Foot position during plantarflexion changes *gastrocnemii* motor unit discharge rate in run-

ners with Achilles tendinopathy, but not in the way you might think.

Gabriel L. Fernandes[*1]; Lucas B. R. Orssatto[2]; Anthony J. Shield[1]; Gabriel S. Trajano[1]

[1] School of Exercise and Nutrition Sciences, Faculty of Health, Queensland University of Tech-

nology (QUT), Brisbane, Australia.

[2] Institute for Physical Activity and Nutrition (IPAN), School of Exercise and Nutrition Sci-

ences, Deakin University, Geelong, Australia

* Corresponding author: Gabriel L. Fernandes; email: gabriel.fernandes@hdr.qut.edu.au

https://orcid.org/0000-0002-1941-3900. School of Exercise and Nutrition Sciences, Faculty

of Health, Queensland University of Technology (QUT), Brisbane, Australia.

Authors' – ORCID

GLF – 0000-0002-1941-3900

LBRO – 0000-0003-3788-3700

AJS – 0000-0002-0393-2466

GST – 0000-0003-4667-4257

Abstract (207 words)

Runners with Achilles tendinopathy (AT) have lower neural drive to *gastrocnemius lateralis (GL)* and it has been shown that modified foot position during plantarflexion activity affects motor unit discharge properties of *GL*. This paper tested the motor unit (MU) responses of GL and *gastrocnemius medialis (GM)* to *feet-neutral* and *feet-in*. We recruited runners with (n=18) and without (n=19) AT for experiment 1(EXP1) and nine healthy individuals for experiment 2(EXP2). High-Density electromyography was used to analyse MU discharge rates (MUDR) for *GL* and *GM*, during an isometric plantarflexion (EXP1) with *feet-neutral* and *feet-in*; and during a 30%-peak torque (EXP2) using a dynamometer. In EXP1 MUDR were lower during *feet-in* in *GL* (p<0.001) and in *GM* (p<0.001), without group difference (p=0.84). Furthermore, cumulative spike train analysis showed no difference in *GL* or *GM* neural drive between group or condition. EXP2 showed no condition differences in *GL* (p=0.91)*; and lower MUDR in *GM* (p=0.01). Finally, the AT group have lower *triceps surae* endurance observed during single leg heel raise. Our data indicate that *feet-in* reduces *gastrocnemii* MUDR, compared to *feet-neutral*, irrespective of tendinopathy. Current understanding of modulation strategies to *GL* driven by feet position is conflicting; thus, plantarflexion using *feet in* may not be a viable treatment strategy for AT.

NEW & NOTEWORTHY:

Plantarflexion performed with feet inwards does not increase the neural drive to *gastrocnemius lateralis* as suggested in the literature. In fact, the *feet-in* condition reduced the motor unit discharge rates in both *gastrocnemii* during isometric plantarflexion and isometric plantarflexion using dynamometry. Therefore, this is not a viable treatment strategy for runners with Achilles tendinopathy. Furthermore, prioritising strategies to improve *triceps surae* endurance should be a key component in physiotherapy program.

Keywords

Achilles tendon; running; feet position; heel lifts; neural drive; triceps surae; high density electromyography; motor unit.

2. Introduction

Achilles tendinopathy, is a highly prevalent injury among runners[1,2], and is associated with deficits in *triceps surae* performance[3,4]. Runners with Achilles tendinopathy exhibit lower neural drive to *gastrocnemius lateralis*, resulting in a 28% lower contribution to submaximal triceps surae isometric force in acute presentations[5] (<3 months) and during dynamic tasks such as bilateral and unilateral heel drops[6]. This lower neural drive to *gastrocnemius lateralis* is also present in chronic Achilles tendinopathy[7] (> 3 months) evidenced by lower motor unit discharge rate during submaximal isometric contraction and seems to be specific to *gastrocnemius lateralis*; since there seems to be no differences in neural drive to *gastrocnemius medialis* and *soleus* compared to healthy controls [5–7]. In fact, *soleus* force contribution was greater than controls in some dynamic tasks[6], like walking, toe walking and unilateral heel drop. Individuals with Achilles tendinopathy seem to have an altered *triceps* surae muscle coordination strategy, favouring *soleus* over *gastrocnemius lateralis*. Therefore, these deficits in *gastrocnemius lateralis* neural drive observed in runners with Achilles tendinopathy[5,7] may negatively impact Achilles intratendinous load distribution; increasing tendon strain and potentially contribute to tendinopathy[8–10]. Therefore, developing rehabilitation strategies that aim to increase *gastrocnemius lateralis* activation and contribution to triceps surae force could be a promising approach to improve plantarflexor neuromuscular control and provide better rehabilitation outcomes for this population.

Recent evidence suggests that foot position during plantarflexion can independently influence *gastrocnemius medialis* and *gastrocnemius lateralis* recruitment and activation, and thereby alter the contribution of each muscle during plantarflexion tasks. Although a synergist muscle group during ankle plantarflexion, individual muscles of the *triceps surae* have partially independent neural drive[11] to allow for joint control and stability. Changes in foot position seems to influence not only the neural drive[11] but also resistance training-related hypertrophy

of the different gastrocnemii heads. These have been reported as an increase in the neural

drive[11] and greater hypertrophy[12] of *gastrocnemius lateralis* occurring when plantarflexion ex-

ercise was performed with the feet pointed inwards. Thus, current evidence suggests that the

contribution of *gastrocnemius lateralis* to plantarflexion can be increased by internally rotating

the hips and lower leg to orient the feet inwards.

This study aimed to investigate whether performing a bilateral isometric plantarflexion

task with feet pointed inwards, would increase the neural drive to *gastrocnemius lateralis* in

runners with Achilles tendinopathy. These data would inform the development of novel reha-

bilitation interventions, using different foot position during ankle plantarflexion. We hypothe-

sised that the feet pointed inwards (*feet in*) condition would increase motor unit mean discharge

rate of *gastrocnemius lateralis* in both Achilles tendinopathy and controls, compared to *feet*

*neutral (*condition was considered participant's comfortable stance with feet pointing forward).

We also aimed to explore changes in coefficient of variation of motor unit discharge rates, as

this could potentially increase for both muscles and groups during the isometric testing position

with *feet in* as this creates a more unstable position to hold a steady contraction. A second

experiment was conducted to better control variables such as balance during sustained testing

position and provide visual feedback to ensure muscle contraction remained constant during

the isometric task. The hypothesis was the same as for experiment 1: *feet in* condition would

increase motor unit mean discharge rate of *gastrocnemius lateralis* in both Achilles tendinopa-

thy and controls, compared to *feet neutral.*

2. Methods

This study compared the influence of two foot positions on *gastrocnemius lateralis and*

gastrocnemius medialis motor unit discharge frequencies during standing isometric contraction

of the plantarflexors. In experiment 1 we assessed runners with Achilles tendinopathy and

healthy counterparts during bilateral standing isometric contraction of the plantarflexors.

Thirty-seven endurance runners were recruited for this study, 18 runners with Achilles

tendinopathy (12 males (66.6%), 43.2 years old ±8.9, 165.5 cm ±38.5, 71.5 kg ±12.8); 13 with

mid-portion and 5 with insertional Achilles tendinopathy; and 19 healthy controls (10 males

(53%), 35.4 years old ±6.5, 172.1 cm ±11.3, 67.5 kg ±13.8). Volunteers were endurance

runners, recruited from local running clubs around Southeast Queensland, Australia, via email

and social media. The Achilles tendinopathy group had an average of 18.8 (±13.3) years of

running experience and a current running routine of 43.6 (±26.8) km/week; the control group

had 12.6 (±8.5) years of running experience and a current running routine of 42.2 (±29.5)

km/week. Diagnosis of Achilles tendinopathy was confirmed by an experienced

physiotherapist during examination if patients presented with localised Achilles tendon pain

for more than three months, pain provoked by physical activities in a dose dependent way and

had pain with palpation of the tendon. Average symptom presentation of Achilles tendinopathy

was 4.9 years (±6.3). Volunteers were excluded if they had previous rupture of or surgery to

the Achilles tendon; clinical findings indicating a differential diagnosis for the Achilles tendon

pain (such as tendon sheath inflammation, an acute tendon tear, plantaris or tibialis posterior

tendon involvement, neural involvement); 4) VISA-A score > 90 points for Achilles

tendinopathy group and < 100 for the control group; any other musculoskeletal injuries of the

lower limb of the affected leg; any neurological disorder; or mental health issues affecting

consent. All participants were free of cardiac, pulmonary, renal or endocrine[13] comorbidities.

Prior to testing, all participants read and signed a detailed informed consent document and

completed the VISA-A questionnaire[14]. The average VISA-A score for the Achilles

tendinopathy group was 65.5 (± 13.2) and 100 (0) for the control group. The Achilles

tendinopathy group also completed the Tampa scale of Kinesiophobia (TSK) to identify if

kinesiophobia was a factor influencing muscle performance[15,16].

The outcomes of our main experiment (experiment 1) were conflicting with the existing

literature[11]. Understanding the importance of addressing these inconsistencies, we conducted a

second experiment (experiment 2) to better understand the contrasting outcomes obtained in

experiment 1. Our primary objective of experiment 2 was to explore whether methodological

factors influenced our outcomes or if our observations simply deviate from current knowledge.

Therefore, we decided to replicate and expand our methods by controlling more variables; thus,

we conducted experiment 2 in a group of 9 healthy non-runners (5 males (55.6 %), 37.7 years

old ± 12.2, 171 cm ± 8.7, 71.1 kg ±11.1) to investigate the motor unit discharge characteristics

of *gastrocnemius lateralis and gastrocnemius medialis* in *feet in* and *feet neutral* positions

using a dynamometer and controlling better for body position. Volunteers were recruited from

around Southeast Queensland, Australia, via email and social media. Exclusion criteria were

the same as that of controls from the main experiment.

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 partially

conducted during the COVID-19 pandemic and all safety procedures followed local state

government policies.

2.1 Data collection and analysis

Experiment 1

Plantarflexor isometric peak torque was measured using an isokinetic dynamometer

(Biodex Medical Systems, Shirley, New York). For the bilateral Achilles tendinopathy

presentations (n=9), 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 which leg they

would use to kick a ball. Participants were seated (75 degrees of hip flexion) with their knee

straight and with the ankle joint at 10 degrees of plantarflexion. Warm up consisted of 2×4 s

isometric contractions of each participant's perceived 30, 50, 70 and 80% maximal voluntary

isometric contraction intensity. After warm-up, participants performed at least 3 maximal

voluntary isometric contractions, until <5% variation was observed between contractions; the

highest value was used. Participants then performed, 8 bilateral standing heel-raise, 4 in each

condition, *feet neutral* and *feet in*; in a randomised order. *Feet neutral* condition was considered

participant's comfortable stance with feet pointing forward; whereas for *feet in* condition,

participants would internally rotate at their hips so that the long axis of their feet were at 90-

degrees to each other[11]. All participants were able to achieve and stay comfortably in *feet in*

position for testing. Participants were instructed to perform a heel raise to full range followed

by a slow drop until their heels lightly touched a 6.5 cm height soft foam (supervised by tester)

and to hold when in position. Participants were also instructed to lightly touch a table placed

in front of them to reduce oscillation and assist balance. Once in position, a 20 second isometric

contraction was recorded. After all contractions were finalised, participants were given ~10

min rest, seated in a chair. Thereafter, a single leg heel raise (SLHR) endurance test was

performed, using a metronome set to 60 bpm[17,18] to control tempo. Participants were instructed

to go as high as possible and come down to fully touch their heel to the ground before going

up again. Test was ceased once performance was no longer correct (reduced plantarflexion

range, bending knees, hip hinge or other compensatory strategies or unable to follow pace, for

2 consecutive repetitions).

Experiment 2

The purpose of experiment 2 was to replicate experiment 1 while controlling certain

variables, not accounted for in experiment 1, that might have influenced our results. The set up

in experiment 2 allowed for control of each participant's relative force in each condition (*feet*

in and *feet neutral*) by utilising a percentage of participant's peak isometric plantarflexor

torque. This control was facilitated using visual feedback and torque trace. Body position was

also controlled, ensuring that *feet in* condition was based solely on medial hip rotation and

therefore, reducing a potential contribution of ankle passive stiffness on eversion torque.

Additionally, only healthy participants were recruited for this methodological experiment,

irrespective of their level of physical activity, as the primary focus was on methodology rather

than group comparisons. Dominant leg plantarflexor isometric peak torque was measured using

an isokinetic dynamometer (Biodex Medical Systems, Shirley, New York). Participants were

seated (75 degrees of hip flexion) with their knee straight and with the ankle joint at 0 degrees

of plantarflexion. Warm up consisted of 2×4 s isometric contractions of each participant's

perceived 30, 60 and 80% maximal voluntary isometric contraction intensity. After warm-up,

participants performed at least 3 maximal voluntary isometric contractions, until <5% variation

was observed between contractions; the highest value was considered. Thereafter, 25% of each

participant's peak isometric torque was calculated and used for the trapezoidal contractions.

Trapezoidal contractions consisted of a 6-second torque rise and decline with a 14-second

plateau, followed by 40 seconds rest between contractions. Participants received real-time

visual feedback of the trapezoidal pathway via a monitor placed ~1 m away from them and had

3 attempts to become familiar with task. Testing protocol involved 2 contractions for each

condition *foot neutral* and *foot in*, and for each muscle (8 contractions in total) in a randomised

order. *Foot neutral* consisted of a neutral position of the isokinetic foot plate and *foot in*

consisted of the same foot plate tilted, creating an angle of ~30 degrees medial rotation from

neutral.

High-density electromyograms recordings and analyses

HD-EMG (Sessantaquattro, OTBioelettronica, Torino, Italy) signals were recorded

from *gastrocnemius medialis* and *gastrocnemius lateralis* using OT Biolab+ software (version

1.3.0., OTBioelettronica, Torino, Italy) during isometric plantarflexion and during the

trapezoidal contractions. A b-mode ultrasound (ESAOTE, MyLabSeven. Genova, Italy) was

used to identify muscle fibre orientation and muscle boundaries, to guide electrode position.

To ensure good skin-electrode contact and conductance, a bi-adhesive layer with a conductive

paste was used. One 64-channel electrode grid (GR08MM1305, OTBioelettronica, Torino,

Italy) was placed over *gastrocnemius medialis* and one 64-channel electrode grid over

gastrocnemius lateralis. The ground strap electrode (WS2, OTBioelettronica, Torino, Italy)

was dampened and positioned around the ankle joint of the tested leg. EMG signals were

recorded in monopolar mode, amplified (256x), band-passed filtered (10-500Hz) and converted

to digital signal at 2048Hz by a 16-bit wireless amplifier (Sessantaquattro, OTBioelettronica,

Torino, Italy), before being stored for offline analysis. The grid adapter device used

(AD1x64SE, OTBioelettronica, Torino, Italy) has only 1 channel for the 64-channel electrode

connection, so each condition had to be performed twice, once with electrodes connected to

the *gastrocnemius medialis* and once with the electrodes connected to the *gastrocnemius*

lateralis, in randomised order. We decided to use a single 64-channel instead of one 32-channel

electrode in each muscle, based on observations from pilot testing and a previous study[7].

Although using 2×32 channels (ne electrode for each muscle) would have enabled us to

measure both muscles during the same contraction, we chose to use the 64-channel electrode

for several reasons. Firstly, we observed that the 32-channel electrode yielded a limited and

lower number of motor units, which is particularly important for *gastrocnemius lateralis* that

typically yields a low number of motor units. Additionally, the larger 64-channel electrode

offered a greater surface area, providing more comprehensive coverage of the muscle's

electrical activity. 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 with DEMUSE tool software (v.4.1;

The University of Maribor, Slovenia)[19]. All contractions were analysed, and motor units were

visually inspected (erroneous discharge times were excluded, and missed discharges included).

Manual inspection is required to reduce automatic decomposition discharge errors and improve

data[19,20]. Only motor units with a pulse-to-noise ratio (PNR) >30dB, sensitivity > 90%, were

used for data analysis[19,21]. The best contraction for each muscle, those with the smallest

deviations from trapezoidal target torques and highest PNRs, was used for analysis. After

identifying the best contraction for each muscle and condition, motor units were tracked

between conditions for each muscle for experiment 1. The operator for the motor unit analysis

and tracking was blinded by group. Tracking motor units across conditions allows a better

understanding of the effect of foot position on the discharge patterns of the same motor unit

and therefore, the influence of each condition on the neural drive to each muscle. Motor units

that discharged intermittently during the 20 second isometric plateau were excluded from the

tracked analysis. Individuals who did not yield motor units after tracking were not included in

statistical analysis of said muscle. Individual motor unit mean discharge rates for each muscle

are also reported.

Due to the lower number of participants, we decided to only use the analysis of individual

motor units in experiment 2 to limit the number of motor unit losses in the tracking analysis.

We compared the results from experiment 2 with those from the main experiment.

The sum of individual motor unit discharge times, the cumulative spike train, represents an

estimation of the neural drive to a muscle during a contraction[11,22–24]. The cumulative spike

train of the individual motor units was compared for each muscle, between conditions (*feet*

neutral and *feet in*) and groups. The analysis for the cumulative spike train was calculated using

the 20 second isometric plateau, using MATLAB (TheMathWorks, Natick, MA, USA). The

binary arrays representing the number of discharge times was summed then smoothed using a

Hanning window of 400-ms duration[11,24,25]. The mean cumulative spike train was normalised

by the total number of motor units, representing an index of neural drive[26], this is referred to

in the manuscript as normalised neural drive.

2.2 Statistical analysis

All analyses were performed using R studio (version 1.3.1093). Models were fitted

using the *lme4 and lmerTest* [27] and *flexplot* package[28]. Separate linear mixed-effect models

were used to compare motor unit discharge rates and coefficients of variation of motor unit

discharge rates from tracked motor units from *gastrocnemius medialis* and *gastrocnemius*

lateralis between conditions (*feet neutral* and *feet in*) and groups (Achilles tendinopathy and

control), and for individual (non-tracked) motor units for each condition and muscle. The

models were tested using participant ID as random intercept to account for the correlation

between repeated observations for each participant. For the tracked motor unit analysis, motor

unit ID was also included in the model as random intercept to estimate individual change in

neural drive in the same motor unit, influenced by condition. A linear mixed model was also

used to compare the normalised neural drive between conditions and between groups. Analysis

of covariance (ANCOVA) was used to compare peak isometric plantarflexor torque between

groups, using body mass as covariate and a Welch two sample t-test was use for single leg heel

raise (SLHR) analysis. Furthermore, a Pearson's r was used to test a possible correlation

between the SLHR values and the kinesiophobia scores of each participant. The estimated

marginal mean difference and 95% confidence intervals (CI) for motor unit mean discharge

rate between groups, were determined using the *emmeans* package[29]. Normality assumptions

were confirmed by analysis of the histogram of residuals (Q-Q Plot). Sensitivity tests were

utilised in some cases to assess the influence of outliers identified by the models in the output,

and these are reported in the results section. For the t-test and ANCOVA, a Bonferroni

correction was used when relevant. Statistical significance was accepted at $P \leq 0.05$ for all

tests. Cohen's d effect size is presented for the t test. Data is presented as mean (± SD) or mean

(95% confidence interval lower to upper limits).

3. Results

3.1 Motor unit identification

We found a total of 1944 motor units in Experiment 1, 1082 motor units in the Achilles

tendinopathy group (15.0 (± 10.6) per participant) and 862 motor units in the control group,

(11.3 (± 11.8) per participant) across the two muscles and conditions. After motor unit tracking

across conditions, there were a total of 560 motor units; 305 (8.5 (± 9.1) per participant) in the

Achilles tendinopathy group and 255 motor units (6.7 (± 9.3) per participant) in the control

group, across the two muscles. In Experiment 2, we found a total of 369 motor units across the

two muscles. The total number, mean and standard deviation (± SD) of identified individual

and tracked motor units, per muscle, group and condition are reported in Table 1.

Table 1. Total number, mean (± SD) of identified individual and tracked motor units (MU) per group, muscle, and condition

Achilles tendinopathy	Mean (± SD)	n	Total number of MU
Tracked motor units			
Gastrocnemius lateralis	6.2 (5.6)	10	68
Gastrocnemius medialis	14.8 (9.0)	16	237
Individual motor units			
Gastrocnemius lateralis			
Feet neutral	11.7 (7.1)	17	198
Feet in	10.2 (9.0)	17	173
Gastrocnemius medialis			
Feet neutral	20.1 (10.4)	17	342
Feet in	21.6 (9.3)	17	367
Cumulative spike train			
Gastrocnemius lateralis			
Feet neutral	11.8 (6.9)	17	200
Feet in	10.3 (8.7)	17	175
Gastrocnemius medialis			
Feet neutral	20.3 (10.2)	17	345

Feet in	21.7 (9.0)	17	368
Control			
Tracked motor units			
Gastrocnemius lateralis	6.9 (6.5)	11	76
Gastrocnemius medialis	11.9 (11.2)	15	179
Individual motor units			
Gastrocnemius lateralis			
Feet neutral	8.6 (8.6)	15	129
Feet in	8.2 (8.4)	13	123
Gastrocnemius medialis			
Feet neutral	16.6 (12.7)	18	281
Feet in	17.9 (13.6)	19	313
Cumulative spike train			
Gastrocnemius lateralis			
Feet neutral	8.7 (8.3)	15	131
Feet in	9.5 (8.0)	13	123
Gastrocnemius medialis			
Feet neutral	15.1 (12.3)	18	282
Feet in	15.8 (13.7)	19	317
Experiment 2			
Gastrocnemius lateralis			
Feet neutral	4.8 (3.7)	8	38
Feet in	6.5 (4.8)	8	52
Gastrocnemius medialis			
Feet neutral	14.8 (8.5)	9	133
Feet in	16.2 (10.8)	9	146

This table shows mean MU used in each condition for statistical analysis, the number of participants (n) who yield MU and total number of MU found per muscle and condition.

3.2 Motor Unit discharge rate

3.2.1 Tracked motor unit analysis

The analysis of *gastrocnemius lateralis* motor unit mean discharge rate was different

between conditions (F=19.73, p<0.001) but not between groups (F=0.04, p=0.84), nor was

there a condition × group interaction (F=0.04, p=0.84). Discharge rate was lower during *feet in*

with mean difference between conditions of -0.57 (-0.8 to -0.3), with mean discharges rate for

feet in of 10.3 pps (9.6 to 11.0) and 10.9 pps (10.2 to 11.5) for *feet neutral* (Figure 1). Individual

group means for each motor unit analysis are reported in Table 2.

Figure 1
Gastrocnemius lateralis motor unit discharge with *feet neutral* and *feet in* between groups
during the tracked motor unit analysis. Figure **A.** Each dot represents a single motor unit data
point, coloured by participants. Figure **B.** represents the mean motor unit data for each partic-
ipant and the light gray line connecting same participant's response across conditions. Mean
and 95% confidence interval are offset to the left to facilitate visualisation. pps = pulse per
second. AT = Achilles tendinopathy

Table 2. Group mean for motor unit discharge rate in the tracked and individual analysis, Coefficient of variation
(Cov) of motor unit discharge rate and normalised neural drive.

Achilles tendinopathy	Mean (95% CI)	Control	Mean (95% CI)
Tracked motor units		**Tracked motor units**	
Gastrocnemius lateralis		*Gastrocnemius lateralis*	
Feet neutral	10.5 pps (10.1 to 10.9)	Feet neutral	11.2 pps (10.7 to 11.6)
Feet in	9.8 pps (9.3 to 10.3)	Feet in	10.6 pps (10.2 to 11.0)
Gastrocnemius medialis		*Gastrocnemius medialis*	
Feet neutral	10.9 pps (10.2 to 11.6)	Feet neutral	11.3 pps (10.6 to 12.1)
Feet in	10.0 pps (9.3 to 10.7)	Feet in	10.8pps (10.0 to 11.5)
Cov of motor unit discharge rate		**Cov of motor unit discharge rate**	
Gastrocnemius lateralis		*Gastrocnemius lateralis*	
Feet neutral	11.9 (11.0 to 12.8)	Feet neutral	14.2 (13.1 to 15.3)
Feet in	14.8 (13.8 to 15.8)	Feet in	15.5 (14.4 to 16.6)
Gastrocnemius medialis		*Gastrocnemius medialis*	
Feet neutral	11.7 (11.2 to 12.2)	Feet neutral	13.0 (12.1 to 13.9)
Feet in	13.3 (12.7 to 13.8)	Feet in	14.2 (13.4 to 15.0)
Individual motor units		**Individual motor units**	
Gastrocnemius lateralis		*Gastrocnemius lateralis*	
Feet neutral	10.4 pps (10.1 to 10.6)	Feet neutral	10.4 pps (10.0 to 10.7)
Feet in	9.9 pps (9.6 to 10.1)	Feet in	10.6 pps (10.2 to 10.9)
Gastrocnemius medialis		*Gastrocnemius medialis*	
Feet neutral	10.2 pps (9.9 to 10.4)	Feet neutral	10.7 pps (10.4 to 10.9)
Feet in	9.8 pps (9.6 to 9.9)	Feet in	10.4pps (10.1 to 10.6)
Normalised neural drive		**Normalised neural drive**	
Gastrocnemius lateralis		*Gastrocnemius lateralis*	
Feet neutral	0.77 (0.73 to 0.81)	Feet neutral	0.78 (0.70 to 0.86)
Feet in	0.88 (0.78 to 0.98)	Feet in	0.79 (0.74 to 0.84)
Gastrocnemius medialis		*Gastrocnemius medialis*	
Feet neutral	0.74 (0.71 to 0.77)	Feet neutral	0.79 (0.75 to 0.83)
Feet in	0.76 (0.74 to 0.78)	Feet in	0.77 (0.73 to 0.81)

This table shows group mean for motor unit discharge rates for tracked and individual analyses, coefficient of variation of motor unit discharge rate and the normalised neural drive. Data is reported as mean (95% CI lower to upper limits).

The analysis of *gastrocnemius medialis* motor unit mean discharge rate showed a condition $\times$ group interaction. However, this interaction was of no relevance to our hypothesis, as post-hoc testing detected a statistically significant difference only between *feet in* values from one group with *feet neutral* values from the other group; *feet in* AT $\times$ *feet neutral* Controls, -1.32 pps (-2.3 to -0.34, p=0.05). Motor unit mean discharge rate was different between conditions (F=226.69, p<0.001) but not between groups (F=1.41, p=0.24). Mean discharge rate

was lower for *feet in* with a mean difference of -0.73pps (-0.79 to -0.63); mean discharge rate

for *feet in* was 11.1pps (10.6 to 11.6) and 10.4pps (9.88 to 10.9) for *feet neutral* (Figure 2).

Figure 2
Gastrocnemius medialis motor unit discharge with *feet neutral* and *feet in* between groups
during the tracked motor unit analysis. Figure **A.** Each dot represents a single motor unit data
point, coloured by participants. Figure **B**. represents the mean motor unit data for each partic-
ipant and the light gray line connecting same participant's response across conditions. Mean
and 95% confidence interval are offset to the left to facilitate visualisation. pps = pulse per
second. AT = Achilles tendinopathy
330

We also analysed the coefficient of variation of motor unit discharge rates for each

condition. *Gastrocnemius lateralis* analysis showed a condition difference (F=52.10, p<001),

with no differences between groups (F=0.03, p=0.86). Both groups had greater variation of

motor unit discharge rate during *feet in* with a mean difference of 2.2 (1.6 to 2.8); mean

variation of discharge rate for *feet in* was 15.7 (14.4 to 17.0) and 13.5 (12.2 to 14.8) for *feet*

neutral. (Figure 3).

Figure 3
Gastrocnemius lateralis coefficient of variation (Cov) of motor unit discharge rate with *feet*
neutral and *feet in* between groups during the tracked motor unit analysis. Each dot repre-
sents a single motor unit data point, coloured by participants. Mean and 95% confidence in-
terval are offset to the left to facilitate visualisation. AT = Achilles tendinopathy
343

In *gastrocnemius medialis* analysis we only found a difference between conditions

(F=67.50, p<0.001), with no difference between groups (F=1.90, p=0.18) and no condition ×

group interaction (F= 1.70, p=0.19). Similarly to what we observed in *gastrocnemius lateralis*,

both groups had greater variation of discharge rate during *feet in*, with a mean difference of 1.4

(1.1 to 1.7); mean variation of discharge rate for f*eet in* was 14.2 (13.1 to 15.2) and 12.7 (11.7

to 13.7) for *feet neutral*. (Figure 4).

Figure 4
Gastrocnemius medialis coefficient of variation (Cov) of motor unit discharge rate with *feet neutral* and *feet in* between groups during the tracked motor unit analysis. 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. AT = Achilles tendinopathy

3.2.1 Non-tracked motor unit analysis

Analysis of *gastrocnemius lateralis* motor unit mean discharge rate showed no difference between conditions (F=1.46, p=0.22), or groups (F=0.08, p=0.77) and no condition × group interaction (F=0.95, p=0.32).

 (Figure 5 A).

Gastrocnemius medialis, motor unit analysis on the other hand; showed similar results to the tracked motor unit analysis, demonstrating difference between conditions (F= 18.28, p<001) but no differences between groups (F=2.30, p=0.13) and no condition × group interaction (F= 0.40, p=0.55). Discharge rate was lower during *feet in* with a mean difference between conditions of -0.38 pps (-0.5 to -0.2), mean discharge rate was 10.4 pps (9.9 to 10.9) for *feet in* and 10.8 pps (10.3 to 11.3) for *feet neutral* (Figure 5 B).

3.2.2 Non-tracked motor unit analysis – Experiment 2

Similarly to the results found in experiment 1, *gastrocnemius lateralis* motor unit mean

discharge rate showed no difference between condition (F=0.01, p=0.91). Mean difference

between conditions was 0.03 pps (-0.51 to 0.57), with mean discharge rate of 8.37 pps (7.49 to

9.24) for *feet in* and 8.34 pps (7.49 to 9.2) for *feet neutral.*

Also in agreement with the results obtained from experiment 1, *gastrocnemius medialis*

motor unit analysis showed a difference between conditions (F= 6.30, p=0.01). Mean discharge

rate for *feet in* was 9.21 pps (8.37 to 10.0) and 9.65 pps (8.81 to 10.5) with *feet neutral (m*ean

difference between conditions; -0.44 (-0.78 to -0.09)).

A.

B.

Figure 5
Motor unit discharge with *feet neutral* and *feet in* between groups during the individual motor
unit analysis. Each dot represents a single motor unit data point, coloured by participants.
Figure **A.** represents data from *gastrocnemius lateralis*; figure **B.** displays data from *gas-trocnemius medialis*. Mean and 95% confidence interval are offset to the left to facilitate vis-ualisation. pps = pulse per second. AT = Achilles tendinopathy

3.3 Cumulative Spike Train analysis (normalised neural drive)

Cumulative Spike Train analysis for *gastrocnemius lateralis* showed no differences between

conditions (F=3.30, p=0.07), or groups (F=0.28, p=0.59) and no condition × group interaction

(F= 1.48, p=0.23), (Figure 6 A).

Cumulative Spike Train analysis for the *gastrocnemius medialis* also showed no differences

between conditions (F=0.03, p=0.85), or groups (F=2.35, p=0.13) and no condition × group

interaction (F= 1.95, p=0.17), (Figure 6 B).

A.

B.

Figure 6
Cumulative spike train analysis during feet positioned inwards and feet neutral between
groups during the individual motor unit analysis. Cumulative sum was normalised by the
number of motor units used in each participant's analysis (normalised neural drive). Each dot
represents data point by participants. Figure **A.** represents data from *gastrocnemius lateralis*;
figure **B.** displays data from *gastrocnemius medialis*. Mean and 95% confidence interval are
offset to the left to facilitate visualisation. pps = pulse per second. AT = Achilles tendinopa-
thy

3.4 Peak torque and SLHR

We found no differences between groups for peak isometric torque (F=2.77, p=0.10). Peak

plantar flexor isometric torque in the Achilles tendinopathy group was 137 N·m (126 to 148)

and 131 N·m (120 to 141) in the control group. Data for the SLHR was not normally distributed

in the control group due to the presence of outliers. However, after running a sensitivity test,

we still found similar statistical results and effect size on both analyses. The Achilles

tendinopathy group performed on average -13.2 (24.5 to 1.82) SLHR (p=0.024, d=-0.77, large

effect size[30]) than controls when tested with outliers. When outliers were removed, the Achilles

tendinopathy group performed about 34 (29 to 39) repetitions, 7.3 (-13.9 to -0.75) fewer SLHR

(p=0.03, d=-0.78) than controls 41 (36 to 45) (Figure 7).

Kinesiophobia values for the Achilles tendinopathy group was 32.1 (±6.5) and there was no

correlation between levels of kinesiophobia and SLHR values, r = 0.1, p=0.69.

Figure 7
Single leg heel raise (SLHR) data is coloured by participant. Mean and 95% confidence interval are offset to the left to facilitate visualisation. AT = Achilles tendinopathy

4. Discussion

4.1. Main findings

We investigated the effect of different foot position during bilateral isometric plantarflexion in *gastrocnemius lateralis and medialis* neural drive, in runners with and without chronic Achilles tendinopathy. We analysed motor unit discharge rates of *gastrocnemius lateralis* and *gastrocnemius medialis* individual responses and tracked across conditions and

compared them between groups and conditions (*feet neutral*, *feet in*). We also looked at the

coefficient of variation of motor unit discharge rate of *gastrocnemius lateralis* and *gastrocnem-*

ius medialis as well as the normalised neural drive to each muscle during the two conditions

and between groups.

Contrary to what is described in the literature, we found that bilateral plantarflexion with feet

pointed inwards reduced both *gastrocnemii* motor unit discharge rate, instead of increasing it.

These inconsistencies between the outcomes from our main experiment and the available evi-

dence motivated us to conduct a second experiment. In experiment 2, we expanded our testing

protocol to control more variables, as explained in detail in the methods section. This time, we

recruited a different subgroup of healthy individuals and utilised a dynamometer for the plantar

flexor isometric tasks using the same two conditions (*feet in* and *feet neutral)*. Despite these

modifications in set up, our results remained consistent with those of experiment 1, and their

implications are discussed in the subsequent paragraphs.

We found no effect of Achilles tendinopathy in *gastrocnemii* motor unit discharge rates during

isometric plantarflexion. In our main experiment in the tracked analysis, plantarflexion with

feet in reduced the motor units discharge rates of both *gastrocnemius lateralis* and *gastrocnem-*

ius medialis compared to *feet neutral*, but in the non-tracked analysis, *feet in* reduced motor

unit discharge rate only in *gastrocnemius medialis* discharge rate. These changes in discharge

rate derived from the different foot position occurred without any changes in neural drive.

Through the tracked analysis we can observe the discharge responses of motor units

and gain valuable insight into how each condition affected their discharge frequencies.

Contrary to our initial hypothesis, the tracked motor unit analysis revealed that, rather than

increasing motor unit discharge rate, turning the *feet in* reduced motor unit firing for both

gastrocnemius lateralis and *gastrocnemius medialis* compared to *feet neutral*. This effect was

observed in participants with and without Achilles tendinopathy. Alongside that, the coefficient

of variation of motor unit discharge rate in both *gastrocnemius lateralis* and *medialis* increased

with *feet in* compared to *feet neutral*, suggesting a higher degree of variability in motor unit

discharge frequency in this position. Coefficient of variation of motor unit discharge rate is

related to force output during submaximal isometric contractions and this increase in the

discharge variability can partially explain the reduction in muscle performance[31]. It is possible

that the sustained plantarflexion position with *feet in* creates a more unstable position, leading

to greater variability in motor unit discharge frequency during the 20-second contraction.

Greater instability can stimulate inhibitory proprioceptive feedback from muscle spindles and

Golgi tendon organs, which may impact motor unit behaviour in various ways[32,33], including

reducing motor unit discharge rate. In addition, it is possible that this increased joint stretch

created by the testing position *feet in*, may increase the contribution of passive joint structures

(stretch-sensitive afferent receptors in ligaments) in conjunction with the ankle muscles to

increase joint stiffness and provide better support and joint stability[33–35]. These afferent

receptors from the ankle ligaments may assist with the adjustment of the activity of other ankle

stabilizers during plantarflexion such as the *soleus* and the *fibularis longus*[36,37]. S*oleus* and

fibularis longus can work as ankle stabilisers and are highly responsive to sudden change in

body position[36,37]; however, this is only speculation as these muscles were not monitored

during testing.

      In experiment 2 we implemented measures to address all the factors we thought could

have influenced our results. We controlled participant's stability during testing position to

minimise the influence of motor unit discharge variability which could have been influenced

by a more unstable base of support in the *feet in* position. By using a seated and stable position,

we could better control and test the hypothesis of inhibitory proprioceptive stimuli influencing

our experiment 1 results. To account for the possible increased contribution of passive joint

structures and reduced plantarflexor demand to maintain isometric testing position, we used

visual trace feedback to ensure participants maintained isometric torque throughout the

contraction. Despite implementing these controls in experiment 2, our results yielded similar

findings as in experiment 1.

The control of muscle force occurs as a modulation of the rate of active motor unit

discharge and the recruitment of new motor units[38]. The tracked motor unit analysis excludes

motor units identified in only one condition. Conversely, the analysis of non-tracked motor

units, considers all identified motor units in both conditions, and thus includes a different

sample of motor units, making it also a relevant analysis. When considering all identified motor

units our results for *gastrocnemius lateralis* were different, showing no difference in motor

unit discharge rates between conditions. On the other hand, g*astrocnemius medialis* had lower

motor unit firing rate during *feet in*, which was a similar response to that observed in the tracked

motor unit analysis. The analysis using all identified motor units, is similar to that by Hug and

colleagues[11] in non-runners; however, their results differ from ours. Specifically, Hug et al.

(2020), found higher motor unit discharge rate in *gastrocnemius lateralis* during *feet in* but no

difference in *gastrocnemius medialis*. It is important to note that our study had more than

double the number of participants and a larger overall sample of motor units for analysis. In

fact, we had twice the amount of motor units for *gastrocnemius lateralis*. Additionally, in our

study, both groups showed similar responses for between condition and muscle analysis. It is

also important to acknowledge that Hug et al. (2020) had the advantage to monitor both

muscles in the same contractions, which is a strength of their study in comparison to ours.

However, our study highlights a significant finding, indicating that *gastrocnemius lateralis*

motor unit discharge rate during isometric standing plantarflexion with different foot positions

displays a high variability amongst individuals, irrespective of the presence of tendinopathy.

This can be observed in Figure 1B where mean motor unit discharge rate by participant is

showed across conditions. In both AT and control groups there were varied responses in motor

unit mean discharge rates between conditions in *gastrocnemius lateralis*, with some individuals

displaying higher and others lower discharge rates when their feet were internally rotated.

Perhaps, the sample assessed in Hug's study[11] had a preferential response to increase motor

unit discharge rates in *gastrocnemius lateralis*. *Gastrocnemius medialis* mean individual

responses to change in feet position, Figure 1B, had lower discharge rate with *feet in* across the

board in both groups. It is worth noting that testing position for *feet in* and *feet neutral*

conditions in our experiment 1 was the same as described in Hug et al. A previous study[7] from

our group observed different responses in motor unit discharge rates in *gastrocnemius lateralis*

in runners with Achilles tendinopathy during the increase in torque intensity, suggesting a

lower neural drive to *gastrocnemius lateralis*, and this was not observed in the present study.

However, in this previous study[7] we measured motor unit characteristics of the triceps surae

during seated unilateral isometric plantar flexion contraction using a dynamometer and a torque

isometric plantarflexion. This time we were unable to measure torque during the isometric task.

Consequently, we cannot make comparisons between these results and those from the other

study that used a different set up.

The Achilles tendon is composed of the *soleus*, *gastrocnemius medialis* and

gastrocnemius lateralis sub-tendons intertwined together[5,39,40]. These sub-tendons are partially

independent and experience heterogeneous strains during movement[5,41,42] A study[39] compared

the effect of feet position in *gastrocnemius medialis* and *gastrocnemius lateralis* sub-tendon`s

distal movement and elongation during ankle plantar flexion in prone position. The authors

observed that a horizontal change in feet position (feet in and feet out) affects the Achilles

tendon motion, and that different foot positions affect individual sub-tendons of the *triceps*

surae differently. During submaximal isometric actions with *feet in*, the *gastrocnemius*

lateralis sub-tendon lengthened more than that of the *gastrocnemius medialis* in the same

position and vice-versa. However, the authors found that during a 20 and 40% maximal

voluntary contraction with *feet in*, there was no difference between *gastrocnemius medialis* and

gastrocnemius lateralis muscle electrical activity (measured using root mean square

electromyography- RMS EMG)[39]. This aligns with the results of our experiment 2 for

gastrocnemius lateralis; where we used a similar submaximal isometric value of 25%.

Furthermore, Nunes et al[12] found greater hypertrophy in *gastrocnemius lateralis* after a period

of resistance training using a leg-press machine using feet positioned inwards. The seated set

up in the leg-press, the added resistance and the dynamic contraction of the plantar flexors may

have induced a different response from the *triceps surae* to what we found in our study. This

could be attributed to the higher strain created in the individual subtendons and muscle fibres

during plantarflexion in the leg-press machine. Perhaps the lower demand on the plantar flexors

from our body weight isometric plantar flexion, or 25% isometrics was not enough to change

the discharge behaviour of the *gastrocnemii* motor units.

Additionally, to better understand the effect different feet positions have on the neural

drive to these muscles, we tested motor units discharge characteristics of the *gastrocnemius*

lateralis and *gastrocnemius medialis* using another analysis, the cumulative spike train analysis

(normalised neural drive)[23,26]. Cumulative spike train analysis gives a reliable and strong

indication of the neural drive to a muscle[11,22–24] as it represents a sum of all identified motor

unit discharges during the contractions. For the cumulative spike train analysis we included all

identified motor units, including ones that discharged intermittently during the 20 second

isometric plateau (these motor unit were excluded from the tracked analysis). This phenomenon

of intermittent discharges, is referred to as motor unit rotation[43], and it is believed to represent

a normal modulation strategy used by the central nervous system. The central nervous system

uses different strategies to adjust for force requirements such as modifying the discharge rates

or the recruitment of motor units. Such changes may occur within the same level of neural

drive[43]. The normalised neural drive represents the summation of all motor unit discharges

during the contraction, reflecting the level of neural drive. The absence of change we observed

in the normalised neural drive between the two conditions demonstrates that the change in feet

position during standing plantarflexion from *feet neutral* vs *feet in* had no effect in the neural

drive to the *gastrocnemii*.

Emerging evidence from studies of both the triceps surae and the quadriceps suggests

that individuals exhibit a highly variable and unique muscle activation patterns[44] during

activities such as cycling and walking, and during isometric contractions in the triceps surae[45]

and the quadriceps[46]. We also observed high inter-individual variability in participant

responses, in particular for *gastrocnemius lateralis* motor unit discharge rates when comparing

internally rotated and neutrally orientated foot positions.

Isometric peak torque was not different between groups, which aligns with other

studies[7,47]. It becomes clear that plantar flexor isometric peak torque is not lower in runners

with chronic Achilles tendinopathy. Conversely, an important muscle deficit observed in this

group, which also seems consistent across studies, is plantar flexor endurance[48–51]. We

measured plantar flexor endurance using a single leg heel raise to failure (SLHR) test, paced

with a metronome[52]. The Achilles tendinopathy group performed 18% fewer repetitions to

failure than controls. This finding is similar to an earlier study[50] from our group where we

found 28% less repetitions during SLHR in the Achilles tendinopathy.

The kinesiophobia scores for the Achilles tendinopathy group represents moderate

kinesiophobia[16]; however our participants still performed a substantial number of SLHRs,

without any Achilles tendon pain during testing. These results are similar to values reported in

a subgroup of active individuals with Achilles tendinopathy[53]. Furthermore, there was no

correlation between kinesiophobia values and SLHR performance Therefore, it is unlikely that

kinesiophobia is a feature influencing muscle endurance in our sample of runners.

4.2 Strengths and Limitations

We tested the two *gastrocnemii* muscles in one of the most used exercises for treatment

of Achilles tendinopathy, the standing heel raise, and tested a position variation suggested in

the literature to increase the neural drive to *gastrocnemius lateralis*. However, we found that

such change is highly variable between individuals regardless of whether they have Achilles

tendinopathy. In some instances, turning the feet inward markedly reduced mean motor unit

discharge rate to both muscles. This modification should not be used as rehabilitation strategy

aiming to improve activation to *gastrocnemius lateralis* as the *feet in* position may have more

variable effects than previous findings have suggested. In fact, when considering the objective

of adopting *feet in,* which was to increase the discharge rate of *gastrocnemius lateralis*, it had

the opposite effect in some participants.

We were unable to test both muscles during the same contractions, as the EMG device

used was limited to up to 1×64-channel adaptor. During pilot testing using one 32-channel

electrode in each muscle to record both muscles together did not yield sufficient motor units

for analysis and several participants had no detectable motor unit firings in *gastrocnemius*

lateralis. Another limitation was not testing the *soleus*. Future studies might consider using

devices that allow recording of all three muscles of the triceps surae during the same

contractions. A between muscle coherence analysis would be another valuable method when

estimating the magnitude of neural drive and the contribution of individual muscles of the

triceps surae to force production in runners with Achilles tendinopathy.

Our study helps to reinforce findings from the literature, showing that deficits in triceps

surae endurance are an issue in runners with Achilles tendinopathy. Such deficits can be

identified with a simple SLHR test that can be easily performed in a clinical setting.

Furthermore, normative data for SLHR should be developed for both recreational and

competitive runners as this would serve as benchmarks to assess runners with Achilles

tendinopathy.

5. Conclusion

Our paper was the first to investigate the effect different feet position has on motor unit

discharge characteristics of *gastrocnemius lateralis* and *medialis* in runners with and without

Achilles tendinopathy. Inward rotation of the feet reduced the neural drive to both muscles,

which is the opposite of what was previously published in the literature using the same

protocol. Therefore, it remains uncertain if performing plantarflexion exercise with feet pointed

in would provide any additional benefits to the treatment of runners with Achilles tendinopathy.

Further understanding of the mechanisms behind neuromodulation strategies to the triceps

surae during plantar flexion driven by modified feet position needs to be investigated further.

It is worth noting that runners with Achilles tendinopathy have low triceps surae endurance

without deficits in peak isometric torque. Thus, muscle endurance deficits should be addressed

during rehabilitation for this group.

Conflict of interests

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

Data availability

Data and R code are available at https://github.com/GabeFernandess/feet_AT

Acknowledgments

The authors declare not having received 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 conducted the experiments. GLF analysed the

data, conducted the statistical analysis in R and drafted the first version of the manuscript. GLF

and LBRO developed a MATLAB script for motor unit discharge rate, coefficient of variation

of motor unit discharge rate and cumulative spike train analysis. All authors critically revised

the manuscript and provided valuable feedback. All the authors read and approved this final

version of the manuscript.

References

1.   Lagas IF, Fokkema T, Bierma-Zeinstra SMA, Verhaar JAN, van Middelkoop M, de

Vos RJ. How many runners with new-onset Achilles tendinopathy develop persisting

symptoms? A large prospective cohort study. *Scand J Med Sci Sport*.

2020;30(10):1939-1948. doi:10.1111/sms.13760

2.   Mousavi SH, Hijmans JM, Rajabi R, Diercks R, Zwerver J, van der Worp H.

Kinematic risk factors for lower limb tendinopathy in distance runners: A systematic

review and meta-analysis. *Gait Posture*. 2019;69(January):13-24.

doi:10.1016/j.gaitpost.2019.01.011

3.   Silbernagel KG, Gustavsson A, Thomeé R, Karlsson J. Evaluation of lower leg

function in patients with Achilles tendinopathy. *Knee Surgery, Sport Traumatol*

Arthrosc. 2006;14(11):1207-1217. doi:10.1007/s00167-006-0150-6

4.   Sancho I, Malliaras P, Barton C, Willy RW, Morrissey D. Biomechanical alterations in

individuals with Achilles tendinopathy during running and hopping: A systematic

review with meta-analysis. *Gait Posture*. 2019;73(June):189-201.

doi:10.1016/j.gaitpost.2019.07.121

5.   Crouzier M, Tucker K, Lacourpaille L, et al. *Force-Sharing within the Triceps Surae:*

An Achilles Heel in Achilles Tendinopathy. Vol 52.; 2020.

doi:10.1249/MSS.0000000000002229

6.   Mylle I, Crouzier M, Vanwanseele B. Triceps surae muscle forces during dynamic

exercises in patients with Achilles tendinopathy : A cross- - sectional study.

2023;(May):1-11. doi:10.1111/sms.14444

7.   Fernandes GL, Orssatto LBR, Sakugawa RL, Trajano GS. Lower motor unit discharge

rates in gastrocnemius lateralis, but not in gastrocnemius medialis or soleus, in runners

with Achilles tendinopathy: a pilot study. *Eur J Appl Physiol*. Published online

November 23, 2022:2022.05.05.22274750. doi:10.1007/s00421-022-05089-w

8.   Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain

the clinical presentation of load-induced tendinopathy. *Br J Sports Med*.

2009;43(6):409-416. doi:10.1136/bjsm.2008.051193

9.   Bojsen-Møller J, Hansen P, Aagaard P, Svantesson U, Kjaer M, Magnusson SP.

Differential displacement of the human soleus and medial gastrocnemius aponeuroses

during isometric plantar flexor contractions in vivo. *J Appl Physiol*. 2004;97(5):1908-

1914. doi:10.1152/japplphysiol.00084.2004

10.   Hug F, Tucker K. Muscle Coordination and the Development of Musculoskeletal

Disorders. *Exerc Sport Sci Rev*. 2017;45(4):201-208.

doi:10.1249/JES.0000000000000122

11.   Hug F, Del Vecchio A, Avrillon S, Farina D, Tucker KJ. Muscles from the same

muscle group do not necessarily share common drive: evidence from the human triceps

surae. *J Appl Physiol*. Published online 2020. doi:10.1152/japplphysiol.00635.2020

12. Nunes JP, Costa BDV, Kassiano W, et al. Different Foot Positioning During Calf

Training to Induce Portion-Specific Gastrocnemius Muscle Hypertrophy. *J strength*

Cond Res. 2020;34(8):2347-2351. doi:10.1519/JSC.0000000000003674

13. Knobloch K. Drug-Induced Tendon Disorders. In: Ackermann PW, Hart DA, eds.

Metabolic Influences on Risk for Tendon Disorders. Springer International Publishing;

2016:229-238. doi:10.1007/978-3-319-33943-6_22

14. Martin RL, Chimenti R, Cuddeford T, et al. The VISA-A questionnaire: An index of

the severity of Achilles tendinopathy I. *J Orthop Sports Phys Ther*. 2018;48(5):9-11.

https://pubmed.ncbi.nlm.nih.gov/11579069/

15. Silbernagel KG, Brorsson A, Lundberg M. The majority of patients with Achilles

tendinopathy recover fully when treated with exercise alone: A 5-year follow-up. *Am J*

Sports Med. 2011;39(3):607-613. doi:10.1177/0363546510384789

16. Chimenti RL, Post AA, Silbernagel KG, et al. Kinesiophobia Severity Categories and

Clinically Meaningful Symptom Change in Persons With Achilles Tendinopathy in a

Cross-Sectional Study: Implications for Assessment and Willingness to Exercise.

Front Pain Res. 2021;2(September):1-14. doi:10.3389/fpain.2021.739051

17. Id LKS, Gutsch SB, Hunter SK. The single-leg heel raise does not predict maximal

plantar flexion strength in healthy males and females. Published online 2021:1-16.

doi:10.1371/journal.pone.0253276

18. Hébert-Losier K, Wessman C, Alricsson M, Svantesson U. Updated reliability and

normative values for the standing heel-rise test in healthy adults. *Physiother (United*

Kingdom). 2017;103(4):446-452. doi:10.1016/j.physio.2017.03.002

19. Del Vecchio A, Holobar A, Falla D, Felici F, Enoka RM, Farina D. Tutorial: Analysis

of motor unit discharge characteristics from high-density surface EMG signals. *J*

Electromyogr Kinesiol. 2020;53:102426. doi:10.1016/j.jelekin.2020.102426

20.   Martinez-Valdes E, Laine CM, Falla D, Mayer F, Farina D. High-density surface

electromyography provides reliable estimates of motor unit behavior. *Clin*

Neurophysiol. 2016;127(6):2534-2541. doi:10.1016/j.clinph.2015.10.065

21.   Del Vecchio A, Negro F, Holobar A, et al. 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*. 2019;597(9):2445-2456.

doi:10.1113/JP277396

22.   Farina D, Negro F, Dideriksen JL. The effective neural drive to muscles is the

common synaptic input to motor neurons. *J Physiol*. 2014;592(16):3427-3441.

doi:10.1113/jphysiol.2014.273581

23.   Holobar A, Glaser V. Cumulative Spike Train Outperforms the Root-Mean-Square

Metric in Muscle Excitation Estimation from Dynamic High-Density EMG BT -

Converging Clinical and Engineering Research on Neurorehabilitation III. In: Masia L,

Micera S, Akay M, Pons JL, eds. *Biosystems and Biorobotics*. Vol 21. Springer

International Publishing; 2019:1143-1147. doi:https://doi.org/10.1007/978-3-030-

01845-0_228

24.   Mazzo MR, Holobar A, Enoka RM. Association between effective neural drive to the

triceps surae and fluctuations in plantar-flexion torque during submaximal isometric

contractions. *Exp Physiol*. 2022;107(5):489-507. doi:10.1113/EP090228

25.   Aeles J, Sarcher A, Hug F. Common synaptic input between motor units from the

lateral and medial posterior soleus compartments does not differ from that within each

compartment. *J Appl Physiol*. 2022;134(1):105-115.

doi:10.1152/japplphysiol.00587.2022

26.   Mazzo MR, Weinman LE, Giustino V, Mclagan B, Maldonado J, Enoka RM. Changes

in neural drive to calf muscles during steady submaximal contractions after repeated

static stretches. *J Physiol*. 2021;599(18):4321-4336. doi:10.1113/JP281875

27. Bates D, Mächler M, Bolker BM, Walker SC. Fitting linear mixed-effects models

using lme4. *J Stat Softw*. 2015;67(1). doi:10.18637/jss.v067.i01

28. Fife D. Flexplot: Graphically-based data analysis. *Psychol Methods*. 2022;27(4):477-

496. doi:10.1037/met0000424

29. Lenth R V. Least-squares means: The R package lsmeans. *J Stat Softw*. 2016;69(1):1-

33. doi:10.18637/jss.v069.i01

30. Lakens D, Daniel L, Lakens D. Calculating and reporting effect sizes to facilitate

cumulative science: A practical primer for t-tests and ANOVAs. *Front Psychol*.

2013;4(NOV):1-12. doi:10.3389/fpsyg.2013.00863

31. Negro F, Holobar A, Farina D. Fluctuations in isometric muscle force can be described

by one linear projection of low-frequency components of motor unit discharge rates. *J*

Physiol. 2009;587(24):5925-5938. doi:10.1113/jphysiol.2009.178509

32. De Luca CJ, Gonzalez-Cueto JA, Bonato P, Adam A. Motor unit recruitment and

proprioceptive feedback decrease the common drive. *J Neurophysiol*.

2009;101(3):1620-1628. doi:10.1152/jn.90245.2008

33. Sojka P, Sjölander P, Johansson H, Djupsjöbacka M. Influence from stretch-sensitive

receptors in the collateral ligaments of the knee joint on the γ-muscle-spindle systems

of flexor and extensor muscles. *Neurosci Res*. 1991;11(1):55-62. doi:10.1016/0168-

0102(91)90066-8

34. Leardini A, O'Connor JJ, Catani F, Giannini S. The Role of the Passive Structures in

the Mobility and Stability of the Human Ankle Joint: A Literature Review. *Foot Ankle*

Int. 2000;21(7):602-615. doi:10.1177/107110070002100715

35. Yoshizuka H, Kuraoka A. Calcaneofibular ligament may act as a tensioner of peroneal

tendons as revealed by a contactless three-dimensional scan system on cadavers. *Sci*

Rep. 2022;12(1):1-8. doi:10.1038/s41598-022-21115-5

36.   Kim KM, Ingersoll CD, Hertel J. Altered postural modulation of Hoffmann reflex in

the soleus and fibularis longus associated with chronic ankle instability. *J*

Electromyogr Kinesiol. 2012;22(6):997-1002. doi:10.1016/j.jelekin.2012.06.002

37.   Kim KM, Hart JM, Hertel J. Influence of body position on fibularis longus and soleus

Hoffmann reflexes. *Gait Posture*. 2013;37(1):138-140.

doi:10.1016/j.gaitpost.2012.06.009

38.   Enoka RM, Duchateau J. Rate coding and the control of muscle force. *Cold Spring*

Harb Perspect Med. 2017;7(10):1-12. doi:10.1101/cshperspect.a029702

39.   Crouzier M, Dandois F, Sarcher A, Bogaerts S, Scheys L, Vanwanseele B. External

rotation of the foot position during plantarflexion increases non-uniform motions of

the Achilles tendon. *J Biomech*. 2022;141(July). doi:10.1016/j.jbiomech.2022.111232

40.   Henry PAPBM, Kopacz AOP, Tomaszewski AMJAWKA. The twisted structure of the

Achilles tendon unraveled : A detailed quantitative and qualitative anatomical

investigation. 2017;(December 2016):1705-1715. doi:10.1111/sms.12835

41.   Crouzier M, Lacourpaille L, Nordez A, Tucker K. Neuromechanical coupling within

the human triceps surae and its consequence on individual force sharing strategies.

2018;(October). doi:10.1242/jeb.187260

42.   Khair RM, Stenroth L, Cronin NJ, Reito A, Paloneva J, Finni T. In vivo localized

gastrocnemius subtendon representation within the healthy and ruptured human

Achilles tendon. *J Appl Physiol*. 2022;133(1):11-19.

doi:10.1152/japplphysiol.00084.2022

43.   Heckman CJ, Enoka RM. Motor unit. *Compr Physiol*. 2012;2(4):2629-2682.

doi:10.1002/cphy.c100087

44.   Hug XF, Vogel C, Tucker K, et al. Individuals have unique muscle activation

signatures as revealed during gait and pedaling. 2022;(22):1165-1174.

doi:10.1152/japplphysiol.01101.2018

45.   Crouzier M, Hug F, Dorel S, Deschamps T, Tucker K, Lacourpaille L. Do individual

differences in the distribution of activation between synergist muscles reflect

individual strategies ? *Exp Brain Res*. 2019;237(3):625-635. doi:10.1007/s00221-018-

5445-6

46.   Avrillon S, Del Vecchio A, Farina D, et al. Individual differences in the neural

strategies to control the lateral and medial head of the quadriceps during a

mechanically constrained task. *J Appl Physiol*. 2021;130(1):269-281.

doi:10.1152/JAPPLPHYSIOL.00653.2020

47.   Hasani F, Vallance P, Haines T, Munteanu SE, Malliaras P. Are Plantarflexor Muscle

Impairments Present Among Individuals with Achilles Tendinopathy and Do They

Change with Exercise? A Systematic Review with Meta-analysis. *Sport Med - Open*.

2021;7(1). doi:10.1186/s40798-021-00308-8

48.   Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Full symptomatic recovery does

not ensure full recovery of muscle-tendon function in patients with Achilles

tendinopathy. *Br J Sports Med*. 2007;41(4):276-280. doi:10.1136/bjsm.2006.033464

49.   O'Neill S, Barry S, Watson P. Plantarflexor strength and endurance deficits associated

with mid-portion Achilles tendinopathy: The role of soleus. *Phys Ther Sport*.

2019;37:69-76. doi:10.1016/j.ptsp.2019.03.002

50.   Fernandes GL, Orssatto LBR, Shield AJ, Trajano GS. Runners with mid-portion

Achilles tendinopathy have greater triceps surae intracortical inhibition than healthy

controls. *Scand J Med Sci Sports*. 2021;(July):1-18.

51.   Quarmby A, Mönnig J, Mugele H, et al. Biomechanics and lower limb function are

altered in athletes and runners with achilles tendinopathy compared with healthy

controls: A systematic review. *Front Sport Act Living*. 2023;4(January).

doi:10.3389/fspor.2022.1012471

52.   Hébert-Losier K, Schneiders AG, Sullivan SJ, Newsham-West RJ, García JA,

Simoneau GG. Analysis of knee flexion angles during 2 clinical versions of the heel

raise test to assess soleus and gastrocnemius function. *J Orthop Sports Phys Ther*.

2011;41(7):505-513. doi:10.2519/jospt.2011.3489

53.   Hanlon SL, Pohlig RT, Silbernagel KG. Beyond the diagnosis: Using patient

characteristics and domains of tendon health to identify latent subgroups of achilles

tendinopathy. *J Orthop Sports Phys Ther*. 2021;51(9):440-450.

doi:10.2519/jospt.2021.10271