherson et al. 1997). Importantly, greater tissue damage is reported following contractions performed at longer muscle lengths (Butterfield and Herzog 2006; Whitehead et al. 2003) with focal damage limited primarily to the overextended sarcomeres with no disruption at other locations in the muscle fibre or in adjacent fibres (Balnave et al. 1997). Notably, the contraction phase in CR stretching is performed with the muscle held in a highly stretched position, increasing the potential for tissue damage and reducing the tensile strength of connective tissue (Butterfield and Herzog 2006; Whitehead et al. 2003). A novel finding of the present study was that similar acute increases in ROM (~4°) were observed after both the CR and SRC techniques, despite the contraction phase being performed in the anatomical rather than highly-stretched position in SRC. Importantly, as the contractions were performed with the muscle 'off stretch' during the SRC technique in the present study the risk of microscopic subcellular damage leading to muscle strain injury is substantially reduced when compared to the traditional CR technique. Given that the muscle length adopted during the muscle contraction phase of CR stretching does not appear to influence the subsequent acute gain in ROM, nor the changes in mechanical or neuromuscular responses, the SRC technique may be useful for safely improving ROM when compared to the standard CR technique. Furthermore, from a practical perspective, the SRC technique is easier to implement because it does not require partner assistance.

In addition to the relative effects of CR and SRC stretching on ROM, the mechanical responses of the MTC were examined, and similar significant acute increases in ROM (~4°) and reductions in the slope of the passive moment curve (~13 and 19 %; indicative of reduced MTC stiffness) were found after both SRC and CR conditions, respectively. Changes in passive moment of this magnitude concomitant with increases in ROM have been previously reported after static stretching (Kay et al. 2015; Morse et al. 2008), although few studies have employed the necessary methodology to quantify tissue-specific changes. However, where ultrasonography has been employed to assess MTC stiffness in vivo, reductions in muscle but not tendon stiffness have been reported (Kay and Blazevich 2009a; Morse et al. 2008). The tissue-specific changes in stiffness following static stretching are probably expected as relaxed muscle is inherently more compliant than the tendon when measured under the current experimental conditions (Blazevich et al. 2014). Therefore, the relatively low forces transmitted through the MTC are predominately expected to influence muscle stiffness. Consistent with previous findings (Kay and Blazevich 2009a; Morse et al. 2008), the ultrasonography data in the present study revealed a reduced GM muscle stiffness



(~21 %) after both CR and SRC stretching, which can likely be explained by the static stretching phase being identical in both CR and SRC conditions. While reductions in muscle stiffness probably contributed to the ROM improvement, no significant correlation was found between increases in ROM and the reduction in muscle stiffness, which is indicative of other mechanisms more prominently underpinning the acute changes in ROM after CR and SRC stretching.

A distinct characteristic of CR stretching compared with other stretching techniques is the inclusion of an intense, often maximal, isometric contraction during the muscle stretch, which places substantial stress on muscular and (unique to CR stretching) tendinous tissues. In the present study, significantly lower forces were transmitted through the tendon during the contraction phase in the SRC condition when compared with the CR condition, which is likely a consequence of the plantar flexors operating largely on the ascending limb of the force-length curve according to their force-length properties (Maganaris 2001, 2003). However, similar reductions in Achilles tendon stiffness (~16 and 21 %) were found between conditions despite the reduced mechanical loading in the SRC condition. These data are consistent with a previous study (Kay et al. 2015) where a similar reduction in Achilles tendon stiffness (~22 %) was observed after an acute bout of CR stretching. Tendons have been shown to withstand substantially greater loading (Kay and Blazevich 2009b) and deformation (Waugh et al. 2012) during maximal contractions than those imposed by static stretching (Blazevich et al. 2014). Therefore, maximal ROM and passive resistance to stretch are most probably dictated by the muscle's tolerance to loading and deformation rather than the tendon's, as maximum tendon tolerance is not tested during most passive stretching protocols. The relative stiffness, and consequently the deformation, of muscle and tendon are distinct during low-velocity passive joint rotations towards maximal ROM (Blazevich et al. 2014; Morse et al. 2008), however the energy transfer through the tendon and muscle are identical as these tissues are arranged in series. Therefore, reductions in tendon stiffness will lower joint moment within the MTC and thus reduce tension within the muscle at a specific joint angle. Reductions in tendon stiffness have been reported following maximal isometric contractions performed without stretch (Kay and Blazevich 2009b; Kubo et al. 2001), with concomitant increases in ROM being reported that are equivalent to the gains observed after static stretching (Kay et al. 2015). Importantly, both CR and SRC stretching techniques cause an acute reduction in muscle and tendon stiffness, and this broader adaptive response may be an important adaptation that underpins the superior efficacy of CR stretching to acutely increase ROM compared with static stretching, which only influences muscle properties (Kay and Blazevich 2009a; Morse et al. 2008).

A possible limitation of the present study was that neuromuscular reflex characteristics (e.g., M-wave/H-reflex characteristics) were not fully examined, although EMG amplitude as a measure of α-motoneuron pool reflex activity was measured at full ROM. However, the rotation velocity employed in the present study was intentionally designed to be too slow to initiate a significant myotatic reflex response (McNair et al. 2001), thus any changes in ROM should not be attributable to inhibition of the  $\alpha$ -motoneuron pool. This was confirmed by the lack of any substantial EMG activity (<5 % MVC) at full ROM in both the pre- and postintervention data, or significant change in EMG activity post-intervention. Thus, as no substantial activation of the musculature occurred, changes in maximum ROM could not be notably influenced by Ib input, thus autogenic inhibition is not likely to be an important mechanism underpinning the increase in ROM following CR stretching in the present study. These data are similar to those reported in previous acute CR studies where EMG magnitude was unchanged at full ROM (Kay et al. 2015; Mitchell et al. 2009; Osternig et al. 1990). However, further study using a wider array of neuromuscular analyses at faster stretch velocities is needed to fully determine the possible role of autogenic inhibition as a mechanism underpinning the efficacy of CR stretching to increase ROM. Notwithstanding, a neurological contribution is at least partly supported by the increase in peak passive joint moment (~13 %) and the strong correlations found between changes in peak passive moment (indicative of improved stretch tolerance) and ROM ( $r_s = 0.63-0.71$ ; P < 0.01) after both CR and SRC stretching. These data are consistent with previous studies (Kay et al. 2015; Mitchell et al. 2009) where increased stretch tolerance was observed following an acute bout of CR stretching. Collectively, these data indicate that whilst inhibition of the  $\alpha$ -motoneuron pool did not occur, altered pain perception is likely an important mechanism that influence ROM changes after CR stretching, however the specific neuromuscular pathways remains to be established.

# **Conclusions**

In summary, a significant increase in ROM with reductions in both muscle and tendon stiffness and a concomitant increase in stretch tolerance were demonstrated after both CR and SRC stretching. Furthermore, the changes in ROM were significantly correlated with changes in stretch tolerance but not changes in muscle, tendon, or whole MTC stiffness. Thus, while mechanical changes in the muscle and tendon may have influenced ROM, changes in stretch tolerance may more strongly underpin the acute increases in ROM. The present study is the first to examine the effect of performing the contraction phase of CR stretching with



the muscle 'off stretch'. As no differences in the changes in any measure were evident between conditions, it is likely that similar mechanisms were responsible for the (comparable) increases in ROM in CR and SRC conditions. regardless of the muscle length at which the contractions were performed. This novel finding is practically important as performing the contractions in the anatomical position (i.e., off stretch) is equally effective as CR but can be performed without partner assistance, is painless and reduces the risk of muscle damage as the contractions are performed at a shorter muscle length, therefore SRC offers a safer yet equally effective stretching model. These practical improvements may improve the capacity of individuals, coaches and clinicians to facilitate the use of this stretching mode as part of a complete injury prevention strategy in healthy and in at-risk populations in both athletic and clinical settings.

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#### Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

#### References

- Avela J, Kyröläinen H, Komi PV (1999) Altered reflex sensitivity due to repeated and prolonged passive muscle stretching. J Appl Physiol 86:1283–1291
- Avela J, Finni T, Liikavainio T, Niemela E, Komi PV (2004) Neural and mechanical responses of the triceps surae muscle group after 1 h of repeated fast passive stretches. J Appl Physiol 96:2325– 2332. doi:10.1152/japplphysiol.01010.2003
- Balnave CD, Davey DF, Allen DG (1997) Distribution of sarcomere length and intracellular calcium in mouse skeletal muscle following stretch-induced injury. J Physiol 502:649–659. doi:10.1111/j.1469-7793.1997.649bj.x
- Bassey EJ, Morgan K, Dallosso HM, Ebrahim SB (1989) Flexibility of the shoulder joint measured as range of abduction in a large representative sample of men and women over 65 years of age. Eur J Appl Physiol Occup 58:353–360. doi:10.1007/ BF00643509
- Beaulieu JE (1981) Developing a stretching program. Phys Sportsmed 9:59–69
- Blazevich AJ, Cannavan D, Waugh C, Fath F, Miller S, Kay AD (2012) Neuromuscular factors influencing the maximum stretch limit of the human plantar flexors. J Appl Physiol 113:1446– 1455. doi:10.1152/japplphysiol.00882.2012
- Blazevich AJ, Cannavan D, Waugh C et al (2014) Range of motion, neuromechanical and architectural adaptations to plantar flexor stretch training in humans. J Appl Physiol 17:452–462. doi:10.1152/japplphysiol.00204.2014
- Butterfield TA, Herzog W (2006) Effect of altering starting length and activation timing of muscle on fiber strain and muscle damage. J Appl Physiol 100:1489–1498. doi:10.1152/japplphysiol.00524.2005
- Cresswell AG, Loscher WN, Thorstensson A (1995) Influence of gastrocnemius muscle length on triceps surae torque development

- and electromyographic activity in man. Exp Brain Res 105:283–290. doi:10.1007/BF00240964
- Duffin AC, Donaghue KC, Potter M et al (1999) Limited joint mobility in the hands and feet of adolescents with type 1 diabetes mellitus. Diabet Med 16:125–130. doi:10.1046/j.1464-5491.1999.00030.x
- Funk DC, Swank AM, Mikla BM, Fagan TA, Farr BK (2003) Impact of prior exercise on hamstring flexibility: a comparison of proprioceptive neuromuscular facilitation and static stretching. J Strength Cond Res 17:489–492
- Herda TJ, Costa PB, Walter AA, Ryan ED, Cramer JT (2014) The time course of the effects of constant-angle and constant-torque stretching on the muscle–tendon unit. Scand J Med Sci Sports 24:62–67. doi:10.1111/j.1600-0838.2012.01492.x
- Hindle KB, Whitcomb TJ, Briggs WO, Hong J (2012) Proprioceptive neuromuscular facilitation (PNF): its mechanisms and effects on range of motion and muscular function. J Hum Kinet 31:105–113. doi:10.2478/v10078-012-0011-v
- Karamanidis K, Stafilidis S, DeMonte G, Morey-Klapsing G, Bruggemann G, Arampatzis A (2005) Inevitable joint angular rotation affects muscle architecture during isometric contraction. J Electromyogr Kines 15:608–616. doi:10.1016/j.jelekin.2005.02.001
- Kawakami Y, Ichinose Y, Fukunaga T (1998) Architectural and functional features of human triceps surae muscles during contraction. J Appl Physiol 85:398–404
- Kay AD, Blazevich AJ (2008) Reductions in active plantarflexor moment are significantly correlated with static stretch duration. Eur J Sport Sci 8:41–46. doi:10.1080/17461390701855505
- Kay AD, Blazevich AJ (2009a) Moderate-duration static stretch reduces active and passive plantar flexor moment but not Achilles tendon stiffness or active muscle length. J Appl Physiol 106:1249–1256. doi:10.1152/japplphysiol.91476.2008
- Kay AD, Blazevich AJ (2009b) Isometric contractions reduce plantar flexor moment, Achilles tendon stiffness and neuromuscular activity but remove the subsequent effects of stretch. J Appl Physiol 107:1181–1189. doi:10.1152/japplphysiol.00281.2009
- Kay AD, Husbands-Beasley J, Blazevich AJ (2015) Effects of contract–relax, static stretching, and isometric contractions on muscle–tendon mechanics. Med Sci Sport Exer 47:2181–2190. doi:10.1249/MSS.00000000000000632
- Kubo K, Kanehisa H, Ito M, Fukunaga T (2001) Effects of isometric training on the elasticity of human tendon structures in vivo. J Appl Physiol 91:26–32
- Kubo K, Kanehisa H, Fukunaga T (2002) Effects of transient muscle contractions and stretching on the tendon structures in vivo. Acta Physiol Scand 175:157–164. doi:10.1046/j.1365-201X.2002.00976.x
- Macpherson PCD, Dennis RG, Faulkner JA (1997) Sarcomere dynamics and contraction-induced injury to maximally activated single muscle fibres from soleus muscles of rats. J Physiol 500:523–533. doi:10.1113/jphysiol.1997.sp022038
- Maganaris CN (2001) Force–length characteristics of in vivo human skeletal muscle. Acta Physiol Scand 172:279–285. doi:10.1046/j.1365-201x.2001.00799.x
- Maganaris CN (2003) Force–length characteristics of the in vivo human gastrocnemius muscle. Clin Anat 16:215–223. doi:10.1002/ca.10064
- Magnusson SP, Simonsen EB, Aagaard P, Sorensen H, Kjaer M (1996) A mechanism for altered flexibility in human skeletal muscle. J Physiol 497:291–298. doi:10.1113/jphysiol.1996. sp021768
- Manal K, Cowder JD, Buchanan TS (2013) Subject-specific measures of Achilles tendon moment arm using ultrasound and videobased motion capture. Physiol Rep 1:e00139. doi:10.1002/ phy2.139
- Mazzullo JM (1978) The gate theory of pain. Br Med J 2:586-587



- McNair PJ, Dombroski EW, Hewson DJ, Stanley SN (2001) Stretching at the ankle joint: viscoelastic responses to holds and continuous passive motion. Med Sci Sport Exer 33:354–358
- Melzack R (1993) Pain: past, present and future. Can J Exp Psychol 47:615–629
- Mitchell UH, Myrer JW, Hopkins JT, Hunter I, Feland JB, Hilton SC (2007) Acute stretch perception alteration contributes to the success of the PNF "contract-relax" stretch. J Sport Rehabil 16:85–88
- Mitchell UH, Myrer JW, Hopkins JT, Hunter I, Feland JB, Hilton SC (2009) Neurophysiological reflex mechanisms' lack of contribution to the success of PNF stretches. J Sport Rehabil 18:343–357
- Morse CI, Degens H, Seynnes OR, Maganaris CN, Jones AJ (2008) The acute effect of stretching on the passive stiffness of the human gastrocnemius muscle tendon unit. J Physiol 586:97–106. doi:10.1113/jphysiol.2007.140434
- Mulholland SJ, Wyss UP (2001) Activities of daily living in non-Western cultures: range of motion requirements for hip and knee joint implants. Int J Rehabil Res 24:191–198
- Murray MP, Guten GN, Baldwin JM, Gardener GM (1976) A comparison of plantar flexion torque with and without the triceps surae. Acta Orthop Scand 47:122–124. doi:10.3109/17453677608998984
- Osternig LR, Robertson RN, Troxel RK, Hansen P (1990) Differential responses to proprioceptive neuromuscular facilitation (PNF) stretch techniques. Med Sci Sport Exer 22:106–111

- Prentice W (1983) A comparison of static stretching and PNF stretching for improving hip joint flexibility. J Athl Train 18:56–59
- Proske U, Morgan DL (1999) Do cross-bridges contribute to the tension during stretch of passive muscle? J Muscle Res Cell M 20:433–442. doi:10.1023/A:1005573625675
- Sharman MJ, Cresswell AG, Riek S (2006) Proprioceptive neuromuscular facilitation stretching: mechanisms and clinical implications. Sports Med 36:929–939. doi:10.2165/00007256-200636110-00002
- Waugh CM, Blazevich AJ, Fath F, Korff T (2012) Age-related changes in mechanical properties of the Achilles tendon. J Anat 220:144–155. doi:10.1111/j.1469-7580.2011.01461.x
- Weppler CH, Magnusson SP (2010) Increasing muscle extensibility: a matter of increasing length or modifying sensation? Phys Ther 90:438–449. doi:10.2522/ptj.20090012
- Whitehead NP, Morgan DL, Gregory JE, Proske U (2003) Rises in whole muscle passive tension of mammalian muscle after eccentric contractions at different lengths. J Appl Physiol 95:1224–1234. doi:10.1152/japplphysiol.00163.2003
- Witvrouw E, Danneels L, Asselman P, D'Have T, Cambier D (2003) Muscle flexibility as a risk factor for developing muscle injuries in male professional soccer players. A prospective study. Am J Sports Med 31:41–46

