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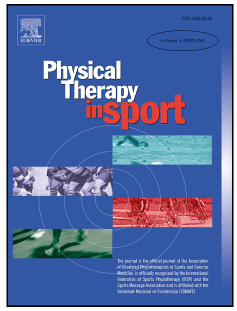

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Plantarflexor strength and endurance deficits associated with mid-portion Achilles tendinopathy: The role of Soleus

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Research report

Plantar Flexor strength and endurance deficits associated with Achilles tendinopathy: The role of the Soleus

Abstract

Objectives

Determine how the strength and endurance of the plantar flexors are affected by Achilles tendinopathy and whether one muscle is more affected than another.

Design

Case control study

Setting

University Laboratory

Participants

39 Runners with mid-portion Achilles tendinopathy and 38 healthy runners participated in this study.

Main Outcome Measures

Isokinetic dynamometry was completed bilaterally in two knee positions on all subjects to assess the torque and endurance capacity of the plantar flexors.

Results

Subjects with Achilles tendinopathy were statistically weaker (by 26.1Nm Concentric 90°/sec, 14,8Nm Concentric 225°/sec and 55.5Nm Eccentric 90°/sec for knee extended testing and 17.3Nm, 10.1Nm and 52.3Nm for the flexed knee respectively) than healthy controls at all isokinetic test speeds and contraction modes irrespective of knee position (p value = <0.001). The endurance capacity of the plantar flexors was significantly reduced (Total work done 613.5Nm less) in subjects with Achilles tendinopathy when compared to the healthy controls (p value = <0.001).

Conclusions

Achilles tendinopathy is associated with large deficits in plantar flexor torque and endurance. The deficits are bilateral in nature and appear to be explained by a greater loss of the soleus force generating capacity rather than the gastrocnemius.

Key terms

Achilles Tendinopathy, Endurance, Strength, Soleus

Clinical relevance:

1. There are large statistically and clinically meaningful differences in plantar flexor strength and endurance between subjects with and without Achilles tendinopathy.
2. Rehabilitation should take into account the specific strength and endurance deficits associated with Achilles tendinopathy.
3. Our data shows healthy runners generate eccentric torque of twice bodyweight, In order to rehabilitate subjects with Achilles tendinopathy to this level substantial external loads will be required.
4. Researchers and clinicians cannot use the non-symptomatic limb as a comparator when assessing strength and endurance, instead normative values need to be considered.

What is known:

We know that changes in plantar flexor force capacity occur in the presence of tendinopathy but we do not know which of the plantar flexors are most affected and how these strength deficits relate to healthy controls. Neither do we understand the effects of tendinopathy on endurance capacity of the plantar flexors.

What this study adds to the literature

This study is the largest study examining force deficits of the plantar flexors between subjects with and without Achilles tendinopathy and it is the first study to examine endurance of the plantar flexors using isokinetic dynamometry. This is the first study to compare healthy subjects to injured subjects, the first to identify that plantar flexor deficits appear to relate to the soleus rather than the gastrocnemius and the first to identify endurance differences between symptomatic limbs and asymptomatic limbs. This study helps us to understand the extent of the force capacity changes associated with Achilles tendinopathy and gives the clinician and researcher a basis for clinical interventions/further research.

Plantar Flexor strength and endurance deficits associated with mid-portion Achilles tendinopathy: The role of the Soleus

1.1 Introduction

Muscle weakness has been identified as an important factor in mid-portion Achilles tendinopathy (AT) with experts suggesting this is the primary modifiable risk factor for athletic tendinopathy.[62] The mid-portion relates to the zone 2-6cm proximal to the insertion whilst the insertion is the more distal section of the tendon. It is not clear whether muscle weakness is important for insertional AT. In particular, the plantar flexor muscles group have been identified as the most important in relation to Mid-portion AT. It appears likely that both the gastrocnemius and soleus may influence the magnitude and distribution of Achilles tendon load, stress and strain and therefore may impact on the patho-aetiology of disease.[22,53,54,63] Various authors have investigated calf muscle strength and identified differences between affected and unaffected legs, some authors have gone on to examine whether the identified deficits change with rehabilitation.[3,4,70,72] The current suggestion from level one evidence is that these neuromuscular changes (torque, work and endurance) offer the best explanation for the observed clinical benefit of loading programs.[54] Further evidence suggests plantar flexor weakness predates the onset of Achilles tendon pain, strengthening the cause and effect relationship between plantar flexor weakness and tendinopathy.[52] However no consideration has been given to which muscle is most affected, gastrocnemius or soleus.

During locomotion the soleus muscle functions in relative isolation from the gastrocnemius,[19,49] throughout ground contact the soleus controls knee flexion by controlling tibial movements in relation to the foot and therefore the floor (knee extensor moment), whilst the gastrocnemius opposes this action and acts to flex the knee.[48] In later stance, the soleus decelerates the leg through its action at the ankle and propels the trunk forwards through its plantar flexor function,[26] producing vertical forces of around 8 times body weight.[24] The soleus is able to produce these large forces by nature of its physiological cross sectional area (PCSA), the largest of any lower limb muscle.[29,84] The gastrocnemius in comparison acts to accelerate the leg and decelerate the trunk during mid single leg stance,[60] although activity in later stance produces acceleration of the trunk,[48] the gastrocnemius produces forces of 3 times body weight.[24] Recently, authors have suggested that dysfunction of the soleus may be most associated with AT, highlighting the importance of further study into this muscle.[85]

The Achilles tendon is comprised of a complex orientation of fascicles and until recently the strain through the tendon was considered to be homogenous, however this has recently been challenged with the current evidence suggesting that stress varies markedly across different tendon zones, fascicles.[27,73,74] Interestingly the literature supports the notion that in the mid-portion the deeper surface of the Achilles tendon, that which is comprised of fascicles linked to the soleus,[25,76] undergoes the greatest displacement[9,13,27,73,74], this same zone appears to be where the typical changes associated with tendinopathy can be observed.[17,30] These findings suggest that interfasicular sliding is occurring and this has been suggested as important in the patho-genesis of AT.[27,77,78] This raises the question of whether neuromuscular function of the plantar flexors may play a pivotal role in the development of AT[22,52,63] and be critical in rehabilitation and injury management. [22,52,63]

Given that the soleus is the main force producer in activities most associated with AT (running and walking) [29,84] and that endurance running rather than sprinting seems to be most associated with AT (endurance being related to soleus function)[44] and that the exact site of tendinopathy appears to involve tendon fascicles most associated with the soleus[17] it would appear feasible that the soleus may be primarily affected. Identification of how the plantar flexors are affected and which of the plantar flexors are primarily affected by AT may also aid rehabilitative decisions. However, there are no studies in the published literature that have assessed either soleus strength or power versus gastrocnemius strength.

The purpose of this study was to determine how the plantar flexors are affected by AT. We hypothesised that there would be significant differences in power and endurance of the plantar flexors when comparing subjects with and without AT and that these deficits would be bilateral in nature and explained by alterations in soleus function.

2 Method

2.1 Study design

Ethical approval for this research protocol (so59-4446) was provided by a university ethics board prior to the start of this study. All subjects underwent a fully informed consent procedure prior to any data collection.

The study used an observational methodology to compare a group of runners with and without AT. The subjects without AT acted as a control group and were age, sex and activity matched to an individual subject with AT. The activity matching was pragmatic in that all subjects needed to be endurance runners who ran more than twice a week, this was chosen over exact distance as there was too much variation in weekly training volume. A University physiotherapy research laboratory which housed the isokinetic dynamometer, ultrasound unit and clinical equipment was used to complete all examinations and testing. The sample was recruited from local running clubs.

A diagnosis of mid-portion AT was made if subjects had localized unilateral mid-portion Achilles tendon pain for more than three months, pain provoked by physical activities in a dose dependent way (i.e. running activities provoke pain more than walking, pain that remains or increases after completion of exercise but reduces over time and is subsequently aggravated with the next loading session/activity), reproduction of pain with palpation of the tendon,[37,66] positive London hospital test and/or Painful arc sign of the Achilles tendon,[50,66] and the identification of ultrasonographic features in keeping with a diagnosis of AT, specifically heterogeneous echogenicity and anterior to posterior diameter greater than 6mm.[8,10,17,61,67,75] A negative scan, normal appearance of the Achilles tendon (homogenous echogenicity) , was used as an exclusion criteria for the AT subjects as ultrasound has a good negative predictive value.[42] A negative scan identifies normal anterior to posterior diameter, <6mm, and homogenous echogenicity. Whilst there is much debate about the relationship between tendon structure and pain, the presence of Achilles tendon pain in the absence of structural changes is exceptionally rare, as such imaging is normally used in both clinical work and research to confirm the diagnosis and exclude other tendon pathologies that may mimic tendinopathy e.g. superficial retrocalcaneal bursitis. Recent work has identified a relationship between Achilles tendon structure and function whilst another study has shown the site of most pathology to correspond to the site of most pain further highlighting the importance of tendon structure to pathology.[16,23] The ultrasound scan was performed by the same experienced physiotherapist (SO'N) who has 10 years experience of ultrasound imaging Achilles tendons and 18 years experience as a physiotherapist. Only the affected limb was imaged.

If subjects were diagnosed with mid-portion tendinopathy then the inclusion and exclusion criteria were applied to them. Inclusion criteria was a diagnosis of unilateral mid-portion AT for >3 months duration, the subject normally ran >2 times per week or was a healthy runner running more than >2 times per week with no lower limb pain, all subjects needed to be between the ages of 18-70 years old and able to give informed consent. This criteria was based on previous studies and common clinical practice.[12,28,37,50,66] Exclusion criteria was any musculoskeletal, vascular or neurological injury/disorder within last six months (except unilateral AT for the injured group), bilateral AT, participates in regular lower limb strength training, or regularly participated in other sports involving high speed running (football, rugby, hockey etc.). Subjects with AT had additional exclusion criteria applied to ensure they represented a similar group of AT patients to existing research: Clinical findings suggestive of a fascia cruris tear, longitudinal tear/split or partial rupture or concurrent presence of insertional tendinopathy, previous rupture of or surgery to Achilles tendon, previous or current treatment for their tendinopathy or a negative ultrasound scan.[39,55,82] Prior to testing all subjects completed a VISA A questionnaire.[69] Ultrasound imaging was not completed for the healthy subjects.

2.1.1 Peak plantar flexor torque

The primary outcome measure was peak torque of the plantar flexors measured by use of an isokinetic dynamometer (Humac Norm, CSMI solutions, USA), measurements were completed at 80° knee flexion and full knee extension using 3 different modes:[1,3,51,52] concentric 90°/sec, concentric 225°/sec and eccentric 90°/sec. The knee extended position allowed both the gastrocnemius and soleus to function,[19,43,47,49] whilst the knee flexed position mechanically disadvantaged the gastrocnemius, thereby testing soleus force production more specificially.[7,20,34-36,46] Previous research has identified that knee flexion of 80° was sufficient and feasible to complete the required testing.[7,20,34-36,46,47,47,65] Each test position adhered to the manufacturer's guidance on subject positioning (Humac Norm, CSMI solutions, USA). A goniometer was used to measure and confirm joint angles for the knee and ankle during the setup of the dynamometer. A neutral foot position of 0° (plantargrade) was used as the starting point and the range of motion was defined as 20° of dorsi-flexion to 30° of plantarflexion.[2-4] The protocols and verbal encouragement were all standardised with a submaximal warmup exercise in each test position and at each speed, further details of the protocol are provided in Al-Uzri et al.[5] Participants performed the test in the extended-knee position first and then the flexed knee position. All subjects underwent testing on their affected leg prior to their un-affected leg. A pilot study assessed for an order effect using this protocol and found there to be none. Along with the typical isokinetic measure of peak torque the humac norm also represents force as a percentage of body weight (Peak torque/BW = %BW), this measure is probably more transferable across different populations as for locomotive muscles force production in relation to body mass is probably more important than force production per se. Our reliability study confirmed this measure was reliable[5] and this parameter could allow broader comparison between individuals of different weight.

The isokinetic protocol used for this study was a replica of Alfredson's et al[3,4] original study,[1] but differed in that both knee flexion and knee extension test positions were utilised. Due to this we completed a large scale reliability study using 37 healthy subjects.[5] As part of this study we calculated minimal detectable change (MDC) for the different speeds and test positions as there was no previous data relating to the MDC, this data can be seen in Al-Uzri et al.[5]

2.1.2 Plantar flexor Endurance

Since there were no published studies of plantar flexor endurance protocols on isokinetic dynamometers, we extensively tested a variety of protocols before choosing a regime of 20 maximal effort concentric-eccentric plantar flexor contractions. This protocol was chosen as it reflected heel raise tests which had previously been shown to differ in subjects with AT.[70,71] The endurance protocol underwent reliability testing using 37 healthy subjects. Due to the variety of endurance measures that can be reported by the CSMI software we assessed which measure, endurance ratio, fatigue index and total work done, was the most reliable. This particular study found the test re-test reliability of the endurance ratio and fatigue index to be insufficient whilst the total work done (TWD) was satisfactory and was therefore used in this study.[41] The MDC for TWD was 321 Nm and the test re-test reliability had an ICC value between 0.75-0.91 dependent on the component of the test (concentric or eccentric phase).

The endurance test was completed in the flexed knee position as it was not feasible to complete the endurance test in both knee positions and we were aiming to test the soleus capacity in isolation due to our initial pilot data which suggested this position would identify any deficits of relevance to runners. The endurance test was the last and final test to be completed on each limb. The endurance test was done last so that all subjects would engage fully with the maximum contractions required for the test protocol.

Previous research has shown that the angular velocity of the ankle joint during running is around 90°/sec, this is nearly double the commonly recommended isokinetic test speed of 45°/sec for the ankle joint and significantly lower than the peak ankle joint velocity during running (200°/sec).[31,58] However, this typical test speed is based on isokinetic and not clinical principles. Previous research has already confirmed that this speed is of practical interest in the population under examination.[2-4,6,21,35,38,57,59,83] Therefore a test speed of 90°/sec was used for both the peak power and endurance tests. Familiarisation was completed using seven submaximal repetitions

2.2 Pain

Extensive pilot testing had shown that pain was not experienced during the maximal isokinetic tests, however all subjects were instructed to complete the maximal tests as pain allowed. To ensure we could account for the effect of pain on force generation all subjects were asked to score any discomfort/pain during the isokinetic testing protocol using a VAS score of 100mm. Statistical analysis

A sample size of 38 subjects in each group was calculated using an a priori power calculation based on our primary outcome measure – plantar flexor peak eccentric and concentric torque. A sample size of 38 subjects per group would give 80% power to detect a mean difference of 8 N·m (SD16.1), using a paired t-test with significance set at 5%. This level was set based on data from McCrory et al[56] and our preliminary testing. This level of difference was chosen as a conservative level despite Alfredson et al reporting higher variations.[3] It was not possible to calculate a sample size for endurance capacity as there was no previous published data.

All parameters in this study were normally distributed and therefore exposed to parametric testing, in this instance paired t tests to compare between limbs and groups. Matching involved individual matching of limbs between subjects using gender and age to within ± 4 years. Matching utilised symptomatic limb being matched with the same side of a healthy control, e.g. right limb AT matched with right limb of healthy control, whilst their left limbs would then also be matched. This process was repeated for all subjects. Activity matching was based on completing more than two endurance runs per week. The matching process utilised in this study does not violate the assumptions required to be met for a paired t test, therefore a paired t test is appropriate. [11,32] Matching was not completed based on height and weight as it was not possible to match on these variables without a much larger sample size, predominately due to the lack of healthy previously uninjured runners.

Calculations were performed on both concentric and eccentric muscle contractions using peak torque and peak torque presented as a percentage of an individuals' body weight (%BW). During matching one female subject had their right leg and left leg matched with two different AT subjects. This decision was taken as it was remarkably difficult to recruit healthy controls who had not actually suffered from recent injuries. We also felt that this decision was unlikely to impact on the study results as we were not actually using the same limb twice. For comprehensiveness statistical testing was completed with and without this data being included. There was no difference in statistical significance whether this subject was excluded ($p = <0.001$) or not ($p = <0.001$). Due to the large number of statistical tests a Bonferroni correction (Alpha correction) was completed, this identified an alpha value of 0.00139 as appropriate.

The MDC^{95} for the isokinetic measurements was determined based on previously published work using the same isokinetic protocol to this study.[40,41] This level was then used to determine if group differences exceeded the measurement error (MDC) and reflected actual differences between limbs or between groups.

TABLE 1. Demographic data split for the Achilles tendinopathy and healthy control group

Variable	Achilles tendinopathy (n=39)	Healthy Controls (n=38)
Age (years)	47 (11.8)	44 (9.9)
Sex	34 Males	35 Males
Height (cm)	177 (6.8)	175 (8.1)
Weight (Kgs)	77 (12.1)	70.4 (10.3)
BMI	24 (2.7)	23 (2.7)

3 Results

A total of 54 subjects with a potential diagnosis of AT attended the clinic for examination. 15 failed to meet the inclusion/exclusion criteria, leaving 39 AT subjects and 38 healthy controls. The basic demographic data including shows little difference except in VISA A scores (Table 1).

VISA A score	56 (17.8)	100 (0)

Data represents Mean and SD in brackets for each variable. The number of subjects in each group is also shown.

3.1.1 Peak plantar flexor torque

The majority of the power measurements revealed minimal differences between the symptomatic limb and the non-symptomatic limb in either knee position (Table 2). The only differences that reached a statistical threshold occurred for concentric 90°/sec in knee extension and eccentric 90°/sec in knee flexion, however neither measure exceeded the previously determined MDC. However once the Bonferroni correction was applied, none of these results were significant.

TABLE 2. A comparison of peak plantar flexor torque between symptomatic and non-symptomatic legs in subjects with Achilles tendinopathy

Knee position	Contraction mode	Symptomatic side	Non-Symptomatic side	p value (comparison of legs)
Extended knee	Concentric 90°/sec	42.2 (16.9)	45.7 (15.6)	0.008*
	Concentric 90°/sec (%BW)	53.9 (19.0)	59.6 (18.9)	0.002*
	Concentric 225°/sec	29.1 (9.1)	30.8 (8.7)	0.084
	Concentric 225°/sec (%BW)	37.9 (9.8)	40.2 (9.1)	0.076
	Eccentric 90°/sec	83.9 (35.0)	84.4 (35.5)	0.900
	Eccentric 90°/sec (%BW)	109.9 (42.1)	109.6 (43.2)	0.952
Flexed knee	Concentric 90°/sec	46.3 (20.1)	48.9 (16.0)	0.246
	Concentric 90°/sec (%BW)	60.1 (21.3)	63.6 (17.6)	0.203
	Concentric 225°/sec	33.5 (10.5)	34.1 (9.0)	0.647
	Concentric 225°/sec (%BW)	44.0 (10.9)	45.2 (9.9)	0.461
	Eccentric 90°/sec	92.6 (47.9)	102.7 (41.7)	0.045*
	Eccentric 90°/sec (%BW)	119.5 (56.3)	132.0 (50.0)	0.068

Data represents Mean and (SD), with force presented as a Newton Metre (Nm) or Percentage Body Weight (%BW)
* = Statistically significant value with a paired t test but not with the Bonferroni correction. None of the results exceeded the MDC^{95}.

The comparison of AT subject's symptomatic and non-symptomatic limbs to healthy controls found all measurements were significantly different irrespective of knee position. More importantly all of these measures exceeded the MDC^{95} (Table 3). In an attempt to express the magnitude of the force deficits being reported the AT subject's data was presented as a percentage force of the healthy controls (Table 4).

Table 3 Comparison of plantar flexor peak torque between subjects with and without Achilles tendinopathy

Knee position	Contraction mode	Symptomatic side	Non-Symptomatic side	Healthy control	p value (control vs symptomatic side)	p value (control vs non-symptomatic side)
Extended knee	Concentric 90°/sec	42.2 (16.9)	45.7 (15.6)	68.3 (24.2)	<0.001*†	<0.001*†
	Concentric 90°/sec (%BW)	53.9 (19.0)	59.6 (18.9)	94.9 (29.80	<0.001*†	<0.001*†
	Concentric 225°/sec	29.1 (9.1)	30.8 (8.7)	43.9 (16.3)	<0.001*†	<0.001*†
	Concentric 225°/sec (%BW)	37.9 (9.8)	40.2 (9.1)	61.6 (20.3)	<0.001*†	<0.001*†
	Eccentric 90°/sec	83.9 (35.0)	84.4 (35.5)	139.4 (44.7)	<0.001*†	<0.001*†
	Eccentric 90°/sec (%BW)	109.9 (42.1)	109.6 (43.2)	194.1 (51.8)	<0.001*†	<0.001*†
Flexed knee	Concentric 90°/sec	46.3 (20.1)	48.9 (16.0)	63.6 (17.6)	<0.001*†	<0.001*†
	Concentric 90°/sec (%BW)	60.1 (21.3)	63.6 (17.6)	88.8 (19.6)	<0.001*†	<0.001*†
	Concentric 225°/sec	33.5 (10.5)	34.1 (9.0)	43.6 (13.6)	<0.001*†	0.001*†
	Concentric 225°/sec (%BW)	44.0 (10.9)	45.2 (9.9)	61.0 (14.7)	<0.001*†	<0.001*†
	Eccentric 90°/sec	92.6 (47.9)	102.7 (41.7)	144.9 (35.7)	<0.001*†	<0.001*†
	Eccentric 90°/sec (%BW)	119.5 (56.3)	132.0 (50.0)	202.3 (38.5)	<0.001*†	<0.001*†

Data represents Mean and (SD), with force presented as a Newton Metre (Nm) or Percentage Body Weight (%BW)
* = statistically significant value with a paired t test and independent t test, †= Difference exceeds the MDC^{95}

Table 4. Force output of AT subjects as a percentage of the control data.

Contraction mode	Force output in knee extension as % of healthy controls	Force output in knee flexion as % of healthy controls	Actual difference between knee flexion and extension
Concentric 90°/sec	61.8	72.8	11
Concentric 225°/sec	66.3	76.8	10.5
Eccentric 90°/sec	60.2	63.9	3.7

The mean of the AT group is reported as a percentage of the healthy control group for each test speed and test position for the symptomatic leg. The actual difference reports the force loss between positions and relates to gastrocnemius force.

3.1.2 Endurance results

The endurance capacity of the plantar flexors (TWD) was significantly different when comparing the symptomatic limb or non-symptomatic limb (Table 5), with both measures exceeding the MDC^{95}. The between limb difference in AT subjects was 177Nm, with the symptomatic limb being weaker, this did not exceed the MDC^{95}. The symptomatic limb was 613.5Nm weaker than a healthy subject's limb and the asymptomatic limb was 436.5Nm weaker than a healthy subjects.

Table 5. The endurance capacity of the plantar flexors expressed as Total Work Done (N·m)

Limb	Achilles tendinopathy group	Healthy controls (n=38)	p value (Control: Achilles subjects)	p value (symptomatic : non-symptomatic leg)
Symptomatic limb	1313.0 (469.6)	1926.5 (512.3)	<0.001**	0.009*
Non Symptomatic limb	1490.0 (511.3)		<0.001**	

Results report the mean for TWD in Nm and the SD in brackets. Between group analysis is reported as p value for comparisons of control subjects with either AT subjects symptomatic or non-symptomatic leg and also between AT subjects legs. * = statistically significant difference. **= Identifies that the results exceeded the MDC95 for endurance (321Nm).

3.1.3 Pain

No pain or discomfort was reported at any time of the test protocol by either AT or healthy subjects.

4 Discussion

4.1 Strength deficits between the Achilles group and healthy controls

This is the first study to compare plantar flexor motor output (peak torque and endurance) between individuals with and without AT. The results clearly show that there are large deficits in strength between subjects with and without AT. The magnitude of deficits is clinically and statistically significant in all test modes and both knee positions. The magnitude of difference exceeds the MDC^{95} identified within previous work.[5] The deficits are greatest in eccentric test mode in the extended knee position.

The data also shows that there are some statistically significant differences between symptomatic and non-symptomatic limbs in subjects with AT without the Bonferroni correction. Importantly these were few and the actual differences very small with none of the differences exceeding the appropriate MDC^{95}. This suggests that the differences should be interpreted as clinically and statistically irrelevant, especially once the Bonferroni correction is applied. This finding contrasts with previous Achilles data from McCrory [56] and Alfredson.[3] Both of these researchers reported differences between limbs, 4Nm and 21Nm respectively. McCrory's results would fall within the likely MDC^{95} for the measurements used whilst Alfredson's would exceed the MDC^{95} value. It is possible that Alfredson's findings differ due to sample size, n=15 versus n=39 in this study.[3]

The identification of bilateral weakness in subjects with AT is in keeping with the findings in upper limb tendinopathy[14,15] and the conclusion of two systematic reviews in this area.[33,64] These findings may relate to central nervous system involvement as identified in lower limb tendinopathy,[68,79] or be a consequence of de-training/de-conditioning or pre-existing weakness.[81]

4.2 Soleus appears most involved

The soleus muscle produces similar force irrespective of the knee position, whereas the gastrocnemius generates markedly less force when the knee is in large degrees of flexion.[7,34-36,46,47,80] If the gastrocnemius was responsible for the observed plantar flexor deficits there would be little difference in plantar flexor force output between the AT and healthy control group in knee flexion and a large difference in the knee extended position (table 4), since this is not the case we must consider that the soleus muscle is most affected by AT. Table 3 and 4 report clear differences between the two groups that are very similar irrespective of knee position, as already acknowledged the soleus muscle produces force maximally in both positions, since the deficits are so similar across test positions we must accept that the force capacity of the soleus appears to be most affected by AT. The small percentage difference in force (healthy control torque/AT torque) observed between knee flexion and extension suggests that the gastrocnemius accounts for between 3.7 -11% of the identified deficits, whilst the soleus may be responsible for the remaining 23.2- 36.1% of the difference, table 4. It is difficult to contrast our findings with previous studies as no other published study has attempted to compare the force production of the soleus and gastrocnemius in subjects with AT. However, soleus electromyographic (EMG) readings have been studied previously, Wyndow et al[85] reported alterations to the soleus EMG, suggesting a mild timing deficit but did not examine force or maximal voluntary contractions.

Whilst there was some expectation that plantar flexor forces in knee flexion would be weaker than knee extension [7,18] this was not observed and probably represents a specific alteration in the individuals included in our study. All subjects were runners and runners habitually produce the largest plantar flexor forces eccentrically during flexed knee positions.[24] This same finding has been observed by others. [46,74]

4.2.1 Endurance capacity

The endurance data shows a clear clinically meaningful and statistically significant difference between subjects with and without AT, this difference exceeded the MDC[95]. The between limb difference in subjects with AT was statistically significant but did not exceed the MDC[95] (321 N·m). No previous data exists on the endurance capacity of the plantar flexors using isokinetic dynamometry in individuals with AT. The only study to have measured endurance in any quantifiable method is Silbernagel [70-72] who used a heel raise endurance test until fatigue. Silbernagel compared symptomatic limbs with non-symptomatic/least symptomatic limbs and did not compare against controls. Silbernagel concluded that they did not find any statistical difference in endurance capacity between symptomatic and non-symptomatic/least symptomatic limbs. However, their testing protocol was very different from the isokinetic protocol utilised within our study and they had a large number of subjects with bilateral symptoms (40%) compared with 0% in our study, this may explain why they reported no between limb difference.

4.3 Pain

Importantly whilst all the subjects within our study were symptomatic and had pain/discomfort during the clinical examination none of the subjects experienced pain during the actual test protocol despite producing MVC of the plantar flexors. This was also observed during our pilot studies and is potentially related to the context of the exercise and novel experience of being within a university laboratory whilst undergoing “elaborate” testing procedures. The lack of pain also suggests that pain may not have affected the torque data.

4.4 Limitations

Due to the power calculation and sample size we can have confidence in the findings of large differences between subjects with and without AT. The main limitations relate to the population studied in that they are all active athletic individuals participating in endurance based running activities, however they are representative of the typical AT population reported in other studies. Due to the specific group characteristics it may be that sedentary subjects or jumping athletes with AT differ, however it is likely that there would be similar findings albeit with the mean torque being higher for jumping athletes and lower in sedentary subjects. The lack of ultrasound imaging of the healthy subjects may have led to the inclusion of subjects with pathology who were asymptomatic. Blinding of the assessors during the isokinetic dynamometry was not possible due to funding constraints.

In order to confirm the observed torque and endurance deficits are attributable primarily to the soleus, it is important for further studies to assess soleus and gastrocnemius function using a combination of Isokinetic measurements, US measures of muscle activation, pennation angle and fascicle length, whilst also collecting EMG data. This combination of measurements would allow a greater depth of analysis of soleus function during testing and highlight the role the gastrocnemius has when the knee is flexed. As a key part of these studies matching should be undertaken based on PCSA of muscles although it is important to identify both the requirement in time, equipment and the likely difficulty with recruiting enough subjects to actually match cases. It is important to identify that a small contribution of force is likely to arise from the deep flexor muscles, tibialis posterior, the peronei group and toe flexors, but due to their mechanical disadvantage this is likely very small.

The previously calculated MDC^{95} may be overly conservative and broad as it was based on a mixed sex, mixed leg dominance and mixed activity level cohort. A more restricted single sex population would present a much narrower figure for the MDCs. However, the cohort used in the study by Al-Uzri et al could be considered a useful cohort as it reflects the variation we see in clinical practice.[5] The high MDC^{95} value ensures that those measures exceeding this value are indeed actual differences and not just measurement error. This study is the only study that has compared plantar flexor strength between the limbs of individuals with unilateral AT and considered previously identified MDC^{95} values. As such the results can be interpreted with a high level of confidence.

Matching was limited by ensuring runners ran >2 times per week. Matching for exact training loads using distance would have been desirable, but proved nearly impossible in practice, as subjects varied so much from week to week. The level of matching used within the study was set at 4 years due to the difficulty with recruiting injury free runners and the lack of evidence suggesting large year by year reductions in plantar flexor strength, it would appear unlikely that a tighter level of matching would influence the results.

4.5 Clinical relevance

The lack of difference in torque between limbs of subjects with AT suggests that clinicians should not aim to rehabilitate peak torque to that of the uninjured/asymptomatic limb as this limb does not appear to have normal power. Clinicians should instead use normative data from relevant populations, e.g. runners in this study, as rehabilitation targets for plantar flexor torque.

The peak torque data (Table 3) demonstrates that healthy subjects eccentric force capacity is around twice body weight, therefore to rehabilitate this capacity high external loads will be required. This has been something that has been particularly lacking in rehabilitation as many have assumed the plantar flexors, particularly the soleus, to be weak. Our introduction highlighted the capacity of the soleus for force generation and our findings of twice bodyweight force output (in knee flexion) equate to soleus intramuscular force of approximately six to eight times body weight when typical lever arms are calculated for the ankle joint. It would appear likely that rehabilitating force capacity to levels of around twice bodyweight would allow the plantar flexors to function within normal physiologic levels during locomotion and that this may account for improvements in the clinical manifestation of AT. [22,45,63] It is important that rehabilitation also target the bilateral weakness identified as this may explain the high rates of the asymptomatic limb becoming symptomatic in the future. Whether it is possible to improve these neuromuscular deficits remains to be tested.

5 Conclusion

There are large plantar flexor torque and endurance deficits between subjects with and without AT. These deficits are bilateral suggesting that the non-symptomatic limb should not be used as a "healthy limb" or to provide between limb comparisons with the symptomatic limb in future studies or during clinical work. Weakness of the soleus appears to be responsible for the majority of the deficits observed in participants with AT. Further work needs to determine how current clinical interventions alter these deficits and whether they link to clinical outcome and whether these changes exist prior to or as a consequence of AT.

References

1. 1. Alfredson H, Pietila T, Josson P, Lorentzon R. Heavy load eccentric calf muscle training for the treatment of chronic achilles tendinosis. *Am J Sports Med*. 1998;26(3):360-366.

2. Alfredson H, Nordstrom P, Pietila T, Lorentzon R. Bone mass in the calcaneus after heavy loaded eccentric calf-muscle training in recreational athletes with chronic achilles tendinosis. *Calcif Tissue Int*. 1999;64(5):450-455.

3. Alfredson H, Pietila T, Jonsson P, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic achilles tendinosis. *Am J Sports Med*. 1998;26(3):360-366.

4. Alfredson H, Pietila T, Lorentzon R. Chronic achilles tendinitis and calf muscle strength. *Am J Sports Med*. 1996;24(6):829-833.

5. Al-Uzri M, O'Neill S, Kelly C, Watson P. Reliability of isokinetic dynamometry of the plantarflexors - in knee flexion and extension. *Physiother Pract Res* . 2017;38(1):49-57.

6. Andersen H. Reliability of isokinetic measurements of ankle dorsal and plantar flexors in normal subjects and in patients with peripheral neuropathy. *Arch Phys Med Rehabil*. 1996;77(3):265-268.

7. Arampatzis A, Karamanidis K, Stafilidis S, Morey-Klapsing G, DeMonte G, BrÃ¼ggemann G. Effect of different ankle-and knee-joint positions on gastrocnemius medialis fascicle length and EMG activity during isometric plantar flexion. *J Biomech*. 2006;39(10):1891-1902.

8. Archambault JM, Wiley JP, Bray RC, Verhoef M, Wiseman DA, Elliott PD. Can sonography predict the outcome in patients with achillodynia? *J Clin Ultrasound*. 1998;26(7):335-339.

9. Arndt AN, Komi PV, BrÃ¼ggemann G-, Lukkariniemi J. Individual muscle contributions to the in vivo achilles tendon force. *Clin Biomech*. 1998;13(7):532-541.

10. Bandinelli F, Prignano F, Bonciani D, et al. Ultrasound detects occult entheseal involvement in early psoriatic arthritis independently of clinical features and psoriasis severity. *Clin Exp Rheumatol*. 2013;31(2):219-224.

11. Bland J, Altman D. Statistics notes: Matching. *General practice*. 1994;309.

12. Blankstein A, Cohen I, Diamant L, et al. Achilles tendon pain and related pathologies: Diagnosis by ultrasonography. *Isr Med Assoc J*. 2001;3(8):575-578.

13. Chimenti R, Flemister A, McMahon J, Flannery M, Xue A, Houck J. Altered tendon characteristics and mechanical properties associated with insertional Achilles tendinopathy. *J Ortho Sports Phys Ther.* 2014;44(9):680-689.

14. Coombes B, Bisset L, Vicenzion B. A new integrative model of lateral epicondylalgia. *Bri J Sports Med*. 2009;43:252-258.

15. Coombes B, Bisset L, Vincenzino B. Thermal hyperalgesia distinguishes those with severe pain and disability in unilateral lateral epicondyalgia. *Clin J Pain*. 2012;28(7):595-601.

16. Corrigan P, Cortes D, Pontiggia L, Silbernagel K. The degree of tendinosis is related to symptom severity and physical activity levels in patients with midportion achilles tendinopathy. *Int J Sports Phys*. 2018;13(2):196.

17. Counsel P, Comin J, Davenport M, Connell D. Pattern of fascicular involvement in midportion achilles tendinopathy at ultrasound. *Sports Health*. 2015;7(5):424-428.

18. Cresswell A, Loscher W, Thorstensson A. Influence of gastrocnemius muscle length on triceps surae torque development and electromyographic activity in man. *EJ Exp Biol*. 1995;105(2):283-290.

19. Cronin NJ, Avela J, Finni T, Peltonen J. Differences in contractile behaviour between the soleus and medial gastrocnemius muscles during human walking. *J Exp Biol*. 2013;216(Pt 5):909-914.

20. Dalton B, Allen M, Power G, Vandervoot A, Rice C. The effect of knee joint angle on plantarflexor power in young and old men. *Exp Gerontol*. 2014:70-76.

21. DanneskioldSamsoe B, Bartels EM, Bulow PM, et al. Isokinetic and isometric muscle strength in a healthy population with special reference to age and gender. *Acta Physiologica*. 2009;197(Sup 673):1-68.

22. Debenham J, Gibson W, Travers M, Campbell A, Allison G. Eccentric loading of the triceps surae modulates stretch shortening cycle behaviour- A possible therapeutic mechanism. *J Sport Rehabil*. 2016;24(epub):1-22.

23. Divani K, Chan O, Padhiar N, et al. Site of maximum neovascularisation correlates with the site of pain in recalcitrant mid-tendon Achilles tendinopathy. *Man Ther*. 2010;15(5):463-468.

24. Dorn T, Schache A, pandy M. Muscular strategy shift in human running: Dependence of running speed on hip and ankle muscle performance . *J Exp Biol*. 2012;215:1944-1956.

25. Edama M, Kubo M, Takabayashi T, et al. The twisted structure of the human achilles tendon. *Scand J Med Sci Sports*. 2014.

26. Francis C, Lenz A, Lenhart R, Thelen DG. The modulation of forward propulsion, vertical support, and center of pressure by the plantarflexors during human walking. *Gait Posture*. 2013:1-5.

27. Franz J, Slane L, Rasske K, Thelen D. Non-uniform in vivo deformations of the human achilles tendon during walking *Gait posture*. 2015;41:192-197.

28. Fredericson M. Common injuries in runners: Diagnosis, rehabilitation and prevention. *Sports Med*. 1996;21(1):49-72.

29. Fukunaga T, Roy R, Shellock F, et al. Physiological cross-sectional area of the human leg muscles based on magentic resonance imaging. *J Orthop Res*. 1992;10(6):926-934.

30. Gibbon W, Cooper R, Radcliffe G. Distribution of sonographically detected tendon abnormalities in patients with a clinical diagnosis of chronic achilles tendinosis. *J Clin Ultrasound*. 1999;28(2):61-66.

31. Granata K, Abel M, Damiano B. Joint angular velocity in spastic gait and the influence of muscle tendon lengthening. *J Bone Joint Surgery Am*. 2000;82(2):174-186.

32. Grau S, Malmberg J, Suni J, et al. Influences of matching populations on kinematic and kinetic variables in runners with iliotibial band syndrome. *J Bone Joint Surgery Am* 2008;79(4):450-457.

33. Heales L, Lim E, Hodges P, Vincenzino B. Sensory and motor deficits exist on the non-injured side of patients with unilateral tendon pain and disability- implications for central nervous system involvement: A systematic review with meta analysis. *Br J Sports Med*. 2014;48:1400-1406.

34. Hebert_losier K, Schneiders A, Garcia J, Sullivan J, Simoneau G. Peak triceps surae muscle activity is not specific to knee flexion angles during MVIC. *J Electromyogr Kinesiol*. 2011;21:819-826.

35. Hebert-Losier K, Willis S, Holmberg H. The reproducability of three different indicators of fatigue from plantar-flexion isokinetic testing at two knee flexion angles is not sufficient to be termed "acceptable". *Isokinet Exerc Sci.* 2013;21:227-236.

36. Hebert-Losier k, Holmberg H. Biomechanics of the heel-raise test performed on an incline in two knee flexion positions. *Clin Biomech*. 2013;28(6):664-671.

37. Hutchison AM, Evans R, Bodger O, et al. What is the best clinical test for Achilles tendinopathy? *J Foot Ankle Surg*. 2013;19(2):112-117.

38. Impellizzeri FM, Bizzini M, Rampinini E, Cereda F, Maffiuletti NA. Reliability of isokinetic strength imbalance ratios measured using the cybex NORM dynamometer. *Clin Physiol Funct Imaging*. 2008;28(2):113-119.

39. Kayser R, Mahlfeld K, Heyde C. Partial rupture of the proximal Achilles tendon : A differential diagnostic problem in ultrasound imaging. *Br J Sports Med.* 2005;39(11):838-842.

40. Kelly C, Al-Uzri M, O'Neill S. Effect of eccentric training on isokinetic endurance of calf .with reliability testing. *Br J Sports Med*. 2014;48(A32).

41. Kelly C, O'Neill S. *Reliability of isokinetic dynamometry to test endurance of the plantarflexors.* [Intercalated BSc]. University of Leicester ; 2013.

42. Khan K, Forster B, Robinson J, et al. Are ultrasound and magnetic resonance imaging of value in the assessment of Achilles tendon disorders? A two year prospective study. *Br J SPorts Med*. 2003;37(2):149-153.

43. Komi P. Measurement of the force-velocity relationship in human muscle under concentric and eccentric contractions. *Biomechanics III*. 1973;8:224-229.

44. Kujala UM, Sarna S, Kaprio J. Cumulative incidence of achilles tendon rupture and tendinopathy in former male elite athletes. *Clin J Sports Med* 2005(15):133-135.

45. Kulmala J, Korhonen M, Ruggiero L, et al. Walking and running require greater effort from the ankle than the knee extensor muscles. *Med Sci Sports Exerc.* 2016;48(11):2181-2189.

46. Landin D, Thompson M, Reid M. Knee and ankle joint angles influence the plantarflexion torque of the gastrocnemius. *J Clin Med Res*. 2015;7(8):602-606.

47. Lauber B, Lichtwark GA, Cresswell AG. Reciprocal activation of gastrocnemius and soleus motor units is associated with fascicle length change during knee flexion. *Physiol Rep*. 2014;2(6):10.14814/phy2.12044. Print 2014 Jun 1.

48. Lenhart RL, Francis CA, Lenz AL, Thelen DG. Empirical evaluation of gastrocnemius and soleus function during walking. *J Biomech*. 2014;47(12):2969-2974.

49. Lenhat R, Francis C, Lenz C, Thelen D. Empirical evaluation of gastrocnemius and soleus function during walking. *J Biomech*. 2014:2969-2974.

50. Maffulli N, Kenward M, Testa V, Capasso G, Regine R, King J. Clinical diagnosis of Achilles tendinopathy with tendinosis. *Clin J SPorts Med*. 2003;13(1):11-15.

51. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on

patients with chronic Achilles tendinosis. *Knee Surg Sports Traumatol Arthrosc*. 2001;9(1):42-47.

52. Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P. Intrinsic risk factors for the development of achilles tendon overuse injury: A prospective study. *Am J Sports Med*. 2006;34(2):226-235.

53. Malliaras P, O'Neill S. Potential risk factors leading to tendinopathy. *Apunts. Medicina de l'Esport*. 2017;52(194):71-77.

54. Malliaras P, Barton C, Reeves N, Langberg H. Achilles and patellar tendinopathy loading programmes : A systematic review comparing clinical outcomes and identifying potential mechanisms for effectiveness. *Sports Med*. 2013;43(4):267-286.

55. Masci L, Spang C, Schie H, Alfredson H. How to diagnose plantaris tendon involvement in midportion achilles tendinopathy- clinical and imaging findings. *BMC Musculoskeletal Disorders*. 2016.

56. McCrory J, Martin D, Lowery R, et al. Etiologic factors associated with achilles tendinitis in runners, *Med Sci SPorts Exerc*. 1999;31:1374-1381.

57. Moller M, Lind K, Styf J, Karlsson J. The reliability of isokinetic testing of the ankle and a heel-raise test for endurance. *Knee Surg Sports Traumatol, Arthros*. 2005;13:60-71.

58. Moore I. *Running self-optimisation: Acute and short-term adaptations to running mechanics and running economy.* [PhD]. ; 2013.

59. MORRIS C, Buchner DM, De Lateur B, Cress ME, Wagner EH. Isokinetic testing of ankle strength in older adults: Assessment of inter-rater reliability and stability of strength over six months. *Arch Phys Med Rehabil*. 1994;75:1213-1217.

60. Neptune R, Kautz S, Zajac F. Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. *J Biomech*. 2001;34(11):1387-1398.

61. Nicol AM, McCurdie I, Etherington J. Use of ultrasound to identify chronic achilles tendinosis in an active asymptomatic population. *J R Army Med Corps*. 2006;152(4):212-216.

62. O'Neill S, Watson P, Barry S. A delphi study of risk factors for Achilles tendinopathy- opinions of world tendon experts. *In J Sports Phys*. 2016;11(5):684-697.

63. O'Neill S, Watson P, Barry S. Why are eccentric exercises effective for Achilles tendinopathy? *Int J Sports Phys* 2015;10(4):552-562.

64. Plinsinga M, Brink M, Vicenzino B, Wilgen P. Evidence of nervous system sensitization in commonly presenting and persistant painful tendinopathies: A systematic review. *JJ Ortho Sports Phys Ther*. 2015(1):34.

65. Reid D, McNair PJ, Johnson S, Potts G, Witvrouw E, Mahieu N. Electromyographic analysis of an eccentric calf muscle exercise in persons with and without Achilles tendinopathy. *Phys Ther Sport*. 2012;13(3):150-155.

66. Reiman M, Burgi C, Strube E, et al. The utility of clinical measures for the diagnosis of achilles tendon injuries: A systematic review with meta-analysis. *J Athl Train*. 2014;49(6):820-829.

67. Richards PJ, Dheer AK, McCall IM. Achilles tendon (TA) size and power doppler ultrasound (PD) changes compared to MRI: A preliminary observational study. *Clin Radiol*. 2001;56(10):843-850.

68. Rio E, Kidgell D, Purdam C, et al. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. *Br J Sports Med*. 2015;49(19):1277-1283.

69. Robinson JM, Cook JL, Purdam C, et al. The VISA-A questionnaire: A valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med*. 2001;35(5):335-341.

70. Silbernagel K, Gustavsson A, Thomee R, Karlsson J. Evaluation of lower leg function in patients with Achilles tendinopathy. *Knee Surg Sports Traumatol Arthros.* 2006;14:1207-1217.

71. Silbernagel KG, Thomee 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.

72. Silbernagel KG, Thomee R, Thomee P, Karlsson J. Eccentric overload training for patients with chronic achilles tendon pain--a randomised controlled study with reliability testing of the evaluation methods. *Scand J Med Sci Sports*. 2001;11(4):197-206.

73. Slane L, Thelen D. Achilles tendon displacement patterns during passive stretch and eccentric loading are altered in middle-aged adults. *Med Eng Phys.* 2015;11(42):1-5.

74. Slane L, Thelen D. Non-uniform displacements within the achilles tendon observed during passive and eccentric loading. *J Biomech.* 2014;47:2831-2835.

75. Syha R, Peters M, Birnesser H, et al. Computer-based quantification of the mean achilles tendon thickness in ultrasound images: Effect of tendinosis. *Br J Sports Med*. 2007;41(12):897-902.

76. Szaro P, Witkowski G, Smigelskil R, Krajewski P, Ciszek B. Fascicles of the adult human achilles tendon – an anatomical study. *Ann Anat.* 2009;191:586-593.

77. Thorpe CT, Birch HL, Clegg PD, Screen HR. The role of the non-collagenous matrix in tendon function. *Int J Exp Pathol*. 2013;94(4):248-259.

78. Thorpe CT, Godinho MS, Riley GP, Birch HL, Clegg PD, Screen HR. The interfascicular matrix enables fascicle sliding and recovery in tendon, and behaves more elastically in energy storing tendons. *J mech behav biomed mat*. 2015;52:85-94.

79. Tompra N, Van Dieen J, Coppieters M. Central pain processing is altered in people with achilles tendinopathy. *Br J Sports Med*. 2016;50(16):1004-1007.

80. Wakahara T, Kanehisa H, Kawakami Y, Fukunaga T. Effects of knee joint angle on the fascicle behaviour of the gastrocnemius muscle during eccentric plantar flexions. *J Electromyogr Kinesiol.* 2009;19:980-987.

81. Warren G, Call J, Farthing A, Baadom-Piaro B. Minimal evidence for a secondary loss of strength after an acute muscle injury: A systematic review and meta-analysis. *Sports Med.* 2016;epub:1-19.

82. Webborn N, Morrissey D, Sarvananthan K, Chan O. Acute tear of the fascia cruris at the attachment to the achilles tendon: A new diagnosis. *Br J Sports Med*. 2015;49(21):1398-1403.

83. Wennerberg D. Reliability of an isokinetic dorsiflexion and plantar flexion apparatus. *Am J Sports Med*. 1991;19(5):519-522.

84. Wickiewicz T, Roy R, Powell P, Edgerton R. Muscle architecture of the human lower limb. *Clin Orthop Relat Res.* 1983;179(275):283.

85. Wyndow N, Cowan SM, Wrigley TV, Crossley KM. Triceps surae activation is altered in male runners with achilles tendinopathy. *J Electromyogr Kinesiol*. 2013;23(1):166-172.

Highlights

1. There are large statistically and clinically meaningful differences in plantar flexor strength and endurance between subjects with and without Achilles tendinopathy.
2. Rehabilitation should take into account the specific strength and endurance deficits associated with Achilles tendinopathy.
3. Our data shows healthy runners generate eccentric torque of twice bodyweight, In order to rehabilitate subjects with Achilles tendinopathy to this level substantial external loads will be required.
4. Researchers and clinicians cannot use the non-symptomatic limb as a comparator when assessing strength and endurance, instead normative values need to be considered.

What is known:

We know that changes in plantar flexor force capacity occur in the presence of tendinopathy but we do not know which of the plantar flexors are most affected and how these strength deficits relate to healthy controls. Neither do we understand the effects of tendinopathy on endurance capacity of the plantar flexors.

What this study adds to the literature

This study is the largest study examining force deficits of the plantar flexors between subjects with and without Achilles tendinopathy and it is the first study to examine endurance of the plantar flexors using isokinetic dynamometry. This is the first study to compare healthy subjects to injured subjects, the first to identify that plantar flexor deficits appear to relate to the soleus rather than the gastrocnemius and the first to identify endurance differences between symptomatic limbs and asymptomatic limbs. This study helps us to understand the extent of the force capacity changes associated with Achilles tendinopathy and gives the clinician and researcher a basis for clinical interventions/further research.

Plantarflexor strength and endurance deficits associated with mid-portion Achilles tendinopathy: The role of Soleus

Ethical disclosure:

Ethics approval was sought and granted from the University of Leicester Ethics committee. Approval numbers and information is reported in the manuscript.