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People with Achilles tendinopathy and a previous calf muscle strain injury have differences in neural motor drive in comparison to uninjured controls: a cross-sectional study

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Declarations

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Consent for publication: Not applicable.

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Review and Editing. Ann-Maree Vallence: Writing – Review and Editing. Casey Whife: Investigation, Data Curation, Writing – Review and Editing. Myles Murphy: Conceptualization, Methodology, Investigation, Validation, Resources, Data Curation, Writing – Original Draft, Supervision, Project Administration, Funding Acquisition.

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Patient and Public Involvement: Our research methodology was informed by people with a lived experience of muscle strain injury and tendinopathy.

Abstract

Objective: Impairments in neural motor drive may delay recovery and increase recurrence of musculoskeletal pain and injury. Our study aimed to explore whether measures of neural motor drive (i.e., voluntary activation, corticospinal excitability, intracortical inhibition or intracortical facilitation) differ between individuals with current Achilles tendinopathy and a recovered calf muscle strain compared to uninjured controls.

Methods: A cross-sectional study including participants currently diagnosed with mid-portion Achilles tendinopathy, or a previous calf muscle strain injury (within the past 10 months and have returned to sport), and uninjured controls. Ankle plantar flexor voluntary activation was assessed using twitch interpolation via the tibial nerve and ankle plantar flexor strength was assessed with maximal voluntary contraction (MVC). Single- and paired-pulse transcranial magnetic stimulation assessed active motor threshold (AMT), short-interval intracortical inhibition (SICI), long interval intracortical facilitation (LICF) and intracortical facilitation (ICF) to quantify corticospinal and intracortical excitability. Generalised estimating equations assessed outcomes for cases versus controls ($p<0.05$).

Results: 54 participants were included; 34 cases (16 Achilles tendinopathy and 18 previous calf muscle strain injury) and 20 uninjured controls. Cases had lower voluntary activation (ß=-4.89, 95%CI=-8.16 to -1.62, p=0.003), lower AMT (ß=-7.26, 95%CI=-12.88 to -1.64, p=0.011), higher LICF (ß=0.29, 95%CI=0.04 to 0.55, p=0.026), and higher ICF (ß=0.28, 95%CI=0.07 to 0.48,

p=0.009) than controls. There were no differences detected between cases and controls for MVC or SICI.

Conclusion: Individuals with Achilles tendinopathy or a previous calf muscle strain have altered neural motor drive when compared to uninjured controls, suggesting changes in the motor cortex are associated with pain, and injury. Whether these alterations in neural drive are associated with recovery or recurrence should be investigated.

Key words

Voluntary activation, transcranial magnetic stimulation, intracortical inhibition, intracortical facilitation, triceps surae, plantar flexor

Key points

1. Individuals with Achilles tendinopathy and previous calf muscle strain injury have ~5% lower voluntary activation when compared to uninjured controls
2. Greater corticospinal excitability and intracortical facilitation are associated with Achilles tendinopathy and previous calf muscle strain injury.
3. Further research is required to investigate the potential mechanisms for neurophysiological changes in these injury populations and how it may impact rehabilitation and recurrence.

Introduction

Achilles tendinopathy and calf muscle strain injury are prevalent in running-related sports, irrespective of competition level (1). These conditions are not only associated with substantial time loss from sporting activity, but also with a high recurrence rate (2,3). Further, Achilles tendinopathy has a significant impact on quality of life, work productivity and has been associated with large financial burden (4).

Muscle strength is routinely evaluated to assess potential impairments in tendinopathy, and inform rehabilitation progression and recurrence risk following muscle strain injuries (5).

Although plantar flexor strength deficits have been reported in Achilles tendinopathy, findings are not consistent across all strength measures (e.g., maximal voluntary contraction [MVC], single leg heel raise test) and comparison groups (6,7). Further, there is limited evidence demonstrating persistent strength deficits following calf muscle strain injury. MVC assessments fail to quantify key neurophysiological mechanisms that underpin the ability to generate maximal force. For example, while MVC can quantify how much force is being produced, it does not provide information about how hard the motor system is working to generate that force.

One condition that can impact the ability to produce following joint injury is arthrogenic muscle inhibition. Arthrogenic muscle inhibition represents the inability of the motor nervous system to voluntarily activate muscle and recognised as a major contributor to poor outcomes and functional limitations following significant knee joint injury such as osteoarthritis (13) and anterior ligament reconstruction (ACLR) (11,12). This deficit may limit the benefit of exercise rehabilitation (8,9), and is often associated with poorer strength (14,15). However, whether muscle inhibition extends to non-joint related injury is largely unknown. Muscle inhibition is typically measured using voluntary activation. Voluntary activation as been assessed once in muscle injury, with impaired voluntary activation demonstrated following hamstring muscle strain injury (10). No studies have assessed voluntary activation following calf muscle strain injury, representing an important gap in the literature as neurophysiological deficits may help explain persistent symptoms/deficits following return to sport and the high recurrence rate attributed to this injury (20).

Two cross-sectional studies investigated voluntary activation in relatively small samples of participants with Achilles tendinopathy, with one study including 23 participants and 22 matched controls (16), and the other including 14 participants and 14 matched controls (6). Neither study identified a significant difference in voluntary activation between the injured and non-injured groups. Both studies assessed plantar flexor voluntary activation using an isokinetic dynamometer with the knee in full extension. While this is common practice and considered the

gold standard for plantar flexor MVC measurement, testing position and equipment likely influence the estimation of voluntary activation (17–19). Therefore, further investigation in Achilles tendinopathy using alternative testing methods to provide a more comprehensive assessment of calf muscle voluntary activation is warranted.

Differences in other markers of neural motor drive have been reported. Neurophysiological variables including corticospinal excitability (i.e., active motor threshold [AMT], motor evoked potential [MEP]) and intracortical excitability (i.e., short-interval intracortical inhibition [SICI], intracortical facilitation [ICF], long-interval intracortical facilitation [LICF]), reflect the excitability and inhibitory/facilitatory properties of cortical pathways involved in voluntary muscle activation, and alterations have been identified in other musculoskeletal injured populations, such as patella tendinopathy (21), ACLR (22), and hamstring muscle strain injury (23). A single small study has reported greater SICI in runners with Achilles tendinopathy (n=11) when compared to controls (n=13) (24). However, further research is needed to confirm this finding and investigate potential alterations in other aspects of cortical function. To date, no studies have investigated these outcomes following calf muscle strain injury.

An understanding of differences in these cortical pathways may help to explain the poor outcomes from these injuries, such as prolonged symptom duration, ongoing functional impairments (i.e., reduced strength) and injury recurrence (25,26). While Achilles tendinopathy and calf muscle strain injury share a common muscle-tendon unit (the triceps surae) and are closely interconnected biomechanically and functionally, the different injury mechanisms (i.e., chronic vs acute) and level of symptoms (i.e., current vs recovered) may led to different adaptions in the motor nervous system. Therefore, the aim of our study was to explore whether voluntary activation, corticospinal excitability (AMT, MEP), short-interval intracortical inhibition (SICI), intracortical facilitation (ICF) or long-interval intracortical facilitation (LICF) differ between individuals with Achilles tendinopathy or previous calf muscle strain injury and uninjured controls.

Methods

Study design

Cross-sectional study performed between June 2025 and June 2026.

Ethical considerations

Our research received ethical approval by the Edith Cowan University Human Research Ethics Committee (2024-06061-MURPHY) and reciprocal approval was provided by The University of Notre Dame Australia (2026-080). The research was performed in accordance with the Declaration of Helsinki. All participants provided informed electronic consent via online survey.

Reporting guidelines

Our study was informed by, and reported in accordance with, the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement (27).

Participants

Participants aged 18 to 60 years old were recruited via clinical networks, posters, social media and word of mouth. Participants were involved in a running-based sport and/or activity (e.g., recreational running, basketball, triathlon) and either i) have active mid-portion Achilles tendinopathy, ii) have had a previous calf muscle strain injury or iii) uninjured controls. Achilles tendinopathy was classified as having self-reported load-induced pain located in the mid-portion of the Achilles tendon with morning stiffness the day after activity for over 3 months, and evidence of tendon structural change on magnetic resonance imaging (28). A calf muscle strain injury was classified as having a self-reported sudden or progressive onset of non-traumatic posterior calf pain during (or after) sport/activity which was severe enough to have required intervention by a health professional and caused time-loss from sport and/or activity within the past 10 months. Full self-reported function and no other cause of calf muscle pain identified on magnetic resonance imaging served as additional inclusion criteria (5,29). Uninjured control participants had no previous history of Achilles tendinopathy or calf muscle strain injury. We excluded people with a prior history of lower limb injury, pathology, and/or surgery on the injured

limb which resulted in significant time-loss from physical activity (e.g. knee ligament rupture) and people with insertional Achilles tendinopathy. We also excluded people with any neurological condition limiting lower limb muscular strength, or if they had contraindications for transcranial magnetic stimulation (TMS) and lower-limb nerve stimulation (Appendix A) (30,31).

Setting

Participant-reported survey data were collected online via Qualtrics (Qualtrics, Provo, UT) within 48 hours prior to the testing session. All physical assessments were performed in a dedicated university neurophysiology laboratory and lasted approximately 90 minutes.

Variables

Demographic data

The following demographic data were self-reported: age (years), sex (male; female; intersex), ethnicity, height (cm); body mass (kg); time since injury onset (months); current medications; previous injury history; physical activity level (The World Health Organisation Global Physical Activity Questionnaire (32)); education level, employment status and house-hold income.

Functional disability

The TENDINopathy Severity Assessment – Achilles (TENDINS-A) was used to quantify Achilles tendinopathy and calf muscle strain injury-related functional disability (33). The TENDINS-A consists of 10 questions that address the subdomains of disability, pain, symptoms and function, to produce a score out of 100, whereby a higher score indicates higher disability. The TENDINS-A has sufficient structural validity, reliability, and measurement invariance (34,35), and is the preferred tool for measuring tendon related disability (36). In the absence of a validated calf muscle strain injury patient reported outcome measure, these participants also completed the TENDINS-A.

Maximal plantar flexion strength

Participants were seated on an adjustable wooden box with hips flexed to 90 degrees and the thigh parallel to the floor. The participant's bare foot was placed on a force plate (PASCO PS-

206 2141, PASCO scientific, Roseville, US) secured within a seated plantar flexion testing frame. The
207 centre of the first and second metatarsophalangeal joints were positioned over the centre of the
208 force plate. The ankle joint was placed into the participant's maximal dorsiflexion and secured
209 with a strap from the testing frame over the knee/distal thigh to limit limb movement (Appendix
210 B). The strap was tensioned maximally to minimise ankle movement from the testing position.
211 Tension was periodically checked throughout the session to ensure it remained secure.
212 Three warm up trials at an estimate of 25%, 50% and 75% perceived maximal effort were
213 performed. Further warm up trials were offered to the participant if they subjectively reported not
214 feeling "warmed-up" enough. Participants then performed three plantar flexion maximal
215 voluntary contraction's (MVCs) lasting 3-seconds per trial, with a minimum of 60-seconds rest.
216 Participants were cued to "push directly through the ball of their foot as hard as you can". Force
217 (N) was recorded using a force plate sampling at 2kHz. Data were collected and recorded by
218 Capstone (PASCO scientific, Roseville, CA, USA). The maximal value of three trials was recorded
219 in Newtons (N). We have previously established excellent reliability (interclass coefficient = 0.91,
220 95% confidence interval = 0.86 to 0.94) and validity (r=0.72, 95% confidence interval = 0.52 to
221 0.84) with this plantar flexion MVC protocol (17,37).
222 *Ankle plantar flexor and dorsiflexor muscle activity*
223 Gastrocnemius (both medial and lateral heads) and soleus muscle activity was recorded via
224 electromyography (EMG) during contraction and in response to the electrical stimulation (M-
225 wave). The muscle activity and M-wave responses of the tibialis anterior were recorded to
226 monitor antagonist muscles activation to refine the set-up protocol. Surface electrode (Kendall
227 ECG electrodes) placement was informed by the surface electromyography for the non-invasive
228 assessment of muscles (SENIAM) guidelines (38). EMG signals were amplified and band-pass
229 filtered at 20-1000 Hz (BioAmp EMG System, ADInstruments NZ Limited, Dunedin, New Zealand)
230 with a sampling rate of 2kHz. Analogue signals were digitised and stored using LabChart
231 (ADInstruments NZ Limited, Dunedin, New Zealand).

Voluntary activation

Plantar flexion voluntary activation was assessed using the interpolated twitch technique (39). The tibial nerve was stimulated in the popliteal fossa with a single 200-μs square-wave pulse delivered at 400 V, via a constant current stimulator (Digitimer DS7A, Digitimer Ltd, Hertforshire, United Kingdom). Single pulse stimulation was selected as it is more comfortable for the participant and there is no difference in ankle plantar flexor voluntary activation levels when either a single or double pulse is used (40). The placement of the cathode (negative) was determined by the location eliciting the highest level of soleus EMG activity and plantar flexion force, with minimal activation of tibialis anterior. A hand-held probe (ADInstruments, NZ Limited, Dunedin, New Zealand) was used to determine the optimal location of cathode (negative) in the popliteal fossa before an adhesive electrode (Kendall ECG electrodes) was applied to the skin over the optimal location. The anode (positive) was placed over the medial tibial condyle. Single stimuli were increased in a stepwise fashion until no further increases in twitch amplitude and force was detected. Stimulation intensity for voluntary activation was set at 120% of the intensity required to evoke a maximum resting twitch in the plantar flexor muscles.

To perform the interpolated twitch technique, participants were instructed to perform an MVC while the assessor (MEC) provided *strong* verbal encouragement (41). When the force reached the participant's maximum, the electrical stimulus was delivered manually by the assessor. The participant was instructed to relax completely, once the EMG and force trace indicated the muscle was at rest, a second potentiated resting stimulus was delivered. A minimum of five voluntary activation trials were conducted with 60 second rest between each effort. A trial was considered valid if the stimulation during the contraction occurred at their highest MVC for the trial. The valid trial with the highest pre-stimulation MVC was then selected. Voluntary activation (%) was calculated as:

$$Voluntary\ activation\ (\%) = \left(1 - \frac{superimposed\ twitch\ amplitude}{resting\ twitch\ amplitude}\right) x\ 100$$

Our voluntary activation protocol is valid against gold standard technique and has demonstrated good test-retest reliability, with an intraclass coefficient of 0.85 (95% confidence interval: 0.59 to 0.94), standard error of the mean of 2.56% (95% confidence interval: 2.02 to 3.27) and coefficient of variation of 2.79% (95% confidence interval: 2.15 to 3.58) (17).

Transcranial magnetic stimulation

TMS was used to assess intracortical and corticospinal excitability. Participants were seated in an isokinetic dynamometer (Biodex System 4, Biodex Medical Systems, Shirley, New York) with their knee fully extended and ankle positioned in neutral (i.e., 0 degrees of plantar flexion). A double cone magnetic coil (11cm outside diameter, Magstim, Whitland, UK) attached to the magnetic stimulator (Bistim2, Magstim, Whitland, UK), was used to evoke responses from the soleus muscle. The coil was held over the motor cortex contralateral to the limb being tested. The final coil position was the one which evoked the largest MEP from the soleus, this was marked on a tight-fitting cap (UnderAmour, Baltimore, United States) covered by tape to allow accurate testing across the session.

Corticospinal excitability

Single-pulse TMS assessed AMT to estimate corticospinal excitability. AMT was determined as the lowest percentage of maximal stimulator output (%MSO) to elicit 3/6 MEPs >0.5 mV in the soleus muscle while the participant held 30% MVC contraction (force guideline was provided on a clearly visible screen) (31). MEP amplitude was measured following single-pulse trials at 120% of AMT delivered while the participant held 30% MVC (termed MEP120%). Pilot testing informed the selection of 30% MVC during TMS to optimise stimulation intensity for participant comfort.

Intracortical excitability

Paired-pulse TMS was used to assess measures of intracortical excitability: short-interval intracortical inhibition (SICI), long-interval intracortical facilitation (LICF) and intracortical facilitation (ICF). For paired-pulse trials, the conditioning stimulus was set as 80% of AMT and test stimulus as 120% of AMT, with an interstimulus interval of 3 ms for SICI, 100 ms for LICF, and

12 ms for ICF. A total of 16 x MEP120%, 16 x SICI, 16 x LICF and 16 x ICF trials were delivered in eight randomised blocks of eight stimulations with 60s rest between each block (2 blocks per condition). Each stimulus block was delivered while the participant produced 30% plantar flexion MVC set by a guideline on a television screen visible to the participant.

Sample size calculation

Our a priori sample size calculation determined that when using voluntary activation as the primary outcome variable, a total sample size of 54 participants was sufficient to detect between-group differences (case vs control), assuming a two-tailed test, effect size of 0.84 (11,13), $\alpha = 0.05$, power $(1 - \beta) = 0.80$, and a 2:1 allocation ratio (cases:controls), based on G*Power (v3.1.9.7).

Statistical analysis

All statistical analyses were performed in R language and environment for statistical computing (version 4.4.3). All variables were described using count, percentage, mean, standard deviation, median, interquartile range (IQR, 25^{th}–75^{th} quartiles) and range (minimum to maximum), where appropriate. Normality of the data was observed visually and assessed with the Sharpiro-Wilk test. Statistical significance was accepted when $p<0.05$.

Generalised estimating equations were used to assess each of the neurophysiological outcomes (i. voluntary activation, ii. MVC, iii. AMT, iv. MEP120%, v. SICI, vi. LICF, vii. ICF) as this model makes no assumptions of data distribution, accounts for correlated observations and provides robust estimates of average effects across the study population (42). For each outcome, two models were performed. Model 1 compared controls and all cases (calf muscle strain injury and Achilles tendinopathy grouped together) to assess if neurophysiological differences exist in the presence of any calf muscle/tendon injury. Model 2 compared the groups individually (control vs calf muscle strain injury and Achilles tendinopathy sub-groups) to explore diagnosis specific differences. Age (years) was included as a co-variate in all models. MVC was included in the voluntary activation model as voluntary activation is directly influenced by the MVC magnitude.

Sex and body mass (kg) were included within the MVC model due to sex and muscle-mass related differences in maximal strength (43). In the intracortical excitability models (SICI, LICF and ICF), the raw values from each trial, as opposed to the ratio was used with single pulse MEP included within each model. Sensitivity analyses were performed by repeating the primary analysis (model 1) with sex and pre-stimulus EMG root square mean (RMS) amplitude as an additional covariate in the model to assess whether the results changed. The collinearity and normality of residuals were assessed. Co-linearity was deemed acceptable if the variance inflation factor was less than 5, and residuals were assessed visually for normal distribution.

Results

We included a total of 54 participants, of whom 34 were cases (20% female sex, n=7) and 20 were uninjured controls (45% female sex, n=9). The cases comprised 16 people with Achilles tendinopathy (19% female sex, n=3) and 18 with a previous calf muscle strain injury (22% female sex, n=4). Detailed participant descriptives are described in Table 1 and Appendix C.

Voluntary activation and MVC

Voluntary activation and plantar flexor MVC, by group, are presented in Table 1 and Figure 1. Cases had lower voluntary activation (ß=-4.89, 95%CI=-8.16 to -1.62, p=0.003, Table 2) than controls. Both calf muscle strain injury (ß=-5.06, 95%CI=-8.86 to -1.26, p=0.009) and Achilles tendinopathy (ß=-4.74, 95%CI=-8.58 to -0.89, p=0.016) had lower voluntary activation than controls (Table 2). Sensitivity analysis including sex as a covariate did not alter the effect estimate or statistical significance of the outcome (Appendix D). No between group difference was detected for plantar flexion MVC (N) (Appendix E).

Corticospinal excitability

Corticospinal excitability outcomes by group are presented in Table 1.

Active motor threshold

Cases had lower AMT (ß=-7.26, 95%CI=-12.88 to -1.64, p=0.011, Table 3) than controls, indicating greater corticospinal excitability. Both calf muscle strain injury (ß=-6.67, 95%CI=-

13.17 to -0.17, p=0.044) and Achilles tendinopathy sub-groups (ß=-7.84, 95%CI=-13.72 to -1.96, p=0.009) had lower active motor threshold than controls (Table 3, Figure 2). Sensitivity analysis including sex as a covariate did not alter the effect estimate or statistical significance of the outcome (Appendix D).

Motor evoked potential 120%

There was no difference in MEP120% between the cases and controls (ß=0.29, 95%CI=-0.26 to 0.84, p=0.307, Table 3). Sensitivity analyses including sex and pre-stimulus EMG RMS amplitude as covariates altered the effect estimate but did not change the statistical significance of the outcome (Appendix D).

Intracortical excitability

Intracortical excitability outcomes by group are presented in Table 1. Participant-level neurophysiological data is available in the supplementary material.

Short interval intracortical inhibition

There was no difference in SICI between the cases and controls (ß=0.04, 95%CI=-0.45 to 0.37, p=0.842, Table 4). Sensitivity analyses including sex and pre-stimulus EMG RMS amplitude as covariates did alter the effect estimate but did not change the significance of the outcome (Appendix D).

Long intracortical facilitation

Cases had higher LICF (ß=0.29, 95%CI=0.04 to 0.55, p=0.026, Table 5) than the controls. Further, calf muscle strain injury (ß=0.36, 95%CI=0.04 to 0.68, p=0.029) but not Achilles tendinopathy (ß=0.23, 95%CI=-0.06 to 0.52, p=0.122) had higher LICF than controls (Table 5). Sensitivity analyses including sex and pre-stimulus EMG RMS amplitude as covariates altered the effect estimate and statistical significance of the outcome (Appendix D).

Intracortical facilitation

Cases had higher ICF (ß=0.28, 95%CI=0.07 to 0.48, p=0.009, Table 5) than the controls. Further, the Achilles tendinopathy sub-group had higher ICF than controls (ß=0.33, 95%CI=0.04 to 0.63,

p=0.027), while there was no between group difference detected for calf muscle strain injury (ß=0.22, 95%CI=-0.01 to 0.44, p=0.059). Sensitivity analyses including sex and pre-stimulus EMG RMS amplitude as a covariate did not change the effect estimate or statistical significance of the outcome (Appendix D).

Discussion

Our cross-sectional study identified that people with a recent calf muscle strain injury and current Achilles tendinopathy have differences in voluntary activation, corticospinal and intracortical excitability compared to uninjured controls. These are novel findings in Achilles tendinopathy, and the first study to investigate neurophysiological differences in individuals with a previous calf muscle strain injury.

People with Achilles tendinopathy and previous calf muscle strain injury had approximately 5% lower plantar flexion voluntary activation compared to uninjured controls. Our findings suggest that people with these injuries have reduced ability to maximally drive their plantar flexor muscles, despite no difference in maximal isometric muscle strength between the injured and uninjured groups, regardless of body mass. It is interesting to note the voluntary activation deficit occurred in both injury groups, despite Achilles tendinopathy being characterised by tendon structural change and chronic symptoms and calf muscle strain injury as an acute injury which had recovered and was reported as asymptomatic. Despite both groups reporting full function during physical activity and sport, there seems to be a persistent impairment in descending drive to the muscle. Maintaining maximal strength despite impaired voluntary activation may require greater contribution from other neurophysiological drivers, potentially increasing fatigue and susceptibility to recurrent injury. This is of particular concern for individuals with a recovered calf muscle strain injury who have returned to sport, as previous history of calf muscle strain is cited as one of the strongest risk factors for recurrence (44). Our findings support that return to sport may reflect readiness to participate, but not full restoration of neurophysiological function, highlighting the potential need for continued rehabilitation beyond return to play (45). Further, a

recent systematic review identified a disconnect between the recovery of strength and voluntary activation over the 12-month period following ACLR, highlighting that improvements in strength following injury may be achieved from muscle hypertrophy and altered recruitment patterns rather than full recovery in neural drive (46).

While our findings contrast with previous investigations into voluntary activation in Achilles tendinopathy (6,16), they reflect findings in other pathologies such as hamstring muscle strain injury (10), knee and hip osteoarthritis (13), or even following an ACLR (11). The discrepant findings in Achilles tendinopathy may be attributed to differences in testing methodology, participant characteristics and sample size. Specifically, our study assessed voluntary activation with the knee flexed and maximal dorsiflexion using a seated, portable set up, whereas previous studies used an isokinetic dynamometer with the knee fully extended and ankle in a neutral position. While stimulation parameters were consistent between studies, our participants had a shorter mean duration of symptoms and symptom severity was measured with different scales (VISA-A vs TENDINS-A), precluding direct comparison. Of these methodological differences, knee position may particularly relevant as greater knee flexion has been associated with higher voluntary activation (19). Additionally, our method has demonstrated good test-retest reliability (17), whereas reliability was not reported for the methods used in the previous studies. Our findings in calf muscle strain injury are novel, and replicate similar voluntary activation deficits identified following hamstring muscle strain injury (10). Together, these findings suggest that reduced voluntary activation may be a feature of lower-limb musculoskeletal injury, regardless of symptom resolution, extending previous evidence from hamstring muscle strain injury to Achilles tendinopathy and calf muscle strain injury.

Our study demonstrated greater corticospinal excitability (i.e., lower active motor threshold) than uninjured controls; however, no differences were observed for MEP120%. Changes in cortical excitability following other pathologies have been assessed with inconsistent results. For example, greater corticospinal excitability has been identified in patellar tendinopathy using

413 stimulus-response curves (21), whereas no differences in active motor threshold have been
414 identified between individuals with patellar tendinopathy and controls (47). Further, no
415 differences in active motor threshold were identified in hip osteoarthritis (15), while lower
416 excitability (i.e., greater motor threshold) has been observed following ACLR (14,48). These
417 findings suggest that differences in motor cortex excitability may be pathology specific and vary
418 across injury presentations. In our study, the differences observed in active motor threshold, but
419 not MEP120%, may suggest both chronic (Achilles tendinopathy) and sub-acute (recent history
420 of calf muscle strain injury) injuries of the ankle plantar flexors alter the threshold for
421 corticospinal neuron activation, but not the magnitude of corticospinal output at suprathreshold
422 intensities. Increased corticospinal excitability may represent a compensatory neuroplastic
423 adaption to the injury, however, whether this change reflects poor outcomes, maladaptive motor
424 control strategies or increased re-injury risk remains unclear. In individuals with Achilles
425 tendinopathy and a recovered calf muscle strain injury, our findings suggest an adaption towards
426 increased corticospinal excitability, highlighting the potential involvement of central nervous
427 system mechanisms in these conditions.
428 Our study demonstrated differences in intracortical facilitation (i.e., ICF and LICF), but not
429 intracortical inhibition (i.e., SICI), in cases compared to controls. While ICF and LICF ratios
430 remained close to 1.0 in control participants, demonstrating little evidence of facilitation, our
431 models showed that facilitation was greater in the combined injured group relative to controls.
432 ICF has been shown to have excellent test-retest reliability in the rectus femoris muscle (49), but
433 has not been assessed in the ankle plantar flexors. Further, the reliability of LICF has not been
434 investigated. Therefore, our findings of greater intracortical facilitation after a calf muscle strain
435 injury and Achilles tendinopathy should be interpreted with caution. However, the increased
436 facilitation observed alongside lower active motor thresholds provides converging evidence for
437 an overall increase of corticospinal excitability, suggesting both corticospinal and intracortical
438 networks may be altered in these injuries. Our results are reflected in other conditions such as

chronic hip osteoarthritis (greater ICF than controls) (15), and acutely following ACLR (increased short-ICF acutely following surgery) (51). While the mechanisms underlying these changes remain uncertain, greater facilitation may represent a compensatory mechanism aimed at maintaining motor performance in the presence injury related impairments, persistent pain or motor control adaptions, but further research is required.

Our study demonstrated no differences in intracortical inhibition between the cases and control groups. This finding is somewhat unexpected as greater inhibition has been observed previous in runners with Achilles tendinopathy (24) and in people with a history of hamstring muscle strain injury in comparison to controls (23). This discrepancy may reflect differences in sample size, injury chronicity, rehabilitation exposure muscle group involved, or natural variations in individual neurophysiological responses. Additionally, the absence of between-group differences in inhibition may have been influenced by the level of voluntary muscle contraction, as lower levels of SICI have been associated with a greater percentage of MVC maintained during testing, or our study being underpowered to detect differences (50,52). Overall, our findings suggest the neurophysiological response to Achilles tendinopathy and calf muscle strain injury may be characterised by greater facilitatory and excitatory processes rather than altered inhibitory control.

The mechanism underpinning these voluntary activation and motor cortex changes is unknown. A potential explanation is that kinesiophobia or the perceived threat associated with the affected tissue may influence neural drive, either in the presence of ongoing symptoms, such as in Achilles tendinopathy, or persisting beyond symptom resolution following calf muscle strain injury (53). Although these conditions differ in injury mechanism and clinical presentation, examining them together allowed us to examine if alterations in calf muscle neurophysiological function are evident in the presence of pathology first, before investigating injury specific changes. Further, current rehabilitation strategies, such as plantar flexor strength training, may not directly target deficits in voluntary activation or cortical drive, potentially allowing these

neural adaptations to continue. Persistent alterations in neural function following return to activity or sport may contribute to compensatory movement strategies and altered loading of other structures, which have been proposed as potential contributors to reinjury following ACLR (54,55). Future research should aim to further investigate these potential mechanisms for neurophysiological adaption.

Clinical implications

Our findings highlight that neurophysiological impairments persist in current Achilles tendinopathy and following recovered calf muscle strain injury, despite self-reported return to full function. Clinically, this highlights the need for ongoing rehabilitation beyond return to sport clearance, and highlights the potential need to include interventions that support neurological function (25). Rehabilitation prescribed by physiotherapist is typically self-paced, where the use of additional visual or verbal cues in conjunction with performing resistance training is not always emphasised. External pacing, such as metronome-paced strength training, has been shown to improve corticospinal excitability and inhibition when compared to self-paced strength training (56,57). Further, transcranial direct current stimulation, a non-invasive brain stimulation protocol, applied during strength training has shown the ability to modulate motor cortex inhibition and facilitation in anterior cruciate ligament rehabilitation (26). Given the findings of our research that people with Achilles tendinopathy and a recovered calf muscle strain injury have increased corticospinal excitability and intracortical facilitation, future research should investigate the effect of these interventions on outcomes in these populations.

Limitations

Our cross-sectional study was designed to explore differences between groups and thus, not designed to examine causation. Future longitudinal studies are required to determine whether these impairments in voluntary activation, corticospinal and intracortical excitability are present and related to injury risk and/or a direct result of injury. Our study was powered to detect differences between case and controls and analysis of the differences between the individual

injury groups and controls were exploratory. Replicating this research with larger sample sizes may be able to identify condition specific neurophysiological impairments. Our protocol was limited to measuring SICI, ICF and LICF, future research may consider including long intracortical inhibition (LICI) and short intracortical facilitation (SICF). Voluntary activation and motor cortex function were assessed under different testing conditions and using different equipment (portable, seated method versus isokinetic dynamometer), which may have influenced the respective measurements. Finally, differences in sex, age and body mass between the groups may confound results, however, we included these as covariates in our additional models to account for the potential influence on our variables.

Conclusion

Individuals with Achilles tendinopathy and previous calf muscle strain injury demonstrated impaired plantar flexor voluntary activation in comparison to uninjured controls. Additionally, the cases had greater corticospinal excitability and intracortical facilitation, suggesting adaptive changes in the motor cortex are associated with injury.

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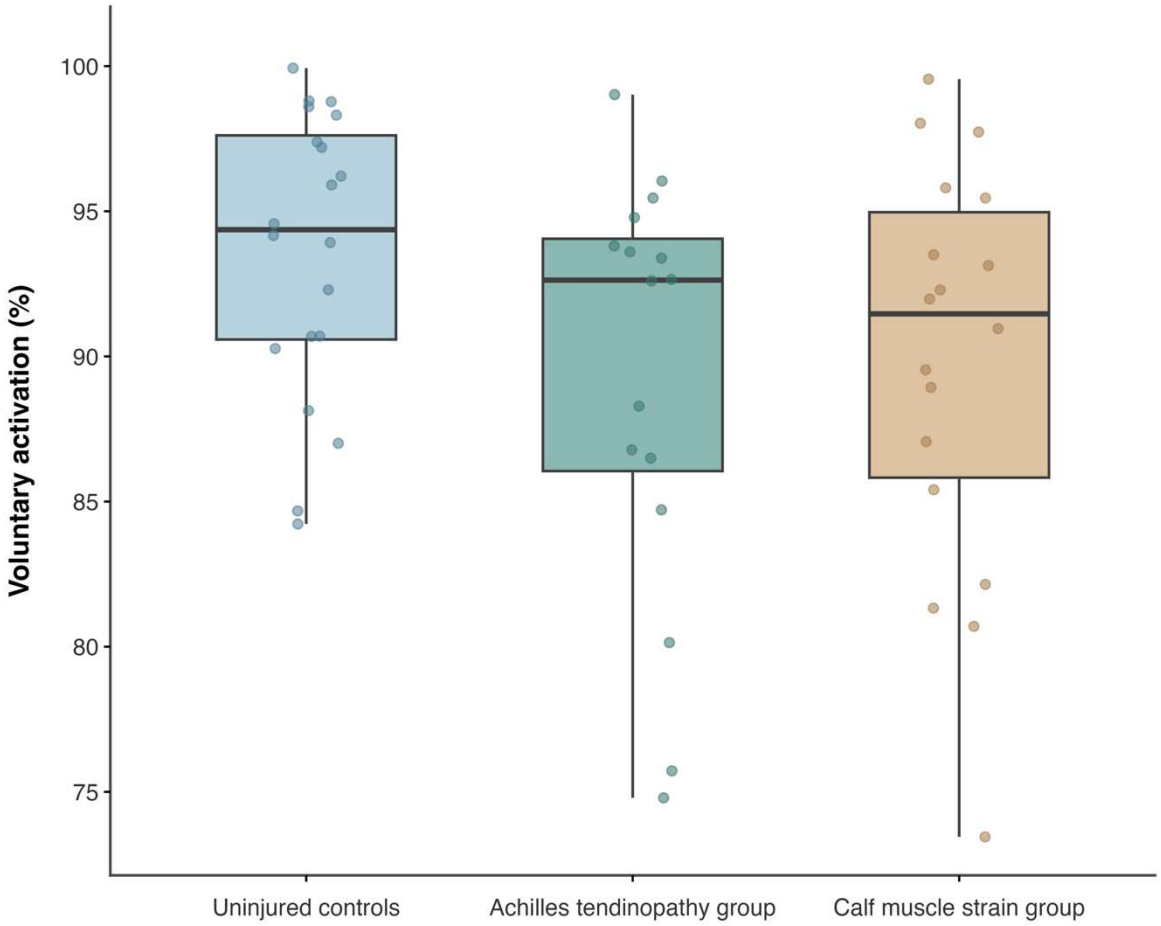
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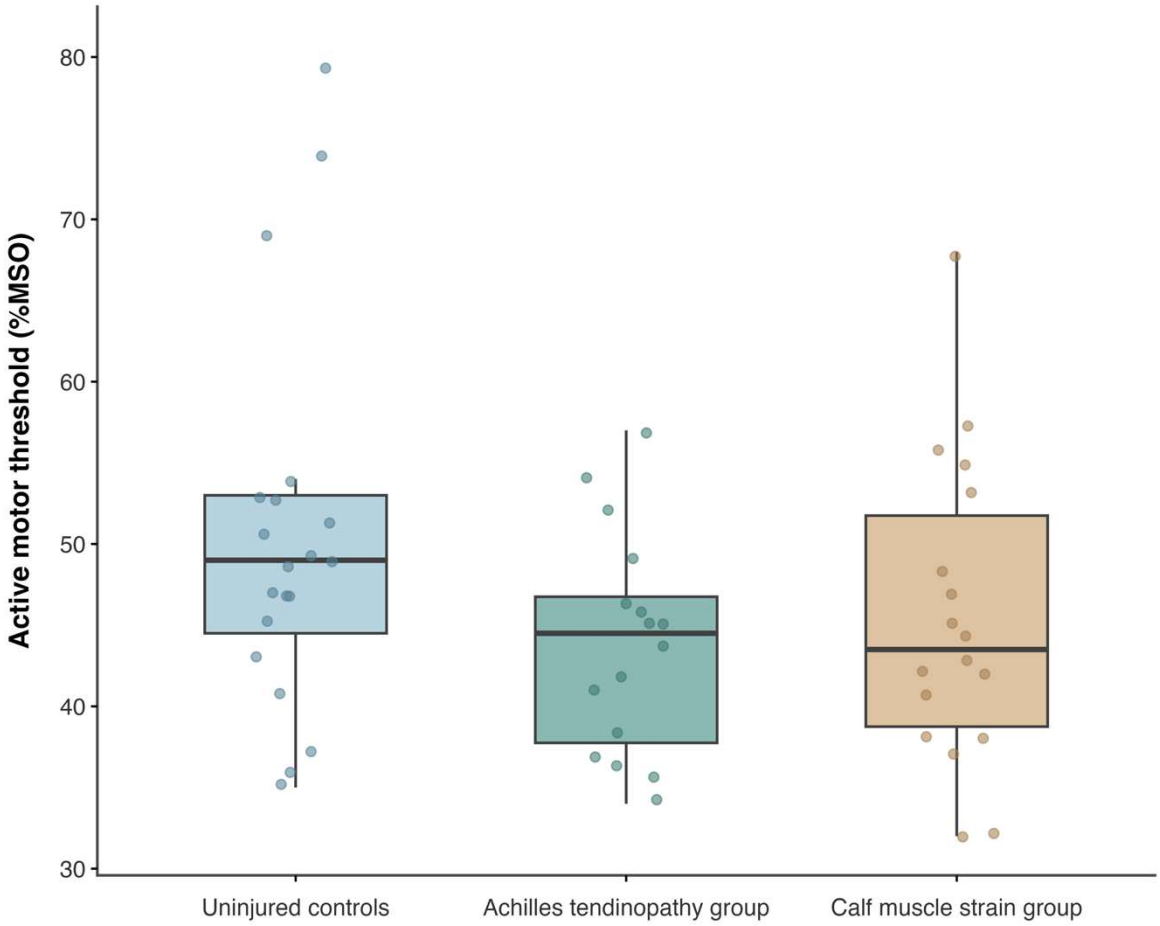
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Figure 1. Voluntary activation (%) by group



Legend: boxes represent the median and interquartile range, with whiskers indicating 1.5 times the interquartile range.

Figure 2. Active motor threshold by group



Legend: boxes represent the median and interquartile range, with whiskers indicating 1.5 times the interquartile range.

Table 1. Participant characteristics by group

	Controls (n=20)			Achilles tendinopathy (n=16)			Calf muscle strain injury (n=18)		
Variable	*Mean (SD)*	*Median [IQR]*	*Range*	*Mean (SD)*	*Median [IQR]*	*Range*	*Mean (SD)*	*Median [IQR]*	*Range*
Demographic information									
Age (years)	28.55 (4.89)	28.5 [26, 30.5]	20–40	35.69 (12.13)	35.5 [26.25, 43.5]	19–57	37.94 (11.89)	34.5 [29.5, 46.75]	21–60
Body mass^ (kg)	73.5 (13.04)	73 [64.5, 80.25]	53–109	77.38 (12.33)	76.5 [69, 84]	58–103	81.83 (13.9)	84 [74.75, 89.5]	57–104
Height^ (cm)	176.48 (9.34)	176 [169.75, 183]	160–196	178.06 (9.28)	179.5 [171.5, 183.75]	161–191	177.44 (12.2)	178.5 [172.5, 184.75]	153–203
TENDINS-A	1.25 (3.77)	0 [0–0]	0–16	45.19 (13.13)	46 [37.75, 53.5]	24–66	18.11 (19.61)	9.5 [1.75, 32]	0–56
Time since injury (months)	-	-	-	13.44 (14.7)	8 [6, 12]	3–60	3.94 (2.77)	4 [2, 5]	1–10
Physical activity (METs/wk)	5054.6 (6488.58)	3600 [2280, 5340]	600–31360	4108.75 (2441.64)	3560 [2100, 5160]	1680–11040	4488.89 (2923.83)	3480 [2390, 5670]	1280–11040
Plantar flexor strength and voluntary activation									
MVC^ (N)	1066.4 (213.62)	1065.73 [932.84, 1226.88]	556.67–1413.93	1048.65 (233.24)	1093.92 [831.99, 1166.97]	675.5–1432	1116.99 (323.98)	1003.48 [874.82, 1410.41]	735.95–1843

Relative MVC (N/kg)	1.49 (0.22)	1.53 [1.35, 1.62]	0.92–1.8	1.39 (0.26)	1.44 [1.18, 1.53]	0.94–1.78	1.40 (0.36)	1.32 [1.18, 1.48]	0.90–2.41
Voluntary activation (%)	93.59 (4.88)	94.36 [90.58, 97.61]	84.23–99.93	89.27 (7.3)	92.63 [86.05, 94.05]	74.8–99.02	89.83 (7.02)	91.47 [85.82, 94.97]	73.45–99.55
Corticospinal excitability									
Active motor threshold	50.45 (11.66)	49 [44.5, 53]	35–79	43.88 (6.8)	44.5 [37.75, 46.75]	34–57	45.44 (9.41)	43.5 [38.75, 51.75]	32–68
MEP120%: MG (mV)	2.23 (1.21)	1.9 [1.52, 2.86]	0.53–5.35	2.7 (1.88)	1.81 [1.51, 3.89]	0.44–7.76	2.3 (1.44)	1.9 [1.15, 3.44]	0.64–5.14
MEP120%: LG (mV)	1.85 (1.18)	1.49 [1.2, 1.96]	0.69–5.3	2.47 (1.82)	2.04 [1.06, 3.62]	0.28–6.87	2.54 (1.84)	2.15 [0.97, 3.91]	0.41–6.16
MEP120%: Soleus (mV)	1.51 (0.73)	1.44 [0.95, 1.64]	0.61–3.45	1.21 (0.48)	1.18 [0.81, 1.5]	0.51–2.21	1.31 (0.48)	1.37 [0.89, 1.67]	0.61–2.16
Intracortical inhibition									
SICI ratio: MG	0.75 (0.18)	0.74 [0.6, 0.82]	0.46–1.09	0.72 (0.19)	0.78 [0.6, 0.86]	0.29–0.98	0.82 (0.33)	0.81 [0.6, 0.91]	0.38–1.88
SICI ratio: LG^	0.77 (0.26)	0.72 [0.64, 0.9]	0.33–1.31	0.71 (0.3)	0.7 [0.48, 0.88]	0.21–1.28	0.74 (0.23)	0.66 [0.6, 0.86]	0.38–1.16

SICI ratio: Soleus	0.84 (0.29)	0.79 [0.69, 0.91]	0.41–1.68	0.84 (0.29)	0.78 [0.65, 0.95]	0.42–1.56	0.86 (0.51)	0.74 [0.59, 0.92]	0.35–2.68
Intracortical facilitation									
ICF ratio: MG	1.05 (0.19)	1.11 [0.93, 1.2]	0.69–1.32	1.23 (0.37)	1.06 [1.01, 1.37]	0.72–2.15	1.25 (0.32)	1.1 [1.02, 1.38]	0.95–2.04
ICF ratio: LG	1.03 (0.19)	1.02 [0.93, 1.14]	0.73–1.44	1.14 (0.25)	1.13 [1, 1.23]	0.52–1.57	1.1 (0.39)	1 [0.86, 1.21]	0.65–2.08
ICF ratio: Soleus	0.94 (0.22)	0.94 [0.8, 1.05]	0.62–1.49	1.11 (0.27)	1.02 [0.95, 1.17]	0.83–1.92	0.99 (0.17)	0.94 [0.88, 1.12]	0.75–1.39
LICF ratio: MG	1 (0.17)	1.02 [0.89, 1.11]	0.64–1.26	1.05 (0.31)	0.99 [0.88, 1.2]	0.59–1.69	1.17 (0.34)	1.04 [0.96, 1.22]	0.71–1.85
LICF ratio: LG	0.96 (0.17)	1 [0.84, 1.06]	0.64–1.26	1.05 (0.21)	1.05 [0.93, 1.18]	0.52–1.4	1.25 (0.56)	1.11 [0.92, 1.28]	0.74–3
LICF ratio: Soleus	0.94 (0.17)	0.98 [0.81, 1.03]	0.65–1.24	1.12 (0.27)	1.03 [0.98, 1.26]	0.65–1.74	1.07 (0.28)	1.01 [0.93, 1.14]	0.65–1.74

Legend: n=number, SD=standard deviation, IQR=interquartile range, kg=kilograms, cm=centimeters, MET=metabolic equivalent of task, MVC=maximal voluntary contraction, N=newtons, MEP=motor evoked potential, MG=medial gastrocnemius, LG=lateral gastrocnemius, SICI=short-interval intracortical inhibition, LICF=long-interval intracortical facilitation, ICF=intracortical facilitation

^Denotes data consistent with a normal distribution as assessed by the Shapiro–Wilk test ($p > 0.05$)

Table 2. Between group differences in voluntary activation

		Estimates (ß)	*95% Confidence Interval*	*p*
Model 1	(Intercept)	75.56	64.78 to 86.34	**<0.001**
	Cases ^	-4.89	-8.16 to -1.62	**0.003**
	Age (years)	0.08	-0.14 to 0.30	0.462
	Plantar flexion MVC (N)	0.01	0.00 to 0.02	**0.001**
Model 2	(Intercept)	80.35	71.40 to 89.30	**<0.001**
	Calf muscle strain injury ^	-5.06	-8.86 to -1.26	**0.009**
	Achilles tendinopathy ^	-4.74	-8.58 to -0.89	**0.016**
	Age (years)	0.08	-0.13 to 0.30	0.444
	Plantar flexion MVC (N)	0.01	0.00 to 0.02	**<0.001**

Legend: MVC=maximal voluntary contraction, ^In reference to controls

Table 3. Between group differences in corticospinal excitability

			Estimates (ß)	*95% Confidence Interval*	*p*
Active motor threshold	**Model 1**	(Intercept)	38.00	30.34 to 45.67	**<0.001**
		Cases ^	-7.26	-12.88 to -1.64	**0.011**
		Age (years)	0.18	-0.02 to 0.39	0.082
	Model 2	(Intercept)	45.39	37.17 to 53.60	**<0.001**
		Calf muscle strain injury ^	-6.67	-13.17 to -0.17	**0.044**
		Achilles tendinopathy ^	-7.84	-13.72 to -1.96	**0.009**
		Age (years)	0.18	-0.03 to 0.38	0.091
Motor evoked potential 120%	**Model 1**	(Intercept)	2.48	1.21 – 3.75	**<0.001**
		Cases ^	0.29	-0.26 to 0.84	0.307
		Age (years)	-0.00	-0.03 to 0.03	0.900
		Region: Medial gastrocnemius (mV)	0.13	-0.30 to 0.56	0.554
		Region: Soleus (mV)	-0.94	-1.38 to -0.50	**<0.001**
	Model 2	(Intercept)	2.17	1.22 to 3.12	**<0.001**
		Calf muscle strain injury ^	0.20	-0.45 to 0.84	0.545
		Achilles tendinopathy ^	0.38	-0.30 – 1.05	0.273
		Age (years)	-0.00	-0.03 – 0.03	0.933

		Region: Medial gastrocnemius (mV)	0.13	-0.30 – 0.56	0.554
		Region: Soleus (mV)	-0.94	-1.38 – -0.50	**<0.001**

Legend: ^In reference to controls

Table 4. Between group differences in intracortical excitability

			Estimates (ß)	*95% Confidence Interval*	*p*
Short interval intracortical inhibition	**Model 1**	(Intercept)	0.24	-0.74 to 1.21	0.635
		Cases ^	-0.04	-0.45 to 0.37	0.842
		Age (years)	0.01	-0.01 to 0.03	0.352
		Region: Medial gastrocnemius (mV)	-0.04	-0.25 to 0.17	0.731
		Region: Soleus (mV)	-0.09	-0.26 to 0.08	0.298
		Motor evoked potential 120%	0.49	0.39 to 0.59	**<0.001**
	Model 2	(Intercept)	0.28	-0.41 to 0.98	0.424
		Calf muscle strain injury ^	-0.02	-0.57 to 0.54	0.955
		Achilles tendinopathy ^	-0.07	-0.41 to 0.27	0.699
		Age	0.01	-0.01 to 0.03	0.381
		Region: Medial gastrocnemius (mV)	-0.04	-0.25 to 0.17	0.731
		Region: Soleus (mV)	-0.09	-0.26 to 0.08	0.299
		Motor evoked potential 120%	0.49	0.39 to 0.59	**<0.001**
Long interval intracortical facilitation	**Model 1**	(Intercept)	0.96	0.42 to 1.51	**0.001**
		Cases ^	0.29	0.04 to 0.55	**0.026**
		Age (years)	-0.00	-0.02 to 0.01	0.728

		Region: Medial gastrocnemius (mV)	-0.01	-0.20 to 0.17	0.880
		Region: Soleus (mV)	-0.36	-0.55 to -0.17	**<0.001**
		Motor evoked potential 120%	0.70	0.61 to 0.79	**<0.001**
	Model 2	(Intercept)	0.68	0.27 to 1.10	**0.001**
		Calf muscle strain injury ^	0.36	0.04 to 0.68	**0.029**
		Achilles tendinopathy ^	0.23	-0.06 to 0.52	0.122
		Age (years)	-0.00	-0.02 to 0.01	0.689
		Region: Medial gastrocnemius (mV)	-0.01	-0.20 to 0.17	0.879
		Region: Soleus (mV)	-0.36	-0.55 to -0.17	**<0.001**
		Motor evoked potential 120%	0.70	0.61 to 0.79	**<0.001**
Intracortical facilitation	**Model 1**	(Intercept)	0.69	0.20 to 1.18	**0.006**
		Cases ^	0.28	0.07 to 0.48	**0.009**
		Age (years)	-0.00	-0.01 to 0.01	0.933
		Region: Medial gastrocnemius (mV)	0.38	0.11 to 0.65	**0.006**
		Region: Soleus (mV)	-0.26	-0.43 to -0.09	**0.003**
		Motor evoked potential 120%	0.75	0.67 to 0.84	**<0.001**
	Model 2	(Intercept)	0.40	-0.01 to 0.81	0.054
		Calf muscle strain injury ^	0.22	-0.01 to 0.44	0.059

		Achilles tendinopathy ^	0.33	0.04 to 0.63	**0.027**
		Age (years)	-0.00	-0.01 to 0.01	0.995
		Region: Medial gastrocnemius (mV)	0.38	0.11 to 0.65	**0.006**
		Region: Soleus (mV)	-0.26	-0.43 to -0.09	**0.003**
		Motor evoked potential 120%	0.75	0.67 to 0.84	**<0.001**

Legend: ^In reference to controls

Appendix A. Contraindications to transcranial magnetic stimulation

Pregnancy
Neurological conditions/illness (including epilepsy/convulsion/seizure)
Vascular, traumatic, tumoural, infectious, or metabolic lesion of the brain
Previous or current implants in their body that may be triggered or heated by an electrical current (e.g. cardiac pacemaker, intracranial shunts, artificial cochlea, medication infusion device)
Any conducting mental implanted in their head (e.g. surgical clips, staples, shrapnel)
Frequent or intense headaches
Ringing in the ears or hearing problems
Previous brain trauma or neurosurgical intervention to the brain or spinal cord
Serious medical complications (e.g. advanced pulmonary, cardiac, liver or kidney disease);
Currently taking neuropsychotropic drugs (e.g. antiepileptics, neuroleptics, benzodiazepines) or drugs with an effect on neuroplasticity (dopamine, fluoxetine or D-amphetamine, sodium or calcium channel blockers, NMDA receptor antagonists)

Appendix B. Participant set up



Appendix C. Demographic information per group

		Control (n)	Achilles tendinopathy (n)	Calf muscle strain injury (n)
Sex	Female	9	3	4
	Male	11	13	15
Ethnicity	White	16	16	16
	Asian	3	0	0
	Hispanic or Latino	1	0	1
	Black	0	0	1
Education	Less than high school	0	0	1
	High school graduate	2	5	3
	Bachelor's degree	5	5	10
	Master's degree	8	5	4
	Doctoral degree	5	0	0
	Professional degree	0	1	
Employment status	Full time	5	11	15
	Part time	5	0	2
	Home maker	0	0	0
	Student	9	4	1
	Retired	0	0	0
	Other	1	1	0
Household income	Less than AUD $30,000	5	2	1
	Between AUD $30,000 - $49,999	2	0	0
	Between AUD $50,000 - $79,999	2	2	0
	Between AUD $80,000 - $99,999	3	0	2

	Between AUD $100,000 - $149,999	2	2	3
	Between AUD $150,000 - $199,999	4	1	4
	More than AUD $200,000	2	9	8

Legend: n=number, AUD=Australian dollars

Appendix D. Sensitivity analysis

Voluntary activation with sex			
Predictors	*Estimates*	*CI*	*p*
(Intercept)	76.55	60.07 to 93.03	**<0.001**
Cases ^	-4.07	-7.16 to -0.98	**0.010**
Age (years)	0.09	-0.12 to 0.29	0.410
Male sex	-5.65	-23.76 to 12.45	0.540
MVC (N)	0.01	-0.01 to 0.03	0.206
Sex × MVC (N)	0.00	-0.02 to 0.02	0.834
Active motor threshold with sex			
(Intercept)	37.59	28.14 to 47.04	**<0.001**
Cases ^	-7.38	-13.85 to -0.92	**0.025**
Age (years)	0.18	-0.02 to 0.39	0.081
Male sex	0.47	-5.78 –to 6.73	0.882
Motor evoked potential 120% with sex			
(Intercept)	1.71	0.49 to 2.92	**0.006**
Cases	0.06	-0.45 to 0.56	0.827
Age (years)	-0.00	-0.03 to 0.03	0.992
Region: Medial gastrocnemius (mV)	0.13	-0.30 to 0.56	0.554
Region: Soleus (mV)	-0.94	-1.38 to -0.50	**<0.001**
Male sex	0.88	0.51 to 1.26	**<0.001**
Motor evoked potential 120% with pre-stimulus EMG RMS amplitude			
(Intercept)	1.20	0.31 to 2.08	**0.008**
Cases ^	-0.03	-0.43 to 0.38	0.894
Age (years)	-0.00	-0.02 to 0.02	0.835
Region: Medial gastrocnemius (mV)	-0.04	-0.41 to 0.32	0.808
Region: Soleus (mV)	-0.92	-1.29 to -0.55	**<0.001**
Pre-stimulus EMG RMS amplitude	2.45	1.75 to 3.15	**<0.001**

Short interval intracortical inhibition with sex			
(Intercept)	-0.11	-0.97 to 0.76	0.810
Cases ^	-0.15	-0.52 to 0.23	0.450
Age (years)	0.01	-0.01 to 0.03	0.294
Male sex	0.40	0.18 to 0.62	**<0.001**
Region: Medial gastrocnemius (mV)	-0.04	-0.25 to 0.17	0.732
Region: Soleus (mV)	-0.09	-0.26 to 0.08	0.292
Motor evoked potential 120%	0.49	0.39 to 0.59	**<0.001**
Short interval intracortical inhibition with pre-stimulus EMG RMS amplitude			
(Intercept)	-0.11	-0.96 to 0.74	0.802
Cases ^	-0.14	-0.50 to 0.22	0.450
Age (years)	0.01	-0.01 to 0.03	0.374
Region: Medial gastrocnemius (mV)	-0.11	-0.31 to 0.10	0.307
Region: Soleus (mV)	-0.18	-0.35 to -0.01	**0.034**
Motor evoked potential 120%	0.45	0.35 to 0.55	**<0.001**
Pre-stimulus EMG RMS amplitude	1.11	0.70 to 1.52	**<0.001**
Long interval intracortical facilitation with sex			
(Intercept)	0.73	0.20 to 1.25	**0.007**
Cases ^	0.22	-0.03 to 0.48	0.089
Age (years)	-0.00	-0.01 to 0.01	0.792
Male sex	0.28	0.09 to 0.46	**0.004**
Region: Medial gastrocnemius (mV)	-0.01	-0.20 to 0.17	0.882
Region: Soleus (mV)	-0.36	-0.55 to -0.18	**<0.001**
Motor evoked potential 120%	0.70	0.61 to 0.79	**<0.001**
Long interval intracortical facilitation with pre-stimulus EMG RMS amplitude			
(Intercept)	0.40	-0.13 to 0.92	0.141
Cases ^	0.10	-0.16 to 0.36	0.440

Age (years)	-0.00	-0.01 to 0.01	0.639
Region: Medial gastrocnemius (mV)	-0.08	-0.27 to 0.10	0.374
Region: Soleus (mV)	-0.41	-0.59 to -0.24	**<0.001**
Motor evoked potential 120%	0.60	0.51 to 0.70	**<0.001**
Pre-stimulus EMG RMS amplitude	1.47	1.00 to 1.95	**<0.001**
Intracortical facilitation with sex			
(Intercept)	0.54	0.05 to 1.03	**0.030**
Cases ^	0.23	0.03 to 0.43	**0.023**
Age (years)	-0.00	-0.01 to 0.01	0.985
Male sex	0.18	0.01 to 0.34	**0.040**
Region: Medial gastrocnemius (mV)	0.38	0.11 to 0.65	**0.006**
Region: Soleus (mV)	-0.26	-0.43 to -0.09	**0.003**
Motor evoked potential 120%	0.75	0.66 to 0.84	**<0.001**
Intracortical facilitation with pre-stimulus EMG RMS amplitude			
(Intercept)	0.73	0.25 to 1.21	**0.003**
Cases ^	0.29	0.09 to 0.50	**0.005**
Age (years)	-0.00	-0.01 to 0.01	0.845
Region: Medial gastrocnemius (mV)	0.36	0.11 to 0.62	**0.006**
Region: Soleus (mV)	-0.27	-0.43 to -0.11	**0.001**
Motor evoked potential 120%	0.76	0.67 to 0.84	**<0.001**
Pre-stimulus EMG RMS amplitude	-0.02	-0.03 to -0.02	**<0.001**

Legend: CI = confidence interval
^In relation to controls

Appendix E. Maximal voluntary contraction generalised estimating equation model

		Estimates	*95%CI*	*p*
Model 1	(Intercept)	361.59	-131.75 to 854.94	0.151
	Cases	-48.81	-168.42 to 70.80	0.424
	Age (years)	-1.52	-7.43 to 4.39	0.614
	Male sex	104.16	-44.18 to 252.51	0.169
	Body mass (kg)	8.74	3.31 to 14.17	**0.002**
Model 2	(Intercept)	439.58	-14.70 to 893.85	0.058
	Calf muscle strain injury	-28.80	-193.83 to 136.23	0.732
	Achilles tendinopathy	-67.73	-191.39 to 55.93	0.283
	Age (years)	-1.69	-7.76 to 4.37	0.584
	Male sex	113.32	-47.23 to 273.87	0.167
	Body mass (kg)	8.34	2.59 to 14.09	**0.004**

Legend: CI = confidence interval, ^In relation to controls

Supplementary Files

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- AppendixBsetup.jpg
- Supplementarymaterial.docx