European Journal of Applied Physiology (2023) 123:633–643
https://doi.org/10.1007/s00421-022-05089-w

ORIGINAL ARTICLE

Lower motor unit discharge rates in gastrocnemius lateralis, but not in gastrocnemius medialis or soleus, in runners with Achilles tendinopathy: a pilot study

Gabriel L. Fernandes[1] · Lucas B. R. Orssatto[1] · Raphael L. Sakugawa[2] · Gabriel S. Trajano[1]

Received: 11 May 2022 / Accepted: 6 November 2022 / Published online: 23 November 2022

Abstract

Objectives Deficits in muscle performance could be a consequence of a reduced ability of a motor neuron to increase the rate in which it discharges. This study aimed to investigate motor unit (MU) discharge properties of each triceps surae muscle (TS) and TS torque steadiness during submaximal intensities in runners with Achilles tendinopathy (AT).

Methods We recruited runners with ($n = 12$) and without ($n = 13$) mid-portion AT. MU discharge rate was analysed for each of the TS muscles, using high-density surface electromyography during 10 and 20% isometric plantar flexor contractions.

Results MU mean discharge rate was lower in the gastrocnemius lateralis (GL) in AT compared to controls. In AT, GL MU mean discharge rate did not increase as torque increased from 10% peak torque, 8.24 pps (95% CI 7.08 to 9.41) to 20%, 8.52 pps (7.41 to 9.63, $p = 0.540$); however, in controls, MU discharge rate increased as torque increased from 10%, 8.39 pps (7.25–9.53) to 20%, 10.07 pps (8.89–11.25, $p < 0.001$). There were no between-group difference in gastrocnemius medialis (GM) or soleus (SOL) MU discharge rates. We found no between-group differences in coefficient of variation of MU discharge rate in any of the TS muscles nor in TS torque steadiness.

Conclusion Our data demonstrate that runners with AT may have a lower neural drive to GL, failing to increase MU discharge rate to adjust for the increase in torque demand. Further research is needed to understand how interventions focussing on increasing neural drive to GL would affect muscle function in runners with AT.

Keywords Achilles tendon · Running · Firing rate · Neural drive · Torque steadiness · Triceps surae · High-density electromyography

Abbreviations

AT	Achilles tendinopathy
GL	Gastrocnemius lateralis
GM	Gastrocnemius medialis
HD-EMG	High-density surface electromyography
MVIC	Maximal voluntary isometric contraction
SOL	Soleus

Communicated by Olivier Seynnes.

✉ Gabriel L. Fernandes
gabriel.fernandes@hdr.qut.edu.au

[1] Faculty of Health, School of Exercise and Nutrition Sciences, Queensland University of Technology (QUT), Brisbane, QLD, Australia

[2] Department of Physical Education, Federal University of Mato Grosso, Cuiaba, MT, Brazil

Introduction

Achilles tendinopathy (AT) is the most prevalent running injuries, accounting for about 6.2–9.5% of all running injuries (Lagas et al. 2020; Mousavi et al. 2019). AT is an overloading injury and although its aetiology is multifactorial (Cook et al. 2016), deficits in muscle performance is suggested to be a key factor (O'Neill et al. 2019; Mahieu et al. 2006), which seems to be maintained long after symptomatic recovery (Silbernagel et al. 2007). Several lines of evidence suggest that neural changes to the triceps surae might underpin some of these chronic motor deficits (Crouzier et al. 2020; Fernandes et al. 2021). In particular, it has been shown that individuals with AT have: (1) lower contribution of gastrocnemius lateralis (GL) to produce plantar flexor force Crouzier et al. 2020 and (b) greater levels of intra-cortical inhibition associated with lower plantar flexor endurance during single leg heel raise test (Fernandes et al. 2021) when compared to controls. Collectively, these findings suggest

that changes in how the central nervous system control muscles coordination (Hug and Tucker 2017) within the triceps surae (force distribution and activation) might impact load distribution to the tendon in individuals with AT. This is of particular importance because altered triceps surae coordination (due to lower individual muscle contribution to muscle force) could create uneven loading of the Achilles tendon and contribute to tendinopathy (Cook and Purdam 2009).

There has been some speculation about differences in recruitment strategies within the triceps surae in people with AT, with conflicting evidence about which muscle is affected. One study (O'Neill et al. 2019) suggested soleus (SOL) would be the main muscle responsible for the strength and endurance deficits observed in these individuals. The AT group had deficits in plantar flexor torque during dynamometry testing, irrespectively of knee position (knee flexed/extended) compared to controls. The authors reasoned that if the gastrocnemii were affected, deficits between groups would be larger during knee extended and smaller during knee flexed. However, determining force deficits in SOL in relation to the gastrocnemii solely by comparing torque measures between flexed and extended knee positions provides very limited and possibly inaccurate information about muscle recruitment patterns, neglecting all the neurophysiological mechanisms that enable force production in the first place (Enoka and Duchateau 2017). Conversely, runners with acute AT (Crouzier et al. 2020) (< 3 months) have about 22% lower contribution of GL during 20 and 40% of peak plantar flexor isometric torque but no differences in gastrocnemius medialis (GM) or SOL, compared to controls. Force-sharing contribution of individual muscles of the triceps surae was estimated for each muscle based on the root mean squared (RMS) of surface EMG (electromyography) signal amplitude and other muscle characteristics (i.e. physiological cross-sectional area). Even though data from surface EMG signal is somewhat limited in estimating changes in neural drive to a specific muscle (Martinez-Valdes et al. 2018), this result suggests that individual muscles recruitment strategies might be altered in AT.

From a neurophysiological perspective, the force exerted by a muscle depends, partly, on the recruitment and discharge rates of the motor units (Enoka and Duchateau 2017). Thus, deficits in motor performance could be a result of a reduced ability to recruit motor units and/or to increase the rate at which motor neurons' discharge (Enoka and Duchateau 2017). The analysis of individual motor unit discharge rates from each muscle of the triceps surae (Hug et al. 2020) is a more reliable way of investigating the central nervous system strategy of recruitment of the triceps surae muscle (i.e. neural drive), than the typical and limited interference EMG (Souza et al. 2018). This method has been also used in other studies to estimate changes in neural drive to specific muscles in individuals with other chronic musculoskeletal conditions such as ACL injury (Nuccio et al. 2021) and patellofemoral pain (Martinez-Valdes et al. 2019).

Furthermore, reduced control of the plantar flexors could create tendon overload, progressing to early stages of tendinopathy (Cook and Purdam 2009). Increased fluctuation in torque (torque steadiness) is associated with painful musculoskeletal conditions such as knee osteoarthritis or patellofemoral pain (Martinez-Valdes et al. 2019; Rice et al. 2015) or following ACL reconstruction (Telianidis et al. 2014) and could occur as consequence of greater variation in motor unit discharge rate (Enoka and Farina 2021). Coefficient of variation of motor unit discharge represents, at an individual muscle level, the ability to effectively control muscle torque and it is an important measure that can help explain motor performance (Enoka and Duchateau 2017).

Thus, this study aimed to: (i) investigate differences in neural drive to each muscle of the triceps surae during submaximal plantar flexor contractions in individuals with AT; (ii) determine between-group differences in coefficient of variation of motor unit discharge rate and torque steadiness. We hypothesised that motor unit mean discharge rate of each individual muscles of the triceps surae would be lower in the tendinopathy group, associated with differences between muscles of the triceps surae in the AT group. We also hypothesised the AT group would have increased variability in motor unit discharge rate and torque steadiness compared to controls.

Methods

This was a cross-sectional study comparing runners with and without mid-portion AT. A sample size of 18 participants (9 per group) was calculated based on a similar study (Gallina et al. 2018) (GPower software parameters: effect size $F = 0.40$; α err prob: 0.05; power 0.95; $n = 9$ per group). 25 endurance runners were recruited for this study, 12 with mid-portion AT (7 males, 44.3 years old ± 95% CI 6.7, 173 cm ± 5.7, 76.2 kg ± 9.3) and 13 healthy controls (7 males, 34.0 years old ± 4.2, 171 cm ± 5.6, 64.8 kg ± 7.0), with a running routine of more than twice weekly for more than 4 months. Runners were recruited from local running clubs, via email and social media. Participants from this study are the same from a separate study (one more participant in the AT group) (Fernandes et al. 2021). Torque measures such as absolute and normalised plantar flexor peak isometric torque and explosive torque were not different between the groups, as have been reported previously (Fernandes et al. 2021).

All volunteers were endurance runners, recruited from local running clubs around Southeast Queensland, Australia. Diagnosis of mid-portion AT was confirmed by an experienced physiotherapist during examination if patients

presented with localised mid-portion Achilles tendon pain for more than 3 months, pain provoked by physical activities in a dose dependent way and had pain with palpation at the mid-portion of tendon. Volunteers were excluded if presenting insertional AT; previous rupture or surgery of the Achilles tendon; clinical findings indicating a differential diagnosis for the Achilles tendon pain (such as tendon tear); regular participation in other sports involving high speed running (football, rugby, AFL etc.), 4) VISA-A score > 90 points for AT group and < 100 for the healthy group; any other musculoskeletal injuries of the lower limb; any neurological disorder; or mental health issues affecting consent. All participants were free of comorbidities such as cardiac, pulmonary, renal, and endocrine of gastrointestinal and were not taking any medication for tendon pain or that would affect tendon structure (Knobloch 2016).

Prior to testing, all participants read and signed a detailed informed consent document and completed the VISA-A questionnaire (Martin et al. 2018). The average VISA-A score for the AT group was 70.1 ± 5.7 and 100 ± 0 for the control group. The AT group had a running routine of 38.7 ± 9.1 km/week and 30.4 ± 8.4 km/week for the control group; mean difference of 8.3 ± 12.2. This study was approved by the Queensland University of Technology Human Research and Ethics Committee in line with the Declaration of Helsinki. Data collection was conducted during the COVID-19 pandemic and all safety procedures followed local state government policies.

Data collection and analysis

Plantar flexor isometric peak torque was measured using an isokinetic dynamometer (Biodex Medical Systems, Shirley, New York). For the bilateral AT presentations ($n = 3$), the most symptomatic leg was used and for the control group, the dominant leg was used for testing. Leg dominance was selected by asking the participants what their preferred leg was, and if participants were unsure, they were asked which leg they would use to kick a ball. Participants were sitting (75 degrees of hip flexion) with their knee straight and with the foot perpendicular to shank. Warm up consisted of 2 × 4 s isometric contractions of each participant's perceived 20, 40, 60 and 80% maximal voluntary isometric contraction intensity. After warm-up, participants performed at least three maximal voluntary isometric contractions, until < 5% variation was observed between contractions, and the highest value was used. Thereafter, participants performed three trapezoidal submaximal isometric contractions with each target intensity based on their peak isometric torque (3 × 10% and 3 × 20% peak torque) in a randomised order. For each intensity participants had four attempts to get familiarised with task before recordings. Rate of torque rise and decline was standardised at 10% peak torque/s between contractions with different intensities, with a 10 s sustained plateau at the top, followed by 1 min of rest between contractions (Boccia et al. 2019; Vecchio et al. 2019). Participants received real-time visual feedback of the trapezoidal pathway, displayed in a monitor placed at 1 m away from the participant. During the plantar-flexors trapezoidal contractions, HD-EMG (Sessantaquattro, OTBioelettronica, Torino, Italy) signals were recorded with OT Biolab + software (version 1.3.0., OTBioelettronica, Torino, Italy), from SOL, GM and GL. After skin preparation (shaving, light abrasion, and cleansing of area with alcohol), electrodes were positioned following the estimated muscle fibres orientation using a bi-adhesive layer with a conductive paste to ensure good skin–electrode contact and conductibility. One 32-channel electrode grid (ELSCH032NM6, OTBioelettronica, Torino, Italy) was placed on GM, one 32-channel electrode grid on GL and two 32-channel electrodes grid on SOL, one laterally and one medially to the Achilles tendon (Fig. 1). Two electrodes were used on SOL to increase the number of identified motor units. Data from both electrodes were clustered into one file to increase motor unit yield, prior to analysis of SOL motor unit characteristics. The ground strap electrode (WS2, OTBioelettronica, Torino, Italy) was dampened and positioned around the ankle joint of the tested leg. The EMG signals were recorded in monopolar mode, amplified (256×), band-passed filtered (10–500 Hz) and converted to digital signal at 2048 Hz by a 16-bit wireless amplifier (Sessantaquattro, OTBioelettronica, Torino, Italy), before being stored for offline analysis. Since the grid adapter device (AD2 × 32SE, OTBioelettronica, Torino, Italy) has only two channels for electrode connection, each intensity of the protocol had to be performed twice, once with electrodes connected to the gastrocnemii and a second time with the electrodes connected SOL, this order was randomised for each intensity. Torque signal was recorded and analysed with OT Biolab + software. HD-EMG signal was recorded and analysed offline, decomposed into motor unit spike trains, then converted into instantaneous discharge rates with specialised software using blind source separation decomposition technique using DEMUSE tool software (v.4.1; The University of Maribor, Slovenia) (Vecchio et al. 2020). For each muscle and for each intensity, the 2 best contractions, with the lowest deviation from trapezoidal torque trajectory, were combined in one file and motor unit tracked across the 2 contractions at the same intensity for analysis. All motor units were visually inspected, erroneous discharge times were excluded, and missed discharges included (Vecchio et al. 2020). Manual inspection is required to reduce automatic decomposition discharge errors and improve data reliability (Martinez-Valdes et al. 2016). Reliability for manual inspection across operators is very high for motor unit mean discharge rate and recruitment with intra-class correlation coefficient (ICC) of > 0.99 (Hug et al. 2021). We have also calculated ICC for

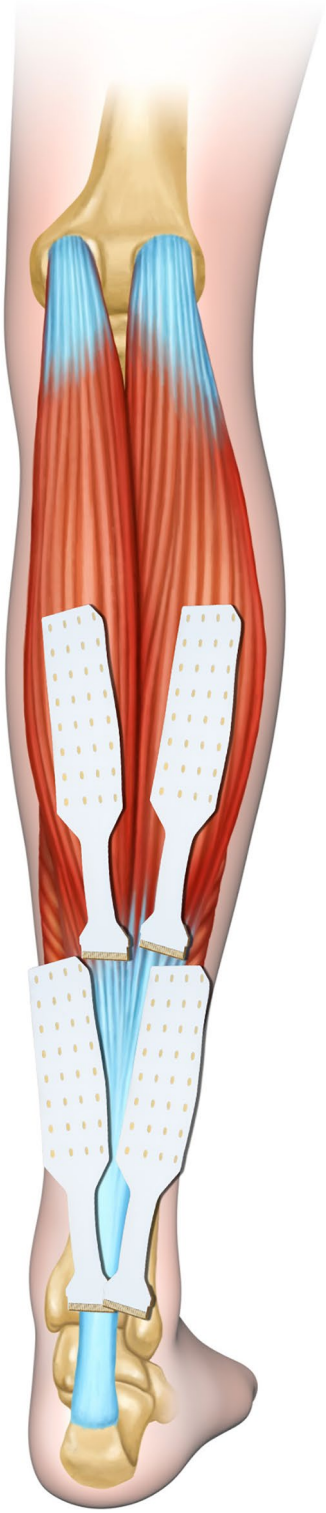

Fig. 1 Schematic representation of electrode positioning used for data acquisition for all three muscles of the triceps surae (*GM* gastrocnemius medialis, *GL* gastrocnemius lateralis and *SOL* soleus. One electrode was placed over GM, one electrode over GL, and two electrodes over SOL, one medially and one laterally to the Achilles tendon

our data for motor unit discharge rate across the 2 contractions for each intensity, per muscle for each group and it is shown in Table 1. Only motor units with a pulse-to-noise ratio (PNR) > 30 dB, sensitivity > 90%, were used for data analysis (Vecchio et al. 2019, 2020). For participants that yield no good quality motor units (PNR > 30 dB) after motor unit tracking across the 2 contractions at the same intensity (Orssatto et al. 2021; Frančič and Holobar 2021), the best single contraction (higher PNR per motor units) was used for analysis with the motor unit discharge characteristics inspected as mentioned above. Due to the reduced number of the same motor units found across intensities, motor unit tracking across intensities was not feasible and, therefore, not used for analysis. Assessor who performed motor unit analysis process was not blinded by group. Motor units were collected from the 10 s isometric plateau, the first and last two seconds were excluded and analysis of mean motor unit mean discharge rate, coefficient of variation of motor unit discharge rate and torque steadiness were performed from the central 6 s of the isometric plateau. Motor unit mean discharge rates and coefficient of variation of motor unit discharge rates were then calculated for each muscle (SOL, GM and GL) and intensity tested (10 and 20% peak torque).

Torque steadiness was analysed as the coefficient of variation in torque for each torque intensity tested. Torque was filtered (10 Hz, 4th order, low pass). To reduce intrasubject variability, data from the coefficient of variability of torque were averaged across the 4 contractions for each of the two torque intensities tested, the 2 contractions recorded during gastrocnemii testing and from the 2 contractions during SOL testing (Jakobi et al. 2020).

Statistical analysis

All analyses were performed using R studio (version 1.3.1093). Models were fitted using the *lme4* package (Bates et al. 2015). Separate linear mixed-effect models were used to compare motor unit mean discharge rates and coefficient of variation of motor unit discharge rate of identified motor units for each muscle (SOL, GM, GL); between intensities (10 and 20%) and groups (AT and control). We tested the model using a random intercept (participant ID) and slope (recruitment threshold by intensity) for each participant in the study to account for the influence of motor unit populations and the correlation between repeated observations in each participant. The estimated marginal mean difference and 95% confidence intervals (CI) for all variables (motor unit mean discharge rate, coefficient of variation of motor unit discharge rate, between groups and torque steadiness) were determined using the *emmeans* package (Lenth 2016). Normality assumptions were confirmed by analysis of the histogram of residuals, Q–Q Plot, and the residual-predicted scatterplot. Independent *t* test was used to compare torque

Table 1 ICC for MU discharge rate across the 2 contractions for each intensity, per muscle for each group

Muscle % peak torque	AT-ICC (95% CI)	AT-CV	Control-ICC (95% CI)	Control-CV
SOL 10% peak torque	0.91 (0.88–0.93)	5.6	0.94 (0.92–0.60)	4.3
SOL 20% peak torque	0.93 (0.90–0.95)	4.4	0.86 (0.79–0.90)	6.5
GM 10% peak torque	0.86 (0.80–0.90)	5.6	0.97 (0.96–0.98)	3
GM 20% peak torque	0.95 (0.95–0.97)	2.6	0.98 (0.97–0.99)	1.8
GL 10% peak torque	0.71 (0.37–0.86)	8	0.95 (0.93–0.97)	3.9
GL 20% peak torque	0.90 (0.82–0.95)	6.4	0.82 (0.64–0.90)	7.7

Data are presented as intra-class correlation coefficient-ICC with lower to upper 95% confidence interval in brackets, and coefficient of variation-CV

steadiness between groups for each torque intensity. An alpha level of 5% was set for statistical significance for all tests, and when appropriate, Bonferroni post hoc analysis was performed. Data are presented as mean ± 95% CI. Data for total number of motor units per group are presented as (mean ± SD).

Results

Motor unit identification

We found a total of 1.055 motor units, 518 motor units in the AT group (43.1 ± 7.0, per participant) and 537 motor units in the control group (41.4 ± 6.3, per participant) across all muscles and intensities. The total number, mean and standard deviation of identified motor units per group, muscle, and contraction intensity are reported in Table 2. The number of identified motor unit for each participant per muscle and intensity is reported in Supplementary material 1. GL was the muscle with the least amount motor unit found in single contractions, and some were lost during motor unit tracking between the two contractions of the same intensity.

Motor unit discharge rate

Analysis of SOL motor unit mean discharge rate showed difference between torque intensities ($F = 20.118$, $p < 0.001$, $\eta^2 p = 0.04$) but no differences between groups ($F = 0.324$, $p = 0.574$) or intensity × group interaction ($F = 0.512$, $p = 0.474$). There was an increase in motor unit mean discharge rate in both groups as torque increased. In the AT group, motor unit mean discharge rate increased from 6.98 pps (6.72–7.24) at 10% peak torque to 7.29

Table 2 Total number, mean and SD of identified motor units per group, muscle, and contraction intensity

AT (%)	Mean (±SD)	*n*	Total number of MU
SOL (10)	10 (±5.5)	12	120
SOL (20)	10 (±5.5)	12	120
GM (10)	11.2 (±8.3)	9	101
GM (20)	11.6 (±9.0)	11	128
GL (10)	2.8 (±2.2)	7	20
GL (20)	3.6 (±2.1)	8	29
Control (%)	Mean	*n*	Total number of MU
SOL (10)	9.7 (±6.6)	13	127
SOL (20)	9.5 (±6.6)	12	114
GM (10)	9.4 (±5.9)	12	113
GM (20)	11.4 (±6.4)	10	114
GL (10)	5.3 (±6.0)	8	43
GL (20)	3.7 (±2.8)	7	26

Data represent mean (standard deviation) number of MU per participant. It is also reported the number of participants (n) who yield MU and total number of MU found per muscle and per contraction intensity. Participants who had no MU were not included in this analysis

pps (7.02–7.55) at 20%; the motor unit mean discharge rate in the control group also increased, from 7.40 pps (7.16–7.63) at 10% peak torque to 7.86 pps (7.55–8.16) (Fig. 2).

Similar to SOL, in GM analysis, we observed differences in motor unit mean discharge rate between different torque intensities ($F = 75.554$, $p < 0.001$, $\eta^2 p = 0.15$) but no differences between groups ($F = 0.488$, $p = 0.492$) or intensity × group interaction ($F = 1.063$, $p = 0.303$). In both groups, motor unit mean discharge rate increases with the increase in torque intensity. In the AT group, motor unit mean discharge rate increased as torque increased from 10%, 8.38 pps (7.44–9.33) to 20% peak torque, 9.54 pps (8.61–10.48). The same was observed in the control group, motor unit mean discharge rate increased as torque increased from 10%, 8.63 pps (7.75–9.51) to 20%, 10.10 pps (9.21–11.00) (Fig. 3). On the other hand, in GL, we found an intensity × group interaction ($F = 27.955$, $p = 0.001$, $\eta^2 p = 0.11$). While in the AT group, motor unit mean discharge rate did not change as torque increased from 10% peak torque, 8.24 pps (7.08–9.41) to 20%, 8.52 pps (7.41–9.63, $p = 0.540$); however, in the control group, motor unit mean discharge rate increased as torque increased from 10%, 8.39 pps (7.25–9.53) to 20% peak torque, 10.07 pps (8.89–11.25, $p < 0.001$), (Fig. 4, ** denotes statistical difference). The control group had a higher motor unit mean discharge rate at 20% torque compared to the AT group.

Coefficient of variation of motor unit discharge rate

SOL had no difference in coefficient of variation of motor unit discharge rate between intensities ($F = 2.963$, $p = 0.086$) or between groups ($F = 0.151$, $p = 0.700$). In the AT group, the coefficient of variation of motor unit discharge rate was 10.3% (8.8–11.8) at 10% peak torque and 9.3% (7.9–10.7) at 20% peak torque; in the control group, the coefficient of variation of motor unit discharge rate was 10.2% (8.8–11.7) at 10% peak torque and 10.0% (8.6–11.5) at 20% peak torque. GM presented difference in coefficient of variation of motor unit discharge rate between intensities ($F = 51.203$, $p < 0.001$, $\eta^2 p = 0.10$) but not between groups ($F = 3.673$, $p = 0.07$) and had no intensity × group interaction ($F = 0.872$, $p = 0.350$). In the AT group, the coefficient of variation of motor unit discharge rate was 12.15% (11.1–13.1) at 10% peak torque and 9.16% (8.1–10.1) at 20% peak torque; in the control group, the coefficient of variation of motor unit discharge rate was 10.75% (9.8–11.7) at 10% peak torque and 8.45% (7.4–9.5) at 20% peak torque. In GL, as well as in GM, we observed difference between intensities ($F = 5.222$, $p = 0.024$, $\eta^2 p = 0.05$) but not between groups ($F = 0.661$, $p = 0.428$) or intensity × group interaction ($F = 2.779$, $p = 0.098$). In the AT group, the coefficient of variation

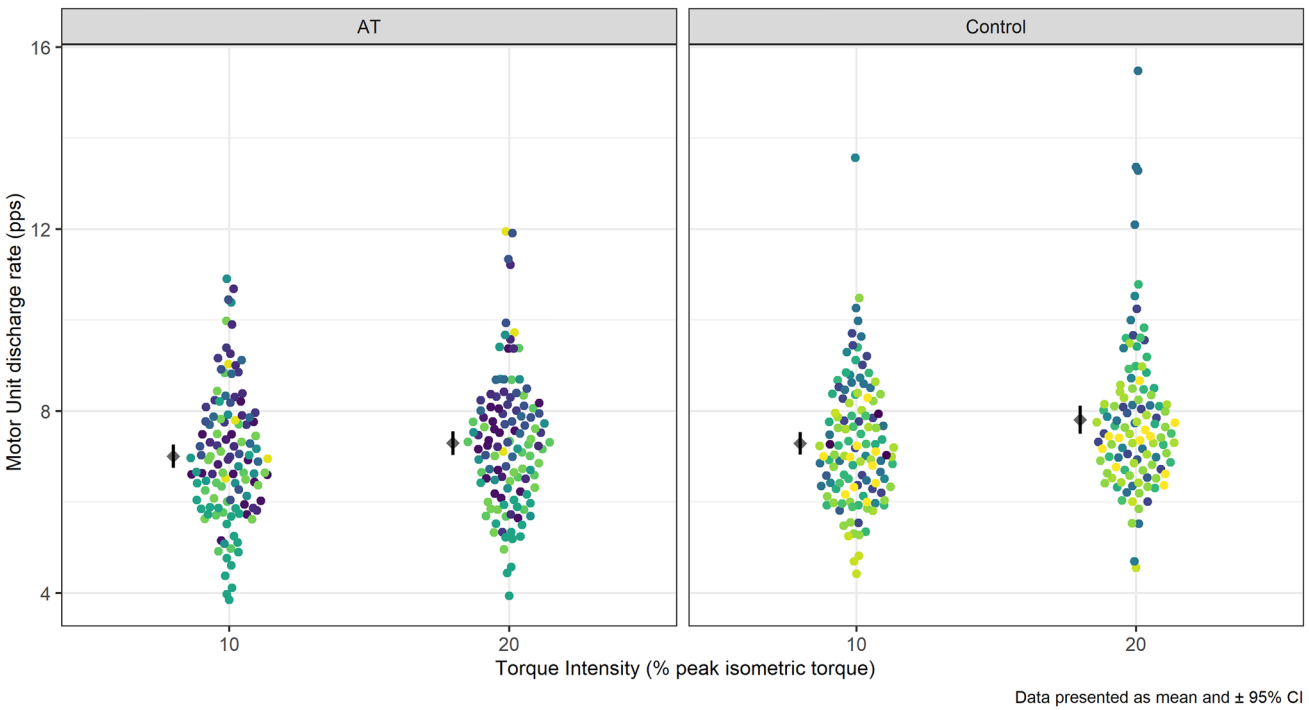

Fig. 2 Motor unit mean discharge rate of soleus during 10 and 20% peak isometric contraction. Each dot represents a single motor unit data point, coloured by participants. Mean and 95% confidence interval are offset to the left to facilitate visualisation. *pps* pulse per second

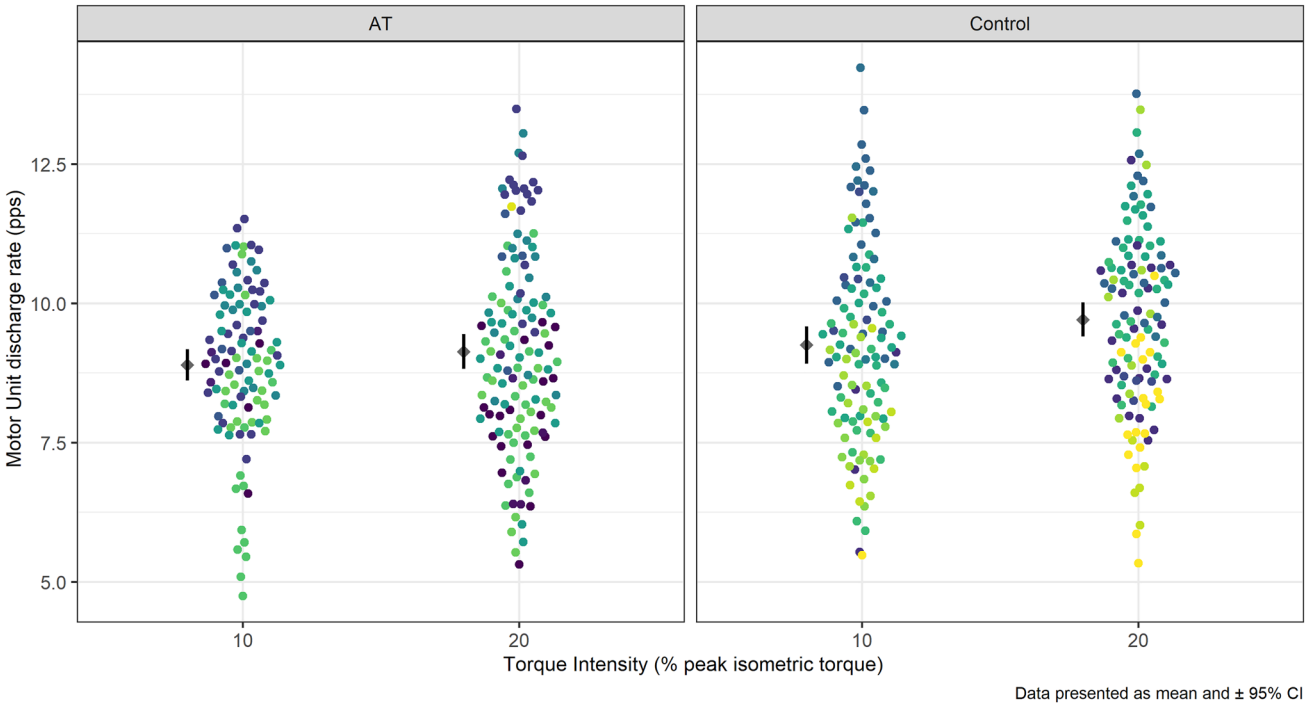

Fig. 3 Motor unit mean discharge rate of gastrocnemius medialis during 10 and 20% peak isometric contraction. Each dot represents a single motor unit data point, coloured by participants. Mean and 95% confidence interval are offset to the left to facilitate visualisation. *pps* pulse per second

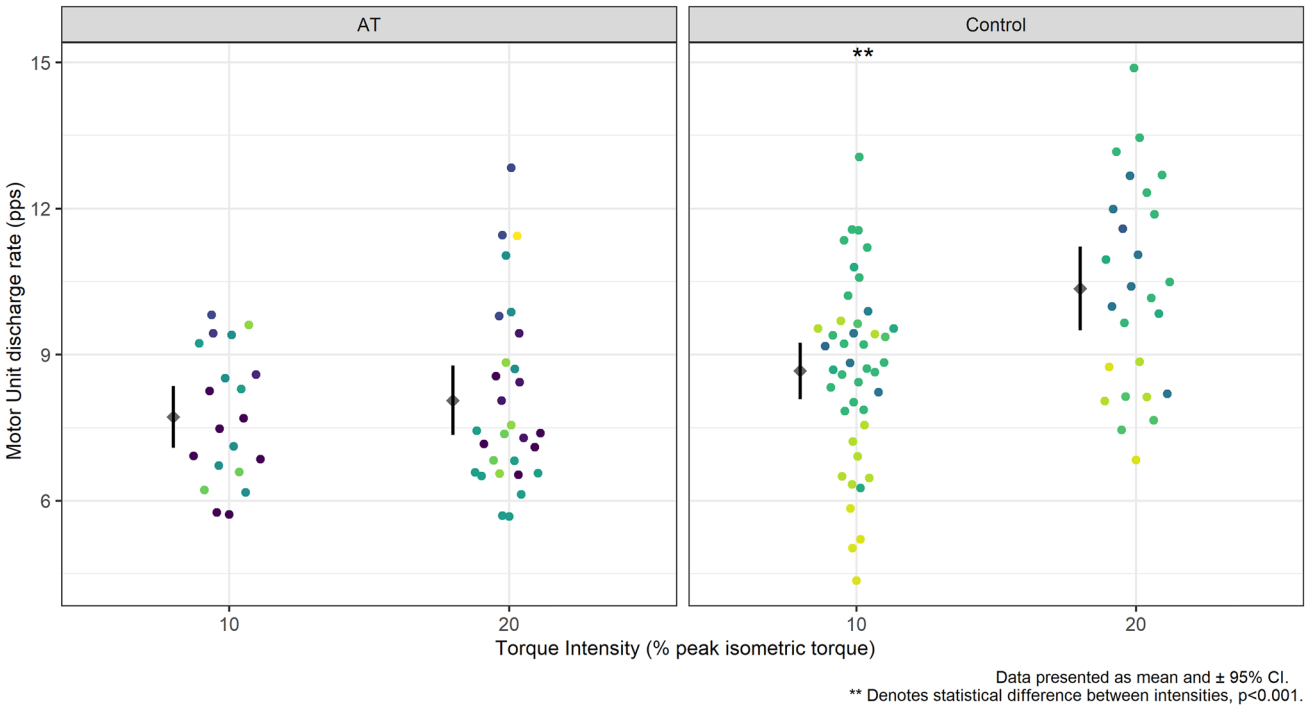

Fig. 4 Motor unit mean discharge rate of gastrocnemius lateralis during 10 and 20% peak isometric contraction. Each dot represents a single motor unit data point, coloured by participants. Mean and 95% confidence interval are offset to the left, to facilitate visualisation. *pps* pulse per second

of motor unit discharge rate was 10.9% (8.5–13.2) at 10% peak torque and 11.9% (9.6–14.0 at 20% peak torque; in the control group, the coefficient of variation of motor unit discharge rate was 12.7% (10.5–14.9) at 10% peak torque and 12.9% (10.5–15.2) at 20% peak torque.

Torque steadiness

There were no differences in torque steadiness analysis between groups in either of the two intensities analysed. Mean coefficient of variation of torque at 10% peak torque, in the AT group was 1.06 (0.79–1.32) and 1.13 (0.90–1.37, $p=0.656$) in the control group; and at 20%, mean coefficient of variation in torque in the AT group was 0.80 (0.57–1.03) and 0.92 (0.73–1.11, $p=0.375$) in the control group.

Discussion

Main findings

The present study aimed to determine if runners with chronic mid-portion AT had lower neural drive to the triceps surae and if there were muscle-specific differences in motor unit discharge characteristics within the triceps surae. For that, we analysed motor unit mean discharge rate and coefficient of variation of motor unit discharge rate of each individual muscle of the triceps surae during isometric contractions of increasing intensities. We also aimed to determine if the AT group had lower torque steadiness.

Our data indicate that runners with AT have lower neural drive to GL during the increase in plantar flexor isometric torque output. We confirmed our primary hypothesis, demonstrating a muscle-specific difference in neural drive in the AT group and a lower neural drive to GL during the increase in plantar flexor torque, not observed in the control group. However, we had also hypothesised that the neural drive to the triceps surae of the AT group would be lower, but GM and SOL were no different from controls. Furthermore, we did not confirm our second hypothesis, as we found no differences in the coefficient of variation of motor unit discharge rate in any of the muscles, nor did we find differences in triceps surae torque steadiness between groups.

Mean motor unit discharge rates

It has been previously identified that the three muscles of the triceps surae, although synergists as ankle plantar flexors, may have an independent neural drive from one another, allowing independent recruitment strategies for better joint control Hug et al. 2020. Our study also observed independent neural drive within the triceps surae. Individuals have a unique muscle activation pattern (Hug et al. 2022). Such activation signature has been shown robust for triceps surae isometric contractions across days and contractions (Hug et al. 2022; Crouzier et al. 2019). Further, we found different neural strategies between groups in only one of the three muscles of the triceps surae. Our data show that the AT group does not use the GL as effectively as healthy controls to match the increase in plantar flexor torque intensity. The AT group had lower motor unit mean discharge rate with the increase in torque, outlining a change in muscle coordination in GL that was not observed in the control group. During a voluntary contraction, muscle force is dependent of the number of the MU recruited and the rate of which they discharge (Enoka and Duchateau 2017). One possible explanation for the lower discharge rate observed in GL in the AT group during the submaximal isometric contractions, without an increase in GM or SOL discharge rate while matching same torque, is the increase in the number of motor unit recruited in GM and/or SOL rather than the increase in motor unit discharge rate. It is also possible instead of the increase in neural drive in GL from 10 to 20% observed in the control group, in the AT group this neural drive was distributed between GM and SOL, yet not sufficiently large to be observed in our sample. Another possible explanation is that the AT group used other muscles to increase plantarflexion torque such as *flexor hallucis longus (FHL)*, which was not measured in this study. Increased FHL EMG activity has been reported in the painful side of unilateral AT presentations during isometric submaximal plantarflexion contractions, compared to asymptomatic side and to controls side (Masood et al. 2014).

Similar findings of lower GL activity have been reported in another study with runners with AT Crouzier et al. 2020. The authors used the physiological cross-sectional area and normalised RMS EMG to calculate the index of force of each muscle and estimate individual muscles' contribution to triceps surae force production. GL had a significantly lower contribution to overall triceps surae force output; suggesting a lower neural drive compared to healthy counterparts. Muscle force depends on motor unit discharge rate, which is proportional to the neural drive to the muscle. In healthy individuals, motor unit discharge rate increases to adjust for an increase in torque intensity (Enoka and Duchateau 2017). Contrary to what was previously suggested in the literature (O'Neill et al. 2019), we found no differences in SOL motor unit mean discharge during the increase in torque, which suggests that at least for the condition and type of task considered in the current study, SOL contribution to plantar flexor force is not impaired in AT.

The lower neural drive to GL observed in our study seems relevant in the persistent muscle deficits observed in AT (Silbernagel et al. 2007). Perhaps current treatment strategies for

AT fail in effectively rehabilitating GL function; therefore, maintaining this lower neural drive and contribution to force production during ankle plantar flexion.

The neural drive to each individual muscles of the triceps surae can be influenced independently by strategies such as modified feet position during plantar flexion (Hug et al. 2020). Therefore, utilising strategies to increase GL recruitment and contribution during exercise is important, as altered muscle coordination may lead to unequal loading to the Achilles tendon (Hug and Tucker 2017). Performing heel raises with the foot positioned with toes pointed inwards significantly increased GL motor unit discharge rate compared to toes neutral in healthy individuals (Hug et al. 2020). Foot position, in healthy individuals, can also selectively affect GM and GL hypertrophy (Nunes et al. 2020). Therefore, implementing different foot positions during rehabilitation could help increase GL activity during plantar flexor resistance training. Rehabilitation programs using different foot positions during triceps surae resistance training should be studied in patients with AT to explore how this lower contribution of GL in triceps surae torque, impacts AT and if implementing treatment strategies to increase the neural drive to GL would influence tendon pain and function in AT.

Coefficient of variation of motor unit discharge rate and torque steadiness

Torque variability was measured during 10 and 20% relative peak isometric torque plateau. We found no differences between groups in coefficient of variation of motor unit discharge in any of the muscles of the triceps surae nor did we find differences in triceps surae torque steadiness. All three muscles of the triceps surae were equally matched to controls in the two submaximal intensities tested. Coefficient of variation of motor unit discharge represents, at an individual muscle level, the ability to effectively control muscle torque and it is an important measures that can help explain motor performance (Enoka and Farina 2021; Negro et al. 2009). Fluctuations in torque, coefficient of variation of motor unit discharge rate and torque steadiness are more variable in lower intensities than in higher torque intensities, hence why 10 and 20% intensities were used for analysis (Enoka and Farina 2021).

Based on our findings, the ability of the triceps surae in controlling torque during submaximal contractions is not affected in runners with AT, which aligns with another study (Vallance et al. 2019). Torque steadiness is affected by pain (Rice et al. 2015), and lower torque steadiness has been reported in other chronic (Rice et al. 2015) and painful musculoskeletal conditions (Martinez-Valdes et al. 2019; Telianidis et al. 2014). In our study, we used submaximal torque intensities and none of our participants reported pain during testing; however, we cannot assert if such changes in torque steadiness and coefficient of variation of motor unit discharge rate would not occur during activities that provoke pain in this group, such as running.

Limitations

We were unable to effectively track the same motor unit across from 10 to 20% peak torque. We tried tracking the same motor units across the two intensities, but this has markedly reduced the number of motor units left for analysis. Therefore, we cannot say with certainty the change in motor unit discharge rate, or lack of thereof, observed from 10 to 20% MVIC across muscles occur in the same motor unit. Although both intensities used in this study are considered as of low threshold, each motor unit is unique from another, and motor unit tracking would have provided more robust information about each motor unit unique response to the increase in torque. The EMG device used was limited to up to 2×32-channel adaptor, not allowing sampling of all three muscles at the same time. Our study shows preliminary evidence of a possibly lower neural drive to GL. However, it is worth mentioning the reduced number of MU found in GL per participant, which reduced sample sizes in both groups. Thus, future studies with larger sample sizes should consider recording all three muscles using electrodes with more channels (i.e. 64-electrode grid) to increase the number of motor unit identified during decomposition when estimating neural drive to the triceps surae allowing tracking of the same motor unit between intensities to confirm our findings. Another limitation that should be highlighted is the type of contraction used for analysis. HD-EMG analysis provides reliable estimates of motor unit discharge rates (Martinez-Valdes et al. 2016); however, it requires isometric contractions for motor unit analysis. Thus, the observations of neural drive from this study cannot be extrapolated into dynamic tasks such as heel raises or running. Furthermore, we used submaximal intensities of relative peak isometric torque, as this facilitates motor unit identification; therefore, it is possible that during higher torque intensities, which demand more torque of each individual muscles, the differences observed in this study would be greater.

Conclusion

Our data suggest that runners with mid-portion AT have a muscle-specific deficit in the triceps surae, possibly creating heterogeneous loading to the Achilles tendon and contributing for the high recurrences (Martin et al. 2018) of AT. We observed lower motor unit discharge rate, (i.e. lower neural drive) in GL during the increase in plantar flexor torque demands but not in GM or SOL. This deficit in neural drive in GL might be greater during activities that require greater

plantar flexor torque, which could contribute to overload the Achilles tendon. Different strategies to try and increase GL activation during plantar flexion resistance training could be beneficial for AT, such as adopting different feet position during heel raise. Such rehabilitation strategy should be studied in patients with AT to further understand how the lower contribution of GL impacts Achilles tendinopathy and how implementing strategies to increase the neural drive to GL would affect AT patient outcomes.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00421-022-05089-w.

Acknowledgements The authors declare having received no funding for this study. The authors thank all the volunteers who participated in this study for their contribution to the development and achievement of this research.

Author contributions GLF and GST designed the study. GLF and LBRO conducted the experiments. GLF analysed the data and drafted the first version of the manuscript. RLS developed a MATLAB script for motor unit discharge rate, recruitment threshold, coefficient of variation of motor unit discharge rate and torque steadiness analyses. GLF, LBRO and GST critically revised the manuscript. All the authors read and approved this final version of the manuscript.

Data availability Dataset and R code are available at https://github.com/GabeFernandess/MU_TS_runners_AT.

Declarations

Conflict of interest The authors declare no conflict of interest with the present research.

References

Bates D, Mächler M, Bolker BM, Walker SC (2015) Fitting linear mixed-effects models using lme4. J Stat Softw. https://doi.org/10.18637/jss.v067.i01

Boccia G, Martinez-Valdes E, Negro F, Rainoldi A, Falla D (2019) Motor unit discharge rate and the estimated synaptic input to the vasti muscles is higher in open compared with closed kinetic chain exercise. J Appl Physiol 127(4):950–958. https://doi.org/10.1152/japplphysiol.00310.2019

Cook JL, Purdam CR (2009) Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med 43(6):409–416. https://doi.org/10.1136/bjsm.2008.051193

Cook JL, Rio E, Purdam CR, Docking SI (2016) Revisiting the continuum model of tendon pathology: what is its merit in clinical practice and research? Br J Sports Med 50(19):1187–1191. https://doi.org/10.1136/bjsports-2015-095422

Crouzier M, Hug F, Dorel S, Deschamps T, Tucker K, Lacourpaille L (2019) Do individual differences in the distribution of activation between synergist muscles reflect individual strategies ? Exp Brain Res 237(3):625–635. https://doi.org/10.1007/s00221-018-5445-6

Crouzier M, Tucker K, Lacourpaille L et al (2020) Force-sharing within the triceps surae: an Achilles heel in Achilles tendinopathy. Med Sci Sport Exerc 52:1076–1087. https://doi.org/10.1249/MSS.0000000000002229

de Souza LML, Cabral HV, de Oliveira LF, Vieira TM (2018) Motor units in vastus lateralis and in different vastus medialis regions show different firing properties during low-level, isometric knee extension contraction. Hum Mov Sci 58(November):307–314. https://doi.org/10.1016/j.humov.2017.12.012

Del Vecchio A, Negro F, Holobar A et al (2019) You are as fast as your motor neurons: speed of recruitment and maximal discharge of motor neurons determine the maximal rate of force development in humans. J Physiol 597(9):2445–2456. https://doi.org/10.1113/JP277396

Del Vecchio A, Holobar A, Falla D, Felici F, Enoka RM, Farina D (2020) Tutorial: analysis of motor unit discharge characteristics from high-density surface EMG signals. J Electromyogr Kinesiol 53:102426. https://doi.org/10.1016/j.jelekin.2020.102426

Enoka RM, Duchateau J (2017) Rate coding and the control of muscle force. Cold Spring Harb Perspect Med 7(10):1–12. https://doi.org/10.1101/cshperspect.a029702

Enoka RM, Farina D (2021) Force steadiness: from motor units to voluntary actions. Physiology 36(2):114–130. https://doi.org/10.1152/physiol.00027.2020

Fernandes GL, Orssatto LBR, Shield AJ, Trajano GS (2021) Runners with mid-portion Achilles tendinopathy have greater triceps surae intracortical inhibition than healthy controls. Scand J Med Sci Sports 32:1–18. https://doi.org/10.1111/sms.14111

Frančič A, Holobar A (2021) On the Reuse of Motor unit filters in high density surface electromyograms with different signal-to-noise ratios BT. In: Jarm T, Cvetkoska A, Mahnič-Kalamiza S, Miklavcic D (eds) 8th European medical and biological engineering conference. Springer International Publishing, Cham, pp 923–931

Gallina A, Hunt MA, Hodges PW, Garland SJ (2018) vastus lateralis motor unit firing rate is higher in women with patellofemoral pain. Arch Phys Med Rehabil 99(5):907–913. https://doi.org/10.1016/j.apmr.2018.01.019

Hug F, Tucker K (2017) Muscle coordination and the development of musculoskeletal disorders. Exerc Sport Sci Rev 45(4):201–208. https://doi.org/10.1249/JES.0000000000000122

Hug F, Del Vecchio A, Avrillon S, Farina D, Tucker KJ (2020) Muscles from the same muscle group do not necessarily share common drive: evidence from the human triceps surae. J Appl Physiol. https://doi.org/10.1152/japplphysiol.00635.2020

Hug F, Avrillon S, Del Vecchio A et al (2021) Analysis of motor unit spike trains estimated from high-density surface electromyography is highly reliable across operators. J Electromyogr Kinesiol 58:102548. https://doi.org/10.1016/j.jelekin.2021.102548

Hug XF, Vogel C, Tucker K et al (2022) Individuals have unique muscle activation signatures as revealed during gait and pedaling. J Appl Physiol 22:1165–1174. https://doi.org/10.1152/japplphysiol.01101.2018

Jakobi JM, Kuzyk SL, McNeil CJ, Dalton BH, Power GA (2020) Motor unit contributions to activation reduction and torque steadiness following active lengthening: a study of residual torque enhancement. J Neurophysiol 123(6):2209–2216. https://doi.org/10.1152/jn.00394.2019

Knobloch K (2016) Drug-induced tendon disorders. In: Ackermann PW, Hart DA (eds) Metabolic influences on risk for tendon disorders. Springer International Publishing, Cham, pp 229–238. https://doi.org/10.1007/978-3-319-33943-6_22

Lagas IF, Fokkema T, Bierma-Zeinstra SMA, Verhaar JAN, van Middelkoop M, de Vos RJ (2020) How many runners with new-onset Achilles tendinopathy develop persisting symptoms? A large prospective cohort study. Scand J Med Sci Sport 30(10):1939–1948. https://doi.org/10.1111/sms.13760

Lenth RV (2016) Least-squares means: The R package lsmeans. J Stat Softw 69(1):1–33. https://doi.org/10.18637/jss.v069.i01

Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P (2006) Intrinsic risk factors for the development of Achilles tendon overuse injury: a prospective study. Am J Sports Med 34(2):226–235. https://doi.org/10.1177/0363546505279918

Martin RL, Chimenti R, Cuddeford T et al (2018) Achilles pain, stiffness, and muscle power deficits: midportion Achilles tendinopathy revision 2018. J Orthop Sports Phys Ther 48(5):A1–A38. https://doi.org/10.2519/jospt.2018.0302

Martin RL, Chimenti R, Cuddeford T et al (2018) The VISA-A questionnaire: an index of the severity of Achilles tendinopathy I. J Orthop Sports Phys Ther 48(5):9–11

Martinez-Valdes E, Laine CM, Falla D, Mayer F, Farina D (2016) High-density surface electromyography provides reliable estimates of motor unit behavior. Clin Neurophysiol 127(6):2534–2541. https://doi.org/10.1016/j.clinph.2015.10.065

Martinez-Valdes E, Negro F, Falla D, De Nunzio AM, Farina D (2018) Surface electromyographic amplitude does not identify differences in neural drive to synergistic muscles. J Appl Physiol 124(4):1071–1079. https://doi.org/10.1152/japplphysiol.01115.2017

Martinez-Valdes E, Boccia G, Nawaz M, Negro F, Rainoldi A, Falla D (2019) Reduced knee-extensor torque steadiness and higher motor unit discharge rate variability in individuals with patellofemoral pain. Physiotherapy 105:e46. https://doi.org/10.1016/j.physio.2018.11.294

Masood T, Kalliokoski K, Bojsen-Møller J, Peter Magnusson S, Finni T (2014) Plantarflexor muscle function in healthy and chronic Achilles tendon pain subjects evaluated by the use of EMG and PET imaging. Clin Biomech 29(5):564–570. https://doi.org/10.1016/j.clinbiomech.2014.03.003

Mousavi SH, Hijmans JM, Rajabi R, Diercks R, Zwerver J, van der Worp H (2019) Kinematic risk factors for lower limb tendinopathy in distance runners: a systematic review and meta-analysis. Gait Posture 69(January):13–24. https://doi.org/10.1016/j.gaitpost.2019.01.011

Negro F, Holobar A, Farina D (2009) Fluctuations in isometric muscle force can be described by one linear projection of low-frequency components of motor unit discharge rates. J Physiol 587(24):5925–5938. https://doi.org/10.1113/jphysiol.2009.178509

Nuccio S, Del VA, Casolo A et al (2021) Deficit in knee extension strength following anterior cruciate ligament reconstruction is explained by a reduced neural drive to the vasti muscles. J Physiol. https://doi.org/10.1113/jp282014

Nunes JP, Costa BDV, Kassiano W et al (2020) Different foot positioning during calf training to induce portion-specific gastrocnemius muscle hypertrophy. J Strength Cond Res 34(8):2347–2351. https://doi.org/10.1519/JSC.0000000000003674

O'Neill S, Barry S, Watson P (2019) Plantarflexor strength and endurance deficits associated with mid-portion Achilles tendinopathy: the role of soleus. Phys Ther Sport 37:69–76. https://doi.org/10.1016/j.ptsp.2019.03.002

Orssatto LBR, Mackay K, Shield AJ, Sakugawa RL, Blazevich AJ, Trajano GS (2021) Estimates of persistent inward currents increase with the level of voluntary drive in low-threshold motor units of plantar flexor muscles. J Neurophysiol 125(5):1746–1754. https://doi.org/10.1152/jn.00697.2020

Rice DA, McNair PJ, Lewis GN, Mannion J (2015) Experimental knee pain impairs submaximal force steadiness in isometric, eccentric, and concentric muscle actions. Arthritis Res Ther 17(1):1–6. https://doi.org/10.1186/s13075-015-0768-1

Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J (2007) Full symptomatic recovery does not ensure full recovery of muscle-tendon function in patients with Achilles tendinopathy. Br J Sports Med 41(4):276–280. https://doi.org/10.1136/bjsm.2006.033464

Telianidis S, Perraton L, Clark RA, Pua YH, Fortin K, Bryant AL (2014) Diminished sub-maximal quadriceps force control in anterior cruciate ligament reconstructed patients is related to quadriceps and hamstring muscle dyskinesia. J Electromyogr Kinesiol 24(4):513–519. https://doi.org/10.1016/j.jelekin.2014.04.014

Vallance P, Crowley L, Hasani F, Malliaras P (2019) Maximal isometric force is more strongly associated with Achilles tendinopathy severity than rate of force development and muscle force steadiness. J Sci Med Sport 2019(22):S111–S112. https://doi.org/10.1016/j.jsams.2019.08.154