Plantarflexor stretch training increases reciprocal inhibition measured during voluntary dorsiflexion

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Agonist-mediated reciprocal inhibition (RI) 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 RI is an important research goal. We examined the efficacy of using chronic passive muscle stretching to augment RI. The influence of 3 wk of plantarflexor stretching (4 × 30 s, two times/day) on RI of soleus and gastrocnemius initiated by tonic, voluntary dorsiflexion contractions [20% of maximum voluntary contraction (MVC)] was examined in 11 healthy men who performed stretch training and in nine nontraining controls. Hoffmann’s reflexes (H-reflexes) were elicited by tibial nerve stimulation during both weak isometric (2% MVC) plantarflexions and dorsiflexion contractions at 20% MVC. Changes were examined at three joint angles, normalized to each subject’s range of motion (ROM; plantarflexed = 10 ± 0°, neutral = −3.3 ± 2.9°, dorsiflexed = −16.5 ± 5.6°). No changes were detected in controls. A 20% increase in ROM in the stretch subjects was associated with a specific change in maximum H-reflex (Hmax): maximum evoked potential (Mmax), measured during 2% dorsiflexion at the plantarflexed and neutral angles in soleus and at the plantarflexed angle in gastrocnemius (P < 0.05–0.01). By contrast, decreases in HmaxMmax during 20% dorsiflexion contract were also seen at each angle in soleus and at the dorsiflexed angle in gastrocnemius. However, a greater decrease in HmaxMmax measured during voluntary dorsiflexion rather than during plantarflexion, 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 RI from tibialis anterior onto soleus-gastrocnemius in young, healthy individuals at dorsiflexed, but not plantarflexed, joint angles.

static stretch; electrical stimulation; tibial nerve; soleus; skeletal muscle

COORDINATED INTERACTIONS BETWEEN THE triceps surae and pretilibial 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 (TA), while activity of its antagonists is reduced. Thus failure to maintain reciprocal inhibitory inputs derived from the TA 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’s reflex (H-reflex) in a distal agonist muscle, while 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 motoneuron pool (Guissard et al. 1988, 2001). Furthermore, longer-term (4 wk) 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 wk) 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. To isolate the effects of RI mechanisms from the direct effects of stretching on Ia synaptic transmission and α-motoneuron pool excitability, these responses were com-
pared with H-reflexes obtained during low-level (2% of peak moment) plantarflexion 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), RF responses were examined at three joint angles ranging from plantarflexion 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 nontraining, control group. Two control subjects did not report for postintervention testing, so the stretch-training and control groups contained 11 and nine subjects, respectively. All volunteers read and signed an informed consent document, the procedures were conducted in accordance with the declaration of Hel- sinki, and the study was approved by Brunel University’s Human Ethics Committee.

Overview

All subjects completed three testing blocks. The first block acted as a familiarization period, where subjects reported on two to three occasions to practice the moment-targeting procedures (described below) 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 postintervention testing periods, each of which required two sessions: 1) Passive ankle range of motion (ROM) testing and 2) measurement of H- and M-wave amplitudes in soleus and gastrocnemius, while performing a 2% maximal voluntary contraction (MVC) plantarflexion and a 20% MVC dorsiflexion. These sessions were performed at the same time of day on separate days and were each preceded by a warmup 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 h preceding testing, and no stimulants or depressants (e.g., caffeine, alcohol) were allowed within 6 h.

Stretch Training

The experimental group performed four, 30-s calf-muscle stretches twice a day (morning and night) for 4 wk (22 days/44 sessions); self-reported compliance was 91.8 ± 3.8% (40.4 ± 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 performed easily by individuals without the need for assistance. All subjects had exercise backgrounds and were able to perform the stretch well.

Passive Ankle ROM (Maximal Stretch Tolerance) Test

The subjects sat in an isokinetic dynamometer (Biodex System 3, Biodex Medical Systems, Shirley, NY) 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, while allowing comfortable ankle rotation to maximum dorsiflexion. With the subject’s eyes closed and the ankle starting from a position of 20° plantarflexion, the dynamometer was programmed to rotate the ankle into dorsiflexion at 2°/s. The subjects depressed a stop button upon reaching maximum tolerable stretch, which immediately released the footplate and allowed the ankle to rotate back into plantarflexion. Two trials were allowed, with a 1-min rest interval, and the greatest ROM 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 ROM 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 ROMs, and these elicited very different reflex responses), ankle positions were set at 0%, 30%, and 60% of the joint range between 10° plantarflexion and a 90% of peak passive tension was obtained in the passive ROM test, measured at pretraining. Testing was not performed at 90% of ankle ROM, as pilot testing showed that the reliability of obtaining H-reflex and M-wave recruitment curves was lower toward the end ROM. 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, respectively). 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, 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 diameter, fabric gel adhesive electrode, ValuTrode CF3200, Nidd Valley Medical, 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) electrodes placed over soleus and gastrocnemius muscles (see below). Additional stimulants were delivered around the intensity at which the H-reflex amplitude appeared maximal, as shown in Fig. 1, to more accurately determine the maximum H-reflex (Hmax) amplitude.

EMG recording. EMG activity of 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 pseudomonopolar configuration with one electrode placed each on soleus (medial aspect, distal to gastrocnemius) and medial gastrocnemius muscle bellies, while a second electrode was placed ~10 cm proximal to the Achilles tendon insertion onto the calcaneum, just above Kager’s triangle (Hadoush et al. 2009; Pinninger et al. 2001). A reference electrode was placed on the lateral malleolus. The electrodes were connected to a Model 1902 isolated differential preamplifier (Cambridge Electronic Design, Cambridge, UK) with an input impedance of 10,000 MΩ and common mode rejection exceeding 100 dB. The data were analog-to-digitally converted at a sampling rate of 2,000 Hz, acquired using a Power1401 data acquisition interface and stored on a computer running Signal software (Cambridge Electronic
The change in RI was then taken as the difference between the maximum achievable at the specific joint angle and then maximum evoked potential (Mmax) ratio (Hmax:Mmax). Changes in Hmax:Mmax over time between 2% plantarflexion and 20% dorsiflexion conditions for each individual, which was computed using the equation:

\[
\frac{\% \Delta H : M_{\text{prepost}}}{20\% \text{ dorsiflexion} - \frac{(\% \Delta H : M_{\text{prepost}})}{2\% \text{ plantarflexion}}}
\]

where a negative score indicates that the reduction in Hmax:Mmax in the dorsiflexion contractions over the training period was greater than the reduction in Hmax:Mmax in the plantarflexion contractions (i.e., an increase in RI). Changes were examined by ANOVA. This variable allowed the determination of the influence of RI on the change in Hmax:Mmax. 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 ROM, Passive Torque, and Peak Isometric Torque

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

**M-wave Amplitude at Hmax**

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 Hmax (and normalized relative to Mmax), 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).

\[
\begin{align*}
H_{\text{max}}:M_{\text{max}} & \quad \text{Amplitude During 2% MVC Plantarflexion} \\
H_{\text{max}}:M_{\text{max}} & \quad \text{Amplitude During 20% MVC Voluntary Dorsiflexion}
\end{align*}
\]

After stretch training, a decrease in the H-reflex amplitude was responsible for a significant decrease in Hmax:Mmax (see Fig. 2), although this effect was only significant when measured at plantarflexed (−16.8 \pm 3.3%; \( P < 0.001 \)) and neutral (−15.2 \pm 5.6%; \( P < 0.001 \)) angles in soleus and the plantarflexed angle in gastrocnemius (−10.6 \pm 5.5%; \( P < 0.05 \)); there were no changes in M-wave amplitude. There was no change in Hmax:Mmax at the dorsiflexed joint angle after training in either muscle, and there was no change in the control group.

\[
H_{\text{max}}:M_{\text{max}} \quad \text{Amplitude During 20% MVC Voluntary Dorsiflexion}
\]

Stretch training resulted in a substantial reduction in Hmax:Mmax (Fig. 3), measured during 20% MVC voluntary dorsi-
flexion 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).

Hmax:Mmax did not change at any ankle angle in the control group.

**DISCUSSION**

Although both increased and decreased RI has 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 (TA) muscle contractions in young, healthy subjects. The major finding of the present study was that Hmax:Mmax measured in both soleus and gastrocnemius during voluntary dorsiflexion, was reduced substantially after the 3-wk stretching intervention, suggesting a rapid reorganization of spinal circuitry occurred with the training that increased RI. The similar responses measured in

![Graph showing change in Hmax:Mmax at different ankle angles.](image)

### Difference in Percent Hmax:Mmax Changes Between Conditions (2% Plantarflexion vs. 20% Dorsiflexion)

Before training, Hmax:Mmax was smaller at all angles during the dorsiflexion contraction compared with 2% plantarflexion in soleus (30.3%, 36.5%, and 28.8% of 2% plantarflexion condition at plantarflexed, neutral, and dorsiflexed joint 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 plantarflexor Hmax:Mmax.

The specific effect of dorsiflexor activation on Hmax:Mmax depression, resulting from the stretch training, was examined by calculating the difference between the percent change in Hmax:Mmax measured during the 20% dorsiflexion contraction and that measured during the 2% plantarflexion contraction (see equation above in METHODS, Data analysis). As shown in Fig. 4,
both soleus and gastrocnemius indicate that, at least when measured in full knee extension, changes in spinal circuitry have a collective influence on the excitability of both motoneuron pools and/or on the magnitude of presynaptic inhibition of Ia afferents. Uniquely, we examined H-reflex changes both during a low-level plantarflexion 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 plantarflexor Ia afferent transmission onto the motoneuron 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 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% plantarflexion conditions would have minimized corticovestibular influences, so signals associated with dorsiflexor activation or emanating from dorsiflexor afferents are the most 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, 2009) control from brainstem-derived descending tracts (Heckman et al. 2008, 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 RI 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 motoneuron excitability.

From a practical perspective, the finding that the increases in RI were only clearly manifested at the dorsiflexed angle (see Fig. 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 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 $H_{\text{max}}:M_{\text{max}}$ responses, measured (at a particular joint angle) during active dorsiflexion and a low level of plantarflexion contraction, were calculated (see Fig. 4).
This allowed us to determine the specific effects of stretch on RI separately from its effect on Ia afferent transmission to the motoneurons. In fact, we found that the effects of stretch on H$_{\text{max}}$M$_{\text{max}}$, measured during low-level plantarflexion, were noticeable only when the plantarflexor muscles were not stretched substantially (i.e., plantarflexed and neutral angles; Fig. 2), whereas the effects on RI were only noticeable when the muscles were well stretched (dorsiflexed angle; Fig. 4). This is important, because first, it is suggestive that pre- and postsynaptic 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 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 motoneuron excitability or increased presynaptic Ia inhibition could be partly responsible for an improved ankle-joint ROM (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 a decrease in reflex stiffness could explain the improved ROM. 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. Although it cannot be discounted that reductions in reflex muscle activity early in the change in ROM might have an influence on ankle ROM, the lack of change in H$_{\text{max}}$M$_{\text{max}}$ magnitude in the dorsiflexed position indicates that this mechanism is unlikely to be a dominant factor leading to an increased ROM.

In conclusion, we have found that 3 wk of twice-daily, static plantarflexor stretching resulted in a significant increase in RI, measured in soleus and gastrocnemius during voluntary, tonic dorsiflexion contractions. Although 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$_{\text{max}}$M$_{\text{max}}$ measured during the dorsiflexion contractions with those measured during low-level plantarflexion 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 motoneuron excitability and/or presynaptic inhibition, measured as a reduction in H$_{\text{max}}$M$_{\text{max}}$ 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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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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