Evidence of altered corticomotor excitability following targeted activation of gluteus maximus training in healthy individuals

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It has been proposed that strengthening and skill training of gluteus maximus (GM) may be beneficial in treating various knee injuries. Given the redundancy of the hip musculature and the small representational area of GM in the primary motor cortex (M1), learning to activate this muscle before prescribing strength exercises and modifying movement strategy would appear to be important. This study aimed to determine whether a short-term activation training program targeting the GM results in neuroplastic changes in M1. Using transcranial magnetic stimulation, motor evoked potentials (MEPs) were obtained in 12 healthy individuals at different stimulation intensities while they performed a double-leg bridge. Participants then completed a home exercise program for ~1 h/day for 6 days that consisted of a single exercise designed to selectively target the GM. Baseline and post-training input–output curves (IOCs) were generated by graphing average MEP amplitudes and cortical silent period durations against corresponding stimulation intensities. Following the GM activation training, the linear slope of both the MEP IOC and cortical silent period IOC increased significantly. Short-term GM activation training resulted in a significant increase in corticomotor excitability as well as changes in inhibitory processes of the GM. We propose that the observed corticomotor plasticity will enable better utilization of the GM in the more advanced stages of a rehabilitation/training program. *NeuroReport* **27:415–421**

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Introduction

Research performed over the last decade suggests that various knee injuries, including anterior cruciate ligament tears, iliotibial band syndrome, and patellofemoral pain, may be the result of poor hip strength and/or control [1, 2]. Reported movement impairments associated with each of these knee injuries include excessive hip adduction and internal rotation [2]. The gluteus maximus (GM) has been proposed as being a key muscle for providing hip stability as it functions as a hip extensor, abductor, and external rotator [2]. Given its multiplanar role in controlling abnormal hip motions that are associated with many knee injuries, strengthening, followed by skill training of this muscle has been advocated [2].

A potential problem with strengthening the GM is that this muscle is difficult to isolate. For example, there are three primary hip extensors: the GM, the hamstrings, and adductor magnus. The latter two muscles also function as hip adductors [3], and if recruited without adequate activation of the GM, may contribute toward the hip motion impairments observed in patients with various knee injuries. Given the redundancy of the hip musculature, an individual is more prone to develop compensatory movements when attempting to activate the GM. As a result, learning to activate the GM before progressing to functional strengthening and skill training components of a rehabilitation program is potentially important.

Using noninvasive techniques, several studies have shown that neuroplastic changes in the primary motor cortex (M1) can occur following skill training of upper extremity muscles in nondisabled adults [4,5]. More specifically, enlargement of the cortical motor representation as well as changes in corticomotor excitability of specific muscles have been well documented. Yet, unlike the target muscles assessed in the above studies, the GM activates to stabilize the trunk during weight bearing and nonskill-based movements [6]. Accordingly, it is of interest to determine whether selective activation training using a form of exercise that specifically targets the GM also leads to measurable neuroplastic changes in the primary motor cortex. Given the importance of the GM for both rehabilitation and injury-prevention purposes, proper activation to ensure targeted strengthening and use of the GM is critical for both rehabilitation and injury-prevention purposes.

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Transcranial magnetic stimulation (TMS) is a non-invasive neuroimaging tool that can be used to evaluate corticomotor excitability of the central nervous system. We have recently developed a feasible and reliable method of assessing corticomotor excitability of the GM during an active-contraction condition [7]. To date, the use of TMS to measure neuroplastic changes in the GM following short-term training has not been examined.

In the current study, measures of neuroplastic changes include the amplitude of the motor evoked potential (MEP) and the cortical silent period (CSP) duration. An increase in MEP amplitude with motor training is an index of synaptic plasticity and brain reorganization [8] and may reflect enhanced capability to recruit the muscle. Stimulation over the target muscle representational area in the primary motor cortex during active contraction of the muscle induces a period of interruption in the ongoing muscular activity, termed CSP. CSP is known to be mediated by corticospinal inhibitory circuits [9,10]. This inhibitory network is activated in tasks with higher complexity and may contribute toward more precise activation of the target muscles [11]. In addition, by assessing MEP amplitude and CSP duration over varying stimulation intensities and generating input–output curves (IOC), we gain knowledge of the efficiency of muscle recruitment. Examination of the slope of the IOC provides a single measure that reflects a change in corticomotor excitability as well as neuronal recruitment efficiency across all stimulation intensities [9].

The aim of the current study was to determine whether selective activation training of the GM results in measurable neuroplastic changes in M1 as determined by TMS. We hypothesized that a short-duration (1 week) training program that focuses on GM activation would result in modulations of both excitatory and inhibitory circuits mediated by different mechanisms to precisely activate this muscle. Increased corticomotor excitability of GM may result in greater potential for strengthening and skill training in the later phases of rehabilitation.

Methods

Participants

Twelve healthy volunteers (seven men, five women) between the ages of 23 and 40 years (mean 27.7) participated in this study. Before testing, all individuals completed a TMS safety questionnaire to establish eligibility. Exclusionary criteria were as follows: history of neurological or psychiatric disorders, seizures, migraines, family history of epilepsy, electrical, magnetic or metal devices implanted in the body, unexplained loss of consciousness, use of medication or alcohol within the last 12 h, or the possibility of pregnancy. All participants were physically active, with no current lower extremity pain or injury. All procedures were explained to each participant and informed consent was obtained as approved by the Institutional Review Board of the University of Southern California (Los Angeles, California, USA).

Study overview

Participants participated in two TMS testing sessions: before and immediately following 1 week of the GM activation training. The first TMS test session (baseline) was performed on day 1, followed by 20 min of instruction and practice performing the GM activation exercise. Participants returned for the second TMS test session (post-training) immediately following completion of the 1-week home training program. A neuronavigation system was used during TMS testing (Brainsight Frameless; Rogue Research Inc., Montreal, Canada) to ensure reproducibility with respect to the positioning of the TMS coil on the skull.

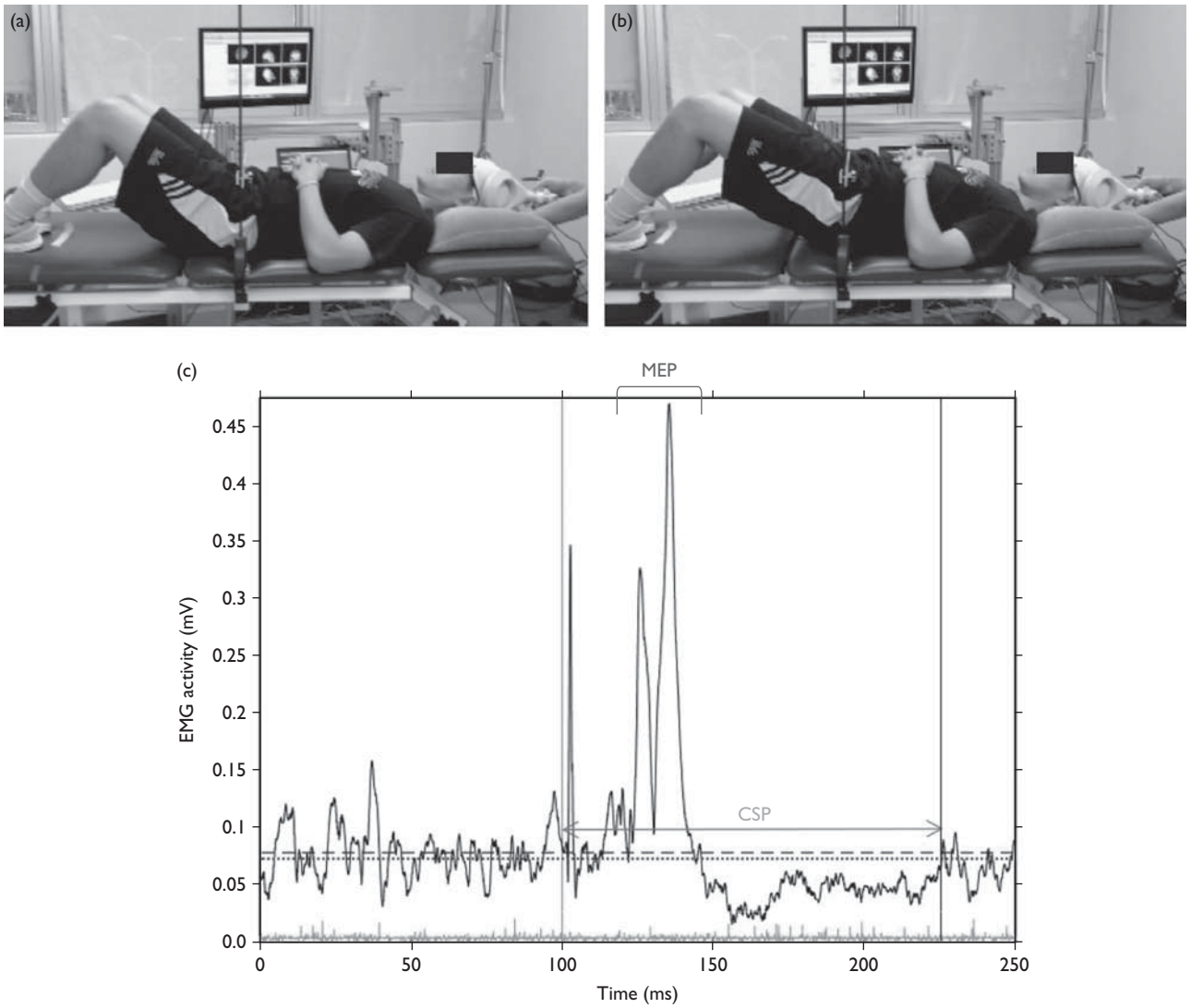
TMS assessment: cortical excitability

A lycra cap marked with a 1 cm grid was placed on the participant's head and surface electromyogram (EMG) electrodes (interelectrode distance, 20 mm, Norotrode 20; Myotronics-Noromed, Inc., Kent, Washington, USA) were placed on the right GM (midpoint between the greater trochanter and the second sacral vertebra). A ground electrode was placed over the second sacral vertebra. During the TMS assessment, participants were positioned supine with the hips and knees flexed to 45° and 90°, respectively. The sampling frequency was 15 003 Hz. The signals were band-pass filtered at 1–1000 Hz, with a gain of 2000 for amplification [customized acquisition tool DataWizard using MATLAB software (The MathWorks, Inc., Natick, Massachusetts, USA)].

Consistent with our previously published reliability study [7], the participants performed a double-leg bridge to generate a sustained contraction of the GM throughout TMS testing (Fig. 1a and b). Given that the resting motor threshold of the GM is usually higher compared with that of upper extremity muscles, we assessed the neurophysiological outcomes under active muscle contraction [7] to decrease the threshold. An anchored bar was placed at 150% of the height between the table and each participant's anterior superior iliac spines. A maximal voluntary isometric contraction (MVIC) was then obtained while participants elevated their pelvis to the designated height. To maintain a consistent level of GM contraction across TMS trials, participants used online visual biofeedback to contract to 25% MVIC ± 5% of the root mean square during the bridge task.

TMS pulses were delivered using a single-pulse magnetic stimulator (Magstim 200; Magstim Company Ltd, Whitland, UK) through a double-cone coil (100 mm) to the GM homunculus of the left primary motor cortex opposite to the trained leg. The hotspot location was identified by systematically moving the coil in 1 cm increments until the locus consistently evoked the

Fig. 1



Setup for TMS testing of the GM and CSP analysis method. (a) Initial position before TMS testing. (b) Pelvis elevated to designated height (active contraction condition) during TMS testing. (c) CSP acquired during 25% of MVIC from one participant. The stimulation intensity was 65% MSO. A single TMS pulse, delivered at 100 ms, was considered the onset of CSP. The dashed line shows the mean prestimulus EMG. The dotted line was the lower limit of variation. CSP offset was determined as the time point at which the EMG signal increased above the lower limit of variation following a silent period. CSP, cortical silent period; EMG, electromyogram; GM, gluteus maximus; MEP, motor evoked potential; MSO, maximum stimulator output; MVIC, maximal voluntary isometric contraction; TMS, transcranial magnetic stimulation.

largest MEP amplitude. The hotspot location of the GM was ~2 cm lateral and 2 cm anterior from the vertex across all participants. The brainsight frameless system was used to mark the hotspot of the GM on a 3D reconstruction brain image for TMS assessments before and after 1 week of exercise. The TMS pulse was delivered at the hotspot following a verbal cue to bridge to the designated height and contract the GM to 25% MVIC.

TMS pulses were delivered at stimulation intensities of 25, 35, 45, 55, and 65% of the maximum stimulator output (MSO). Seven stimulation pulses were delivered at each intensity (a total of 35 pulses). The order of blocks of TMS pulses at each stimulation intensity was randomized for each participant.

TMS assessment: cortical inhibition

The CSP was analyzed using a graphical method [12]. The seven trials of raw EMG acquired in each MSO were filtered, averaged, and rectified to provide a processed EMG dataset. The mean consecutive difference in prestimulus EMG was calculated to estimate the overall variation. The CSP onset was defined as the time of TMS pulse. The offset was defined as return of EMG

activity to the lower limit of variation from the formula: mean prestimulus EMG–(mean consecutive difference × 2.66), which yielded a measure of 99.76% limit of variation (Fig. 1c). As CSP duration is approximately linearly associated with the stimulus intensity [13], the present study only analyzed CSP at 45, 55, and 65% MSO. The lower intensities (25 and 35% MSO) did not elicit CSP consistently across different TMS testing sessions and were thus not included in the data analysis.

GM activation training

Immediately following the initial TMS session, participants were instructed on the performance of a triplanar hip exercise to target the right GM in the quadruped (hips and knees flexed to 90°) position, with a resistance band positioned above the knees (mini exercise bands, 9′ × 2′; Perform Better, Cranston, Rhode Island, USA). Participants were instructed to move the hip into a position of 45° of extension, 45° of abduction, and 30° of external rotation (Fig. 2). This specific exercise was chosen, given the fact that the GM is a hip extensor, abductor, and external rotator [3].

Three resistance bands of increasing resistance levels were provided. All participants started with the lowest level resistance. After verbal confirmation from each participant that the exercise was targeting the GM, participants were instructed to hold this position for 1 min. Participants who could not hold the desired triplanar position for a minute with the lowest resistance band were told to hold the position for as long as possible until muscle fatigue set in or they felt themselves compensating. Participants were stopped and manually cued if they showed excessive rotation of the pelvis or lumbar spine. Participants were instructed to build up to a full-minute hold as the week progressed, with the lowest level resistance band, before progressing to the more advanced bands. A static hold was chosen over a dynamic motion to increase patient concentration while performing the exercise. This static hold required prolonged focus and concentration, thus potentially facilitating an increase in corticomotor excitability of the GM.

Activation training of 20 min occurred on day 1. Participants were instructed to perform 60 min of the exercise at home each day for the rest of the week (6 days). Exercise compliance was monitored using a log. Participants were advised to break the hour into three 20 min sessions throughout the day to avoid muscle fatigue.

Fig. 2

Unilateral triplanar exercise performed in quadruped that targets the GM. Hip positioned in 45° extension, 45° abduction, and 30° external rotation. GM, gluteus maximus.

Data analysis

Peak-to-peak MEP amplitude was calculated to represent cortical excitability in each TMS testing session and then the seven peak-to-peak MEP amplitudes were averaged for each stimulation intensity. An IOC for each TMS testing session was obtained by plotting the average MEP amplitude against its corresponding percentage of MSO. Linear regression was performed to estimate the slope of the IOC for each participant, which quantified the neuroplastic changes after exercise [14].

Statistical analysis

Differences in the slopes of the IOC for MEP and CSP before and after activation training were evaluated using a paired *t*-test. Within-group differences in baseline and post-training mean CSP were compared for each intensity using a paired *t*-test as well. Maximum values of MEP and CSP acquired at 65% MSO were also compared before and after training. Statistical analyses were carried out using SPSS, version 20 (SPSS Inc., Chicago, Illinois, USA) with a significance level of P less than 0.05.

Results

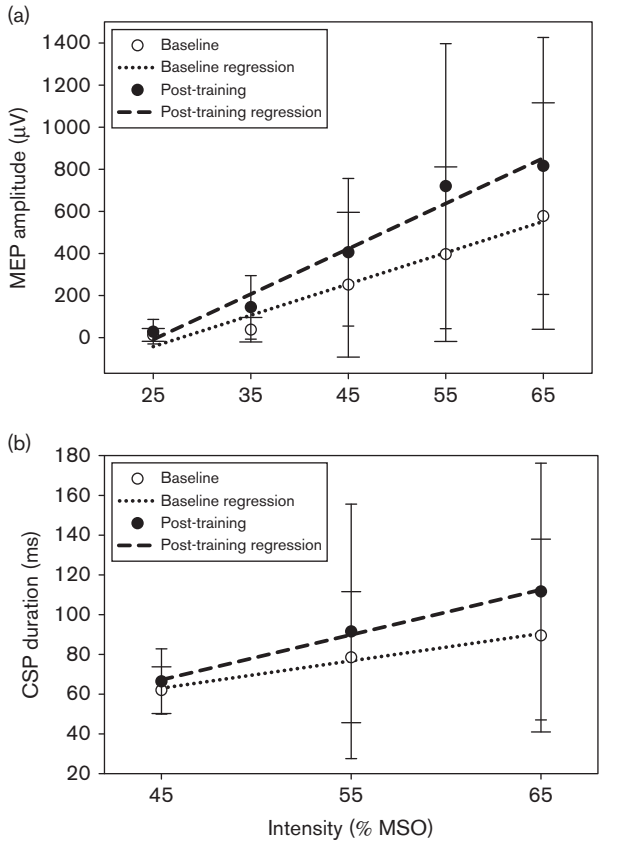
The average values of MEP and CSP at maximum MSO (65%) as well as the slopes of the IOC for each variable calculated from linear regression (baseline and post-training) are presented in Table 1. Maximum MEP and CSP were measured to determine changes in corticomotor excitability and inhibition, respectively. The slopes of the IOC for each measure provided a read-out of changes in activation training-induced efficiency of the

Table 1 Values for outcome measures (mean ± SD)

	Baseline	Post-training	*P* values of paired *t*-test
MEP (μV)			
65% MSO	577.54 ± 538.05	815.66 ± 610.15	0.04
MEP IOC slope	15.46 ± 12.41	20.89 ± 13.47	< 0.01
CSP (ms)			
65% MSO	89.48 ± 48.48	111.64 ± 64.57	0.01
CSP IOC slope	13.74 ± 20.61	22.64 ± 30.81	0.04

CSP, cortical silent period; IOC, input–output curve; MEP, motor evoked potential; MSO, maximum stimulator output.

Fig. 3



Average baseline and post-training IOCs of MEP (μV) and CSP (ms) from all participants. Data were presented in mean ± SD (baseline: white circle; post-training: black circle). Linear slopes of baseline and post-training IOC are represented by dotted and dashed lines, respectively. (a) The slope of MEP IOC improved from 15.46 to 20.89 after completing the activation training ($P < 0.01$). (b) The slope of CSP IOC improved from 13.74 to 22.64 after completing the activation training ($P = 0.04$). CSP, cortical silent period; IOC, input–output curve; MEP, motor evoked potential; MSO, maximum stimulator output.

corticomotor system. There was a statistically significant increase in the linear slope for MEP amplitude of the post-training IOC compared with the linear slope of the IOC at baseline (15.46 ± 12.41 vs. 20.89 ± 13.47, $P < 0.01$) (Fig. 3a). In addition, the maximum MEP amplitude at 65% MSO was statistically different at baseline compared with postactivation training (577.54 ± 538.05 vs. 815.66 ± 610.15 μV, $P = 0.04$). The slope of the CSP IOC was significantly greater following activation training of the GM (13.74 ± 20.61 vs. 22.64 ± 30.81, $P = 0.04$) (Fig. 3b). Furthermore, compared with the pretraining value, CSP acquired at 65% MSO was statistically longer after training (89.48 ± 48.48 vs. 111.64 ± 64.57 ms, $P = 0.01$).

Discussion

Consistent with our hypothesis, the results of our study show that a total of ~6 h of GM activation training resulted in significant modifications in corticomotor excitability. This was indicated by significant increases in the linear slope of both the MEP and CSP IOC after training. The slope of the IOC is considered to provide a measure of efficiency of the system being investigated [15]. The observed increase in the IOC slope after activation training indicates that the GM system (primary motor cortex to the GM through the corticospinal tract) could produce a more robust response at each stimulation intensity.

Interestingly, greater excitability as shown by the slope change was observed in a task that did not isolate the GM (i.e. bridging). This suggests that the neuroplastic changes following specific GM activation training may be generalized to movements involving other hip muscles (i.e. hamstrings). Although an increase in system efficiency was most likely because of changes in synaptic strength, it is not possible to determine the mechanism of this increase from the current study. It could potentially have been driven by a change in neurotransmitter release at the synapse, enhanced strength of inhibitor neurotransmission (gamma aminobutyric acid) or decline in excitatory neurotransmission (glutamate), changes in receptor density, or other potential causes [15,16].

Although the observed changes in cortical excitability following the GM activation training are clearly distinguished from the numerous studies that have shown similar changes with practice of skilled finger and hand manipulation [4,5,17–19], comparable studies have investigated muscles with less skilled and/or more stabilizing function [20–23]. For example, studies have shown that following strengthening programs, cortical adaptations with increased MEP amplitude and enhanced recruitment efficiency of tibialis anterior [20], soleus [20], and quadriceps [21] have been observed. However, the unique aspect of the current work is that we report altered corticomotor processing following a simple short-term activation training in a muscle with a primary stabilizing, nonskill-based function. Unlike a strength-training paradigm, the current study suggests that short-term activation training not only led to improved recruitment efficiency but also enhanced inhibitory processes to refine selective muscle activation.

CSP is a neurophysiological hallmark of cortical inhibition and in the motor cortex, it has been shown that more complicated movements, such as bimanual asymmetrical movements (simultaneous movement of the arms to different target amplitudes for example), lead to increased CSP duration. The inhibitory mechanism modulates M1 excitability to achieve the goal of precisely contracting certain muscles [11]. In the present study, the specificity of the GM activation may require the same inhibitory mechanism to accurately contract the target muscle. Therefore, prolonged CSP that was observed following specific GM activation practice may benefit

future strengthening or movement re-education. The current results suggest that during this prestrengthening period, an enhanced inhibitory mechanism may serve to modulate cortical facilitation and lead to more precise control of the muscle.

To the best of our knowledge, the current study is the first to report changes in corticomotor excitability of the GM after a short-duration activation training program. As noted above, an increase in corticomotor excitability of the GM implies better efficiency of the GM corticospinal motor system, which, in theory, would allow for better recruitment of the GM during more advanced, skill-based hip strengthening exercises. The increase in CSP duration may reflect a refined inhibitory mechanism to ensure muscle recruitment specificity. Thus, this GM activation exercise may be considered a method to prime the brain for subsequent GM strengthening by enabling an individual to target GM activation more precisely. As recruiting the GM agonists such as the hamstrings and/or adductor magnus may have an undesired influence on lower limb kinematics as both muscles act as hip adductors, adequate GM muscle activation would appear to be critical for injury prevention. Further study is needed to determine whether the observed changes in corticomotor excitability of the GM results in greater muscle activation during hip-strengthening exercises. Moreover, such plastic change in M1 can be considered an initial central adaptation before a peripheral adaption such as increased muscle strength or muscle hypertrophy. A long-term follow-up to investigate the persistence of this plastic change with a self-driven home program is warranted.

A limitation of the current study is that we only evaluated healthy, active young adults. It would be important to confirm our findings in patients with anterior cruciate ligament tears, iliotibial band syndrome, and patellofemoral pain as these conditions have been reported to be associated with hip muscle weakness and/or control. A second limitation of our study is that our participants performed the GM exercise at home without supervision. While exercise compliance was documented, we cannot be 100% certain that the exercise was performed correctly for the prescribed amount of time. A third limitation of the current study is that we did not collect strength data or behavioral measures to determine whether neural excitability was related to behavioral improvement. However, a muscle hypertrophy effect would not be expected after only 1 week of exercise. Future studies will utilize a control group that does not participate in the exercise intervention to confirm that altered cortical excitability is in fact because of the activation training. Yet, we do not expect to observe changes without training on the basis of previous studies that changes were only observed with skill training [24,25]. Moreover, comparison groups will also be added to assess training specificity, ensuring that cortical excitability changes are because of adequate activation of GM and not synergistic muscles such as hamstrings, adductor magnus, or erector spinae.

Conclusion

Our findings show that short-term activation training increased corticomotor excitability of the GM, with prolonged CSP. The increase in corticomotor excitability as evidenced by increased MEP amplitude and active inhibitory processes (as evidenced by an increase in CSP duration) reflects a more efficient GM corticospinal motor system with ability to modulate muscle activation specificity. We propose that the changes in corticomotor excitability following activation training would make the GM more available in advanced stages of training programs such as specific strengthening and skill training.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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