Plantarflexor Stretch Training Increases Reciprocal Inhibition Measured During Voluntary Dorsiflexion

Running title: Stretch training increases reciprocal inhibition

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ABSTRACT

Agonist-mediated reciprocal inhibition in distal skeletal muscles is an important neurophysiological phenomenon leading to improved movement coordination and efficiency. It has been shown to be reduced in aged and clinical populations, so the development of interventions augmenting reciprocal inhibition is an important research goal. We examined the efficacy of using chronic passive muscle stretching to augment reciprocal inhibition. The influence of 3 weeks of plantar flexor stretching (4×30 s, 2·day⁻¹) on reciprocal inhibition of soleus and gastrocnemius initiated by tonic, voluntary dorsiflexion contractions (20% of maximum voluntary moment [MVC]) was examined in 11 healthy men who performed stretch training and in 9 non-training controls. H reflexes were elicited by tibial nerve stimulation during both weak isometric (2% MVC) plantar flexions and dorsiflexion contractions at 20% MVC. Changes were examined at three joint angles, normalized to each subject’s range of motion (plantarflexed = 10 ± 0°, neutral = -3.3 ± 2.9°, dorsiflexed = -16.5 ± 5.6°). No changes were detected in controls. A 20% increase in range of motion in the stretch subjects was associated with a significant decrease in $H_{max}:M_{max}$ measured during 2% plantar flexion at the plantarflexed and neutral angles in soleus and at the plantarflexed angle in gastrocnemius ($p<0.05-0.01$). By contrast, decreases in $H_{max}:M_{max}$ during 20% dorsiflexion contract were also seen at each angle in soleus and at the dorsiflexed angle in gastrocnemius. However, a greater decrease in $H_{max}:M_{max}$ measured during voluntary dorsiflexion than during plantar flexion, which indicates a specific change in RI, was detected only at the dorsiflexed angle (-30.7 ± 9.4% and -35.8 ± 6.8% for soleus and gastrocnemius, respectively). These results demonstrate the efficacy of soleus-gastrocnemius stretch training in increasing agonist-mediated reciprocal inhibition from TA onto soleus-gastrocnemius in young healthy individuals at dorsiflexed, but not plantarflexed, joint angles.

Key Words: Static stretch, electrical stimulation, tibial nerve, soleus, skeletal muscle
INTRODUCTION

Coordinated interactions between the triceps surae and pre-tibial muscles are integral in the performance of quiet standing, walking, running and jumping (Fitzpatrick et al. 1992; Lavoie et al. 1997; Petersen et al. 1999; Stein and Thompson 2006; Tokuno et al. 2009). For example, foot clearance during walking requires the activation of dorsiflexion muscles, in particular tibialis anterior, whilst activity of its antagonists is reduced. Thus, failure to maintain reciprocal inhibitory inputs derived from the tibialis anterior onto soleus (i.e. reciprocal inhibition; RI) would be a major factor limiting walking performance.

RI in humans is commonly studied by comparing the amplitude of the electrically-evoked Hoffmann (H-) reflex in a distal agonist muscle whilst activity in an antagonist is simultaneously elicited by nerve stimulation or voluntary activation (Crone et al. 1987; Crone and Nielsen 1989). It has been shown that RI is variably increased or decreased in patients with Parkinson’s disease (Delwaide et al. 1993; Lelli et al. 1991; Meunier et al. 2000), cerebral palsy (Leonard et al. 1990) and multiple sclerosis (Crone et al. 1994) or in patients with spinal cord or brain lesions caused by spinal cord trauma or stroke leading to spasticity (Crone et al. 1994; Lamy et al. 2009; Nakashima et al. 1989; Yanagisawa et al. 1976). RI has also been shown to decrease with age (Hortobagyi et al. 2006) and may well contribute to reduced movement performance in aged and clinical populations. The development of intervention strategies to improve or maintain the integrity of the neural pathways controlling RI is therefore of substantial clinical and scientific interest.

Researchers have clearly demonstrated the efficacy of using physical training interventions to augment RI in both normal and clinical populations (Crone et al. 1994; Geertsen et al. 2008; Perez et al. 2007; Thompson et al. 2009). Intriguingly, however, researchers have yet to
examine the effects of chronic stretching training on RI augmentation in either healthy or clinical individuals. Investigations of spinal excitability during acute agonist muscle stretch have shown a reduced efficacy of the Ia-afferent pathway, as assessed by a reduction in H-reflex amplitude, which may reflect a reduction in the excitability of the homonymous motor neuron pool (Guissard et al. 2001; 1988). Furthermore, longer-term (4 weeks) stretching training has been shown to reduce resting H-reflex amplitude in normal subjects (Guissard and Duchateau 2004), suggesting that a course of stretching can induce adaptations in spinal circuitry function.

In the present study, the effects of longitudinal (3 weeks) passive triceps surae stretching on TA-mediated RI, measured as a change in soleus and gastrocnemius H-reflex amplitudes elicited during low-level voluntary dorsiflexion contractions, were examined. In order to isolate the effects of RI mechanisms from the direct effects of stretching on Ia synaptic transmission and α-motor neuron pool excitability, these responses were compared to H-reflexes obtained during low-level (2% of peak moment) plantar flexion contractions. Finally, as joint angles vary throughout normal human movements and the influence of afferent feedback is joint angle-dependent (Baxendale and Ferrell 1981; Hwang 2002), RI responses were examined at three joint angles ranging from plantar flexion to dorsiflexion.

METHODS

Subjects

Twenty-two men (age = 18.7 ± 0.8 yr, height = 1.81 ± 0.06 m, mass = 73.4 ± 7.1 kg) with no reported neuromuscular or inflammatory disorders or lower limb musculoskeletal injury volunteered for the study. The subjects were randomly assigned to either a stretch training or a non-training control group. Two control subjects did not report for post-intervention testing,
so the stretch training and control groups contained 11 and 9 subjects, respectively. All
volunteers read and signed an informed consent document, the procedures were conducted in
accordance with the declaration of Helsinki and the study was approved by the University’s
Human Ethics Committee.

Overview

All subjects completed three testing blocks. The first block acted as a familiarization period
where they reported on 2-3 occasions to practice the moment targeting procedures (described
later) and become familiar with the stimulation protocols. During these sessions, estimates of
tolerable stimulation intensities were obtained. The second and third testing blocks
corresponded with the pre-and post-intervention testing periods, each of which required two
sessions: 1) passive ankle range of motion testing, and 2) measurement of H- and M-wave
amplitudes in soleus and gastrocnemius while performing a 2% maximal voluntary
contraction (MVC) plantar flexion and a 20% MVC dorsiflexion. These sessions were
performed at the same time of day on separate days and were each preceded by a warm-up
involving a 5-min cycle at 60 rpm with a 1-kg load on a Monark cycle ergometer (Monark
Exercise AB, Sweden) and four isometric plantar- and dorsiflexion contractions performed at
50, 70, 90 and 100% of perceived maximum effort at a 0° ankle angle (plane of foot
perpendicular to a line between lateral malleolus and lateral condyle). No intense exercise
was allowed in the 48 hours preceding testing, and no stimulants or depressants (e.g. caffeine,
alcohol) were allowed within 6 hours.

Stretch training

The experimental group performed four 30-s calf muscle stretches twice a day (morning and
night) for three weeks (22 days/44 sessions); self-reported compliance was 91.8 ± 3.8% (40.4
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± 1.6 sessions). Stretching was performed by leaning against a wall with one foot in front of the other and the back leg (knee) straight such that a ‘strong’ stretch of the triceps surae was elicited (Alter 2004). This stretch exercise was chosen as it can be easily performed by individuals, without the need for assistance. All subjects had exercise backgrounds and were able to perform the stretch well.

2.4 Passive ankle range of motion (maximal stretch tolerance) test

The subject’s sat in an isokinetic dynamometer (Biodex System 3, Biodex Medical, NY, USA) with the knee of their test leg fully extended and their trunk at 105° to their thigh (the slightly reclined position was used to minimize the tension felt at the back of the knee during dorsiflexion and allowed subjects to comfortably stretch the ankle joint). The foot was strapped firmly to the dynamometer’s ankle plantar/dorsiflexion footplate, with the lateral malleolus aligned with the axis of the dynamometer. Additional strapping was used to minimize foot movement from the footplate whilst allowing comfortable ankle rotation to maximum dorsiflexion. With the subject’s eyes closed and the ankle starting from a position of 20° plantar flexion the dynamometer was programmed to rotate the ankle into dorsiflexion at 2°·s⁻¹. The subjects depressed a stop button upon reaching maximum tolerable stretch, which immediately released the footplate and allowed the ankle to rotate back into plantar flexion. Two trials were allowed, with a 1-minute rest interval, and the greatest range of motion was subsequently used for analysis.

H- and M-wave amplitude test

H- and M-wave amplitudes were measured in soleus and gastrocnemius with the ankle joint in each of three positions while the subjects were seated on the dynamometer in a quiet, temperature controlled (23° C) room. To account for variability in each subject’s ankle range
Stretch training increases reciprocal inhibition of motion, and to ensure parity of the stretch intensity between subjects (in pilot testing we noted that passive torque and subjective ratings of passive stretch were very different at the same joint ranges of motion, and these elicited very different reflex responses), ankle positions were set at 0, 30 and 60% of the joint range between 10° plantar flexion (i.e. minimal passive moment recorded in any subject) and the angle at which 90% of peak passive tension was obtained in the passive range of motion test, measured at pre-training. Testing was not performed at 90% of ankle range of motion as pilot testing showed that the reliability of obtaining H-reflex and M-wave recruitment curves was lower toward the end range of motion. On average, the three angles were: 1) 10 ± 0°, 2) -3.3 ± 2.9° and 3) -16.5 ± 5.6° (hereafter, these will be referred to as plantarflexed, neutral and dorsiflexed joint angles). Post-training testing was performed at the same absolute joint angles. Testing always progressed from plantar- to dorsiflexed angles to minimize the possible effects of muscle stretch on subsequent tests.

Electrical Stimulation

Stimulations were delivered using a constant current stimulator (DS7A, Digitimer Ltd, Hertfordshire, UK) to the tibial nerve using square pulses of 1-ms duration at random intervals always longer than 10 s. The anode (3.2-cm dia., fabric gel adhesive electrode, Valutrode CF3200, Nidd Valley Medical Ltd., UK) was placed on the patella. The cathode was placed in the popliteal fossa overlying the nerve at a position that provided the greatest H-wave amplitude at a small stimulus intensity; this position was found by stimulating at different sites over the skin’s surface with relatively low current. Electrodes were secured to the skin with zinc oxide tape to prevent movement during testing. Stimulus intensity gradually increased from near zero to an intensity where a clear plateau was observable in the peak-to-peak amplitude of the M-wave, as recorded by surface electromyographic (EMG)
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electrodes placed over the soleus and gastrocnemius muscles (see below). Additional stimuli were delivered around the intensity at which the H-reflex amplitude appeared maximal, as shown in Figure 1, to more accurately determine the maximum H-reflex ($H_{\text{max}}$) amplitude.

EMG recording

EMG activity of the soleus and gastrocnemius muscles was recorded using self-adhesive neonatal electrodes with a 10-mm diameter recording surface (28-mm diameter adhesive surface, Tyco Healthcare, Neustadt, Germany). The electrodes were placed in a pseudo-monopolar configuration with one electrode placed each on the soleus (medial aspect, distal to gastrocnemius) and medial gastrocnemius muscle bellies whilst a second electrode was placed approximately 10-cm proximal to the Achilles tendon insertion onto the calcaneum, just above Kager’s triangle (Hadoush et al. 2009; Pinniger et al. 2001). A reference electrode was placed on the lateral malleolus. The electrodes were connected to a 1902 isolated differential preamplifier (Cambridge Electronic Design, Cambridge, UK) with an input impedance of 10,000 M$\Omega$ and common mode rejection exceeding 100 dB. The data were analogue-to-digitally converted at a sampling rate of 2000 Hz, acquired using a Power1401 data acquisition interface and stored on a computer running Signal software (CED, Cambridge, UK). Permanent skin markings were used to ensure accurate replacement of electrodes at post-intervention testing.

H-reflex measurement at rest and during active isometric dorsiflexion

H-reflex amplitudes in response to the electrical stimulations were measured in two conditions, when the subject had achieved a stable voluntary 1) plantar flexor moment at 2% of the maximum achievable at the specific joint angle, and then 2) dorsiflexor moment at 20% of the maximum achievable at the specific joint angle. The use of a 2% MVC
 contraction was shown in pilot testing to improve the reliability of measurements above that
 achieved at absolute rest, but have little effect on H-reflex amplitudes. Maximum joint
 moments were obtained during isometric contractions at each angle performed 3 min prior to
 the stimulations. Targeting a specific moment rather than integrated EMG level has been
 used previously (Meunier et al. 2000) and the normalized level of EMG was the same at pre-
 and post-intervention, however between-angle comparisons were not done because of the
 possibility of small within-subject variability in the neural drive required to achieve the given
 joint moment. Condition 1 (2% plantar flexion moment), which was used to test Ia afferent
 transmission onto the efferent motor neuron pool (soleus-gastrocnemius), was always
 completed before condition 2, which was used to test the level of voluntary antagonist-driven
 RI (Crone and Nielsen 1989). Each condition involved the delivery of approximately 18-30
 stimuli. At the end of testing, a single maximal voluntary plantar flexion contraction was
 performed at the first (10°) angle and compared to the value at the commencement of testing.
 The peak isometric joint moments were within ±5% of each other suggesting that no
 considerable fatigue was induced by the test protocol.

 Data analysis

 The mean of the three largest peak-to-peak (non-rectified) H-reflex and M-wave amplitudes
 were used in each condition to compute a H$_{max}$/M$_{max}$ ratio (H$_{max}$:M$_{max}$). Changes in maximum
 dorsiflexion range of motion (i.e. flexibility) were tested by a separate two-way ANOVA
 with repeated measures (group × time). The change in RI was then taken as the difference
 between the changes in H$_{max}$:M$_{max}$ over time between 2% plantar flexion and 20%
 dorsiflexion conditions for each individual, which was computed using the equation:

 \[
 \%\Delta H: M_{pre-post}^{20\%\text{dorsiflexion}} - \%\Delta H: M_{pre-post}^{2\%\text{plantarflexion}} \quad \text{[eq. 1]}
 \]
where a negative score indicates that the reduction in H\textsubscript{max}:M\textsubscript{max} in the dorsiflexion contractions over the training period was greater than the reduction in H\textsubscript{max}:M\textsubscript{max} in the plantarflexion contractions (i.e. an increase in RI). Changes were examined by ANOVA. This variable allowed the determination of the influence of reciprocal inhibition on the change in H\textsubscript{max}:M\textsubscript{max}. Bonferroni post-hoc tests were used to determine the location of any significant differences. Significance for all tests was accepted at an alpha level of 0.05.

RESULTS

Changes in ankle joint range of motion, passive torque and peak isometric torque

The experimental (stretch training) group increased their maximum dorsiflexion range of motion, measured from 10° plantarflexion, by 7.9 ± 4.7° (40.6 ± 10.0° to 48.5 ± 10.9°; 20 ± 11%; p<0.001) over the 3-week period. There was no change in the control group (2.7 ± 3.0°, 8 ± 10%). Passive torque recorded at each joint angle did not change for the experimental (pre- vs. post-training: 0.3 ± 0.9 vs. 0.5 ± 0.9 Nm, 6.8 ± 1.4 vs. 5.9 ± 1.8 Nm and 18.3 ± 4.2 vs. 15.0 ± 3.6 Nm, at plantarflexed, neutral and dorsiflexed angles, respectively) or control (1.0 ± 1.0 vs. -0.1 ± 0.2 Nm, 6.7 ± 1.7 vs. 5.4 ± 1.7 Nm and 16.5 ± 4.6 vs. 14.3 ± 4.0 Nm) groups. Also, the training did not alter peak isometric plantarflexor torque in the experimental (pre- vs. post-training: 149 ± 39 vs. 152 ± 36 Nm, 202 ± 41 vs. 205 ± 39 Nm and 244 ± 47 vs. 248 ± 46 Nm, at plantarflexed, neutral and dorsiflexed angles, respectively) or control (161 ± 32 vs. 150 ± 33 Nm, 208 ± 34 vs. 198 ± 45 Nm and 236 ± 50 vs. 242 ± 61 Nm) groups.

M wave amplitude at H\textsubscript{max}

Although there was some variation between subjects and between muscles (i.e. soleus vs. gastrocnemius) in the peak-to-peak amplitude of the direct M response recorded at H\textsubscript{max} (and...
normalized relative to $M_{\text{max}}$ there was no systematic change detected after the stretch training intervention, indicating that identical stimulation conditions were achieved at all test sessions (data not shown).

**$H_{\text{max}}:M_{\text{max}}$ amplitude during 2% MVC plantar flexion**

After stretch training, a decrease in the H-reflex amplitude was responsible for a significant decrease in $H_{\text{max}}:M_{\text{max}}$ (see Figure 2), although this effect was only significant when measured at plantarflexed (-16.8 ± 3.3%, $p<0.001$) and neutral (-15.2 ± 5.6%, $p<0.001$) angles in soleus and the plantarflexed angle in gastrocnemius (-10.6 ± 5.5%, $p<0.05$); there were no changes in M-wave amplitude. There was no change in $H_{\text{max}}:M_{\text{max}}$ at the dorsiflexed joint angle after training in either muscle, and there was no change in the control group.

**$H_{\text{max}}:M_{\text{max}}$ amplitude during 20% MVC voluntary dorsiflexion**

Stretch training resulted in a substantial reduction in $H_{\text{max}}:M_{\text{max}}$ (Figure 3) measured during 20% MVC voluntary dorsiflexion at all joint angles in soleus (-17.3 ± 6.0%, -31.3 ± 8.6% and -29.3 ± 8.6%, at plantarflexed, neutral and dorsiflexed joint angles, respectively). A similar trend was seen in gastrocnemius, although mean decreases observed in plantarflexed and neutral angles (-13.0 ± 7.5% and -19.6 ± 9.1%, respectively; $p<0.1$) were not as great as the decrease measured at the dorsiflexed angle (-25.5 ± 9.3%, $p<0.05$). $H_{\text{max}}:M_{\text{max}}$ did not change at any ankle angle in the control group.

**Difference in $\%H_{\text{max}}:M_{\text{max}}$ changes between conditions (2% plantar flexion vs. 20% dorsiflexion)**

Before training, $H_{\text{max}}:M_{\text{max}}$ was smaller at all angles during the dorsiflexion contraction when compared to 2% plantar flexion in soleus (30.3%, 36.5% and 28.8% of 2% plantarflexion
condition at plantarflexed, neutral and dorsiflexed angles, respectively) and gastrocnemius
(32.3%, 45.0% and 35.6%). This difference largely persisted after the stretch training and
indicates a significant effect of voluntary dorsiflexion on plantar flexor $H_{\text{max}}:M_{\text{max}}$.

The specific effect of dorsiflexor activation on $H_{\text{max}}:M_{\text{max}}$ depression resulting from the
stretch training was examined by calculating the difference between the % change in
$H_{\text{max}}:M_{\text{max}}$ measured during the 20% dorsiflexion contraction and that measured during the
2% plantar flexion contraction (Eq. 1). As shown in Figure 4, the slightly greater decrease in
$H_{\text{max}}:M_{\text{max}}$ measured during active dorsiflexion than measured during 2% plantarflexion at
the plantarflexed joint angle was not statistically significant. However, a greater decrease in
the % change in $H_{\text{max}}:M_{\text{max}}$ was seen during the dorsiflexion contractions at the dorsiflexed
joint angle in both soleus (-30.7 ± 9.4%, $p<0.01$) and gastrocnemius (-35.8 ± 6.8%, $p<0.01$),
indicating a change in agonist-mediated (TA) RI in antagonist (soleus-gastrocnemius) motor
neurons. No changes were seen in the control group.

DISCUSSION

Although both increases and decreases reciprocal inhibition (RI) have been reported in
clinical subjects, a reduced RI of the triceps surae muscle group during voluntary dorsiflexion
activation, as seen in aged and clinical populations, may well be problematic for the
performance of important daily tasks such as standing and walking (Chalmers and Knutzen
2000; Cramp 1998; Wolpaw and Tennissen 2001). Here, we examined the efficacy of using
chronic passive muscle stretching to augment agonist-mediated RI, measured during
voluntary dorsiflexor (tibialis anterior, TA) muscle contractions in young, healthy subjects.
The major finding of the present study was that the H-M wave ratio ($H_{\text{max}}:M_{\text{max}}$) measured in
both soleus and gastrocnemius during voluntary dorsiflexion was reduced substantially after
the 3-week stretching intervention, suggesting that a rapid re-organization of spinal circuitry occurred with the training that increased RI. The similar responses measured in both soleus and gastrocnemius indicates that, at least when measured in full knee extension, changes in spinal circuitry have a collective influence on the excitability of both motor neuron pools and/or on the magnitude of presynaptic inhibition of Ia afferents. Uniquely, we examined H-reflex changes both during a low-level plantar flexion contraction (2% MVC) and during voluntary dorsiflexion (20% MVC) and found an additional effect during the dorsiflexion trials. This is clearly indicative of mechanisms in addition to those that normally lead to a decreased influence of plantar flexor Ia afferent transmission onto the motor neuron pool causing a reduced H-reflex amplitude.

Although there are practical benefits to the use of voluntary rather than elicited dorsiflexor muscle activation, one drawback is that it is not possible to clearly establish the mechanisms underpinning the increased RI. One possibility is that the stretching training altered muscle-tendon mechanical properties and/or reduced muscle spindle gain in response to stretch (Guissard and Duchateau, 2004), which could have induced bias in favor of elevated soleus-gastrocnemius disfacilitation. This would have reduced the magnitude of the stretch reflex and favored the normal RI condition. However, such changes would be reflected in a change in $H_{\text{max}}:M_{\text{max}}$ measured in the 2% plantarflexion contraction also, so the additional changes seen in the 20% dorsiflexion contraction cannot be completely explained by this mechanism. Another hypothesis is that changes in the supraspinal drive from cortico- or rubrospinal (or, less likely, vestibulospinal) tracts or Ib excitation during the agonist muscle contraction (Cavallari et al. 1985; Day et al. 1983; Hultborn et al. 1987; Mercuri et al. 1997; Rothwell et al. 1984) reinforced the descending inhibitory control to antagonist-coupled interneurons (Hongo et al. 1984; Nielsen and Kagamihara 1992), i.e. there was a change in ionotropic
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input to the Ia reciprocal interneuron. However, although there is some limited evidence in baboons (Hongo et al. 1984), there are no definitive data to our knowledge detailing the adaptive process in humans. Another possibility is that cortico- and rubrospinal-mediated changes may have occurred. The use of identical body and head positions and exact replication of the testing protocols in both 20% dorsiflexion and 2% plantar flexion conditions would have minimized corticovestibular influences, so signals associated with dorsiflexor activation, or emanating from dorsiflexor afferents, are most the likely influence.

Finally, it should also be considered possible that there was a change in the excitability of the Ia reciprocal interneuron itself. This could possibly be mediated through variations in neuromodulatory (Heckman et al. 2008; Heckman et al. 2009) control from brainstem-derived descending tracts (Heckman et al. 2008; Heckman et al. 2009), which would provide a monoaminergic influence increasing interneuron excitability. It is well known that norepinephrine has a substantial effect on spinal interneurons (Skoglund 1961) and both norepinephrine and serotonin have been shown to facilitate interneurons mediating reciprocal inhibition from Ia afferents (Jankowska et al. 2000). Mitigating against this possibility, however, is the evidence that Ia facilitation is not as great as in the motoneurons themselves (Hammar and Jankowska 2003; Jankowska et al. 2000), and it is currently not known whether monoaminergic control of interneurons (or, in fact, motoneurons) is affected by muscle stretching. However, this is a mechanism worthy of future examination. Regardless of the mechanisms responsible, these data show, for the first time, that passive stretch training results in a long-lasting (at least days) increase in voluntarily-initiated RI in humans, and that this adaptation is additional, i.e. separate, to the Ia-mediated transmission depression or changes in general motor neuron excitability.
From a practical perspective, the finding that the increases in RI were only clearly manifested at the dorsiflexed angle (see Figure 4) is important. The influence of stretching training on RI about the ankle joint was greatest when the plantarflexors were in a lengthened position and the dorsiflexors were shortened. In this configuration the plantarflexors apply a more substantial passive resistive moment (Guissard and Duchateau 2004; Kay and Blazevich 2009) and the dorsiflexors apply a weaker active moment (Koh and Herzog 1995), according to their passive and active force-length relations, respectively. It is at these ankle positions that older individuals and clinical populations with dorsiflexion difficulties (e.g. foot drop) will likely be most affected and thus locomotor performance can be most impaired (Petersen et al. 1999), so the results underline the potential for chronic stretch training to have practically meaningful effects on movement performance. Further research is therefore warranted in order to test whether these joint angle-specific increases in RI substantially influence ankle joint kinematics during locomotor and other complex tasks.

A novel aspect of the present study was that differences between the changes in the maximal H\textsubscript{max}:M\textsubscript{max} responses measured (at a particular joint angle) during active dorsiflexion and a low-level of plantar flexion contraction were calculated (see Figure 4). This allowed us to determine the specific effects of stretch on RI separately to its effect on Ia afferent transmission to the motor neurons. In fact, we found that the effects of stretch on H\textsubscript{max}:M\textsubscript{max} measured during low-level plantar flexion were noticeable only when the plantarflexor muscles were not substantially stretched (i.e. plantarflexed and neutral angles; Figure 2), whereas the effects on RI were only noticeable when the muscles were well stretched (dorsiflexed angle; Figure 4). This is important because, firstly, it is suggestive that pre- and post-synaptic inputs influencing the change in RI are at least partly divergent to those that normally result in decreases in the H reflex. However, it is also important because decreases
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in resting H-reflex responses measured in soleus at a neutral ankle angle after chronic stretch training have been suggested to indicate that a decreased motor neuron excitability or increased pre-synaptic Ia inhibition could be partly responsible for an improved ankle joint range of motion (Guissard and Duchateau 2004). In that study, the decrease in resting H-reflex response occurred simultaneously with a decrease in passive stiffness of the muscle-tendon unit (MTU), suggesting that either a change in the mechanical properties of the MTU or decrease in reflex stiffness could explain the improved range of motion. Our data also reveal a decrease in the (near) resting H-reflex response after chronic stretch training, but only when the ankle was in a plantarflexed or near-neutral (-3.3 ± 2.9°) position. While it cannot be discounted that reductions in reflex muscle activity early in the change in ROM might have an influence on ankle range of motion, the lack of change H_max:M_max magnitude in the dorsiflexed position indicates that this mechanism is unlikely to be a dominant factor leading to an increase range of motion.

In conclusion, we have found that three weeks of twice-daily static plantarflexor stretching resulted in a significant increase in RI measured in soleus and gastrocnemius during voluntary, tonic dorsiflexion contractions. While it is not possible to pinpoint the precise mechanisms underpinning the change, it is possible that changes in cortico- and rubrospinal drive or altered Ib excitation, or changes in the excitability of the Ia reciprocal interneuron, were influential. Comparison of the changes in H_max:M_max measured during the dorsiflexion contractions to those measured during low-level plantar flexion contractions revealed a clear and separate effect of stretch training on RI, although the augmented RI occurred only with the ankle in dorsiflexion. This is in opposition to our finding that decreases in Ia-mediated motor neuron excitability and/or presynaptic inhibition, measured as a reduction in H_max:M_max magnitude during a low-level plantarflexion contraction, were seen only at plantarflexed or
near-neutral angles. The result is clearly indicative of these adaptations being, at least partly, of separate origin. From a practical perspective, the present results are important because they verify the potential for stretch training to influence RI during locomotor and other complex tasks.

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FIGURE CAPTIONS

Figure 1. H- and M-wave recruitment curves for soleus (top) and gastrocnemius (bottom) for one subject. An example of a single stimulation eliciting an M- and H-wave in soleus is also shown (inset). Stim. – Nerve stimulation trigger trace.

Figure 2. Change (±SD) in H: $H_{\text{max}}:M_{\text{max}}$ (%) measured during 2% MVC plantar flexion contractions after the 3-week intervention at each ankle joint angle. There was a clear decrease at the plantarflexed angle in both soleus and gastrocnemius, but no change at the dorsiflexed angle. *denotes statistical significance.

Figure 3. Change (±SD) in $H_{\text{max}}:M_{\text{max}}$ (%) measured during 20% MVC voluntary dorsiflexion contractions after the 3-week intervention at each joint angle. There was a clear decrease at the dorsiflexed angle in both soleus and gastrocnemius, but only a moderate change in soleus at the plantarflexed angle.

Figure 4. The difference the % change in $H_{\text{max}}:M_{\text{max}}$ measured during a 20% MVC dorsiflexion and that measured during a 2% MVC plantar flexion (±SD). A significant change (decrease) in the difference score was found only at the dorsiflexed angle, indicating that increases in RI were only substantive at this angle.
Change H:M (%) Change H:M (%)

Soleus

Control  Stretch

Gastrocnemius

plantarflexed  neutral  dorsiflexed

Ankle Angle
Control

Difference in H:M change (%)

Soleus

Control  Stretch

-10
-20
-30
-40
-50

Gastrocnemius

Control  Stretch

-10
-20
-30
-40
-50

Ankle Angle

plantarflexed  neutral  dorsiflexed

*