Achilles Tendinopathy Has an Aberrant Strain Response to Eccentric Exercise

NICOLE L. GRIGG[1], SCOTT C. WEARING[2,3], and JAMES E. SMEATHERS[1]

[1]Institute of Health and Biomedical Innovation, Queensland University of Technology, Queensland, AUSTRALIA; and [2]Faculty of Health Sciences and Medicine, Bond University, Queensland, AUSTRALIA; and [3]Centre of Excellence for Applied Sport Science Research, Queensland Academy of Sport, Queensland, AUSTRALIA

ABSTRACT

GRIGG, N. L., S. C. WEARING, and J. E. SMEATHERS. Achilles Tendinopathy Has an Aberrant Strain Response to Eccentric Exercise. *Med. Sci. Sports Exerc.*, Vol. 44, No. 1, pp. 12–17, 2012. **Purpose**: Eccentric exercise has become the treatment of choice for Achilles tendinopathy. However, little is known about the acute response of tendons to eccentric exercise or the mechanisms underlying its clinical benefit. This research evaluated the sonographic characteristics and acute anteroposterior (AP) strain response of control (healthy), asymptomatic, and symptomatic Achilles tendons to eccentric exercise. **Methods**: Eleven male adults with unilateral midportion Achilles tendinopathy and nine control male adults without tendinopathy participated in the research. Sagittal sonograms of the Achilles tendon were acquired immediately before and after completion of a common eccentric rehabilitation exercise protocol and again 24 h later. Tendon thickness, echogenicity, and AP strain were determined 40 mm proximal to the calcaneal insertion. **Results**: Compared with the control tendon, both the asymptomatic and symptomatic tendons were thicker ($P < 0.05$) and hypoechoic ($P < 0.05$) at baseline. All tendons decreased in thickness immediately after eccentric exercise ($P < 0.05$). The symptomatic tendon was characterized by a significantly lower AP strain response to eccentric exercise compared with both the asymptomatic and control tendons ($P < 0.05$). AP strains did not differ in the control and asymptomatic tendons. For all tendons, preexercise thickness was restored 24 h after exercise completion. **Conclusions**: These observations support the concept that Achilles tendinopathy is a bilateral or systemic process and structural changes associated with symptomatic tendinopathy alter fluid movement within the tendon matrix. Altered fluid movement may disrupt remodeling and homeostatic processes and represents a plausible mechanism underlying the progression of tendinopathy.
Key Words: TENDON, REHABILITATION, ULTRASOUND, FLUID FLOW, BIOMECHANICS, CONDITIONING

Eccentric exercise has become the treatment of choice for Achilles tendinopathy (23). However, little is known about the acute response of tendons to eccentric exercise or the mechanisms underlying its beneficial effect. Previous research has demonstrated that eccentric exercise results in an immediate decrease in tendon thickness in healthy young adults (9). The marked anteroposterior (AP) strain response (≈20%) observed *in vivo* (9) was largely thought to reflect radial extrusion of fluid from the tendon (16,32). Such fluid movement likely plays an important homeostatic role by enhancing the penetration of relatively large solutes, in the order of 40 kDa, into the moderately avascular tendinous tissue (6,11) and may represent a biologically plausible mechanism by which eccentric exercise promotes tendon healing. To date, however, the AP strain response of tendons to eccentric exercise has only been examined in participants without tendinopathy. It is imperative to determine the AP strain response in individuals with Achilles tendinopathy because the degenerative process is associated with structural changes of the tendon matrix that may restrict fluid movement with the application of tensile load. Changes such as collagen fibril disorganization and thinning (14,18) would, in theory, affect the extension and realignment of collagen with loading, whereas an increase in the concentration of hydrophilic glycosaminoglycans (14,18) may decrease the proportion of free water within the tendon (4). As such, fluid movement may be restricted in the presence of tendinopathy.

The aim of the current research, therefore, was to evaluate the sonographic characteristics and acute AP strain response of control (healthy), asymptomatic, and symptomatic Achilles tendons to an eccentric exercise protocol that is widely used in the treatment of Achilles tendinopathy.

Address for correspondence: Nicole L. Grigg, Ph.D., Institute of Health and Biomedical Innovation, Queensland University of Technology, 60 Musk Avenue, Kelvin Grove, Queensland 4059, Australia; E-mail: nicole.grigg@qut.edu.au.
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METHODS

Participants. Eleven male adults with unilateral Achilles tendinopathy and nine control male adults participated in

the research. Participants with Achilles tendinopathy recorded a mean score of 62 ± 4 on the Victorian Institute of Sport Assessment–Achilles questionnaire, where a score of 0 represents the worst symptoms possible and a score of 100 represents no symptoms (25). Median symptom duration was 10 months and ranged between 2 and 30 months. Inclusion criteria for Achilles tendinopathy participants were pain and a sensation of stiffness particularly at the onset of loading, palpable focal thickening of the Achilles tendon 20–60 mm proximal to the calcaneal insertion, and unilateral symptoms of greater than 6 wk in duration (8). Volunteers presenting with insertional tendinopathy, bilateral symptoms, or a history of Achilles tendon surgery, rupture, partial tear, or calf muscle injury were excluded. Nine of the 11 tendinopathy participants were individually matched to nine control participants by age, height, and mass. Volunteers were excluded from the control group if they presented with a history of Achilles tendon pain or calf muscle injury. The study received university human research ethics approval, and participants provided written informed consent in accordance with the regulations.

Procedure. All participants were required to refrain from physical activity, in excess of that required to perform necessary daily tasks, 24 h before and throughout the study period. Exercise sessions were scheduled to start at approximately 6:00 a.m. Participants were requested to minimize their activity on these mornings, and to aid this process, participants drove to and were provided with a parking lot at the test location. Each participant with Achilles tendinopathy performed the testing protocol twice, such that both the symptomatic and asymptomatic limbs performed isolated eccentric exercise on separate occasions. Allocation of the asymptomatic or symptomatic limb to eccentric exercise in the first or second testing session was counterbalanced across participants. Participants with tendinopathy were requested to refrain from seeking treatment or performing prescribed rehabilitation exercises in the week before testing, the days between testing sessions, and during the 24-h study periods. Control participants performed the testing process once with the allocation of left or right limbs to eccentric exercise counterbalanced.

Exercise protocol. The exercise protocol was based on a widely implemented therapeutic program for Achilles tendinopathy, first described by Alfredson et al. (1), in which the triceps surae muscle tendon unit of the affected limb is eccentrically loaded. For eccentric loading, participants were required to stand with their forefoot positioned on the edge of a 105-mm-high step with their ankle maximally plantar flexed. Eccentric loading occurred as the participant lowered the heel below the level of the forefoot to a position of maximal dorsiflexion. No concentric loading followed. Rather, the forefoot of the previously non–weight-bearing contralateral limb was placed on the step. All weight was then transferred to this limb, which was used to return the body to the starting position. The process was repeated 15 times per exercise set. Consistent with the clinical protocol cited by Alfredson et al. (1), three exercise sets were performed with a straight knee, and three were performed with the knee slightly flexed. To standardize knee position, participants performed the exercise protocol wearing a postoperative knee brace (DonJoy TROM; DJO, LLC, Vista, CA), which effectively immobilized the knee at 0° and 20° of flexion. The exercise protocol was performed without footwear.

Kinematics. The orientations of lower limb segments were recorded by an 11-camera motion analysis system sampling at 200 Hz (Vicon, Oxford Metrics Group, Oxford, United Kingdom). The Plug-In Gait (SCAR) model within Vicon Nexus (version 1.4.116; Vicon) was used to model the lower body as seven rigid segments (pelvis, right and left upper leg, lower leg, and foot segments) (29). The model required the attachment of 15 passive markers (∅ 14 mm) to the participant in accordance with the modified Helen Hayes marker system. Markers were attached bilaterally to the dorsum of the second metatarsal head, lateral malleolus, posterior superior calcaneus, midtibia, lateral femoral condyle, midthigh, and anterior superior iliac spine (ASIS). A marker was also positioned on the midpoint of the sacrum. Anthropometric inputs for the model included body height measured to the nearest millimeter using a stadiometer, body mass measured to the nearest gram with clinical scales (Tanita BWB-600; Wedderburn, Queensland, Australia), leg length (ASIS to the medial malleolus) measured to the nearest millimeter using a tape measure while the participant was standing, and knee width (mediolateral width perpendicular to the line of knee axis) and ankle width (mediolateral distance across the malleoli) (29) both measured to the nearest millimeter using anthropometric calipers while the participant was seated, with his/her knee flexed to 90° and ankle neutral (0° of dorsiflexion).

Sonographic imaging. Sonographic images of the Achilles tendon were acquired immediately before (PRE), immediately (within 5 min) upon completion of the eccentric exercise protocol (IMMPOST), and again 24 h later (24 h POST). Images were obtained in B-mode, at a frequency of 8 MHz, using a linear-array transducer (LOGIQ Book XP; GE Healthcare, Wauwatosa, WI). All images were collected by a single operator, experienced in musculoskeletal imaging, using standardized ultrasound settings and an acoustic standoff pad. Images were collected with the participant prone and the plantar surface of the foot positioned perpendicular to the tibia (0° of dorsiflexion). Sonographic imaging was conducted with the transducer attached to a FaroArm (FARO Technologies, Inc., Lake Mary, FL), which provided global three-dimensional coordinates for the transducer. Initially, a sagittal image was acquired with the calcaneal insertion positioned at the center of the field of view. Using the FaroArm coordinates, the probe was translated proximally by 40 mm, and sagittal images of the tendon were acquired at this location. Thus, the center of the image corresponded to a standard reference point on the Achilles tendon, 40 mm proximal to the

tendon insertion. At each time interval (PRE, IMMPOST, and 24 h POST), three images were collected for analysis.

Kinematic analysis. All marker displacements and joint kinematics were calculated by and exported from the Vicon Nexus. Kinematic data were segmented using MATLAB software (version 7.6.0.324 R2008a; The MathWorks, Inc., Natick, MA) such that the 15 eccentric repetitions of each exercise set were isolated for analysis. The vertical displacement of the second metatarsal head was used to identify the positioning of the foot on the step, whereas the vertical displacement of the calcaneus was used to identify the beginning and end of loading. The duration of eccentric loading was calculated using the number of samples recorded for each repetition and the known sampling frequency. The maximum and minimum sagittal ankle joint angles and ankle joint range of motion (ROM) were determined for each exercise repetition. For each variable, the 15 repetitions were averaged to provide a mean value for each exercise set.

Sonographic image analysis. Sonographic images were exported in Digital Imaging and Communications in Medicine format and postprocessed using MATLAB software. The anterior and posterior edges of the Achilles tendon were manually digitized at the center of each image, with the aid of a grayscale profile. The axial image resolution was 0.19 mm, and the pixel resolution was 0.07 mm. Each of the three images captured at each time point were digitized on two separate occasions, and the average of the two measures was used for further analysis. The true AP strain (ε) of the Achilles tendon was calculated using the following equation:

$$\varepsilon = \left(\ln\left(\frac{L}{L_0}\right)\right) \times 100 \quad [1]$$

where L_0 represents PRE AP tendon thickness and L represents the AP tendon thickness at either the IMMPOST or the 24 h POST time points. The 95% limits of agreement for repeated measures of tendon AP strain were ±4.6%. Tendon echogenicity was estimated by calculating the mean grayscale value (arbitrary units [U]) for the area encompassed by the anterior and posterior boarders of the tendon and five pixels proximal and distal to the measurement site. High-average grayscale values are representative of tendon hyperechogenicity, whereas low values depict hypoechogenicity (white = 225, black = 0). The 95% limits of agreement for repeated measures of tendon echogenicity were ±12.9 U.

Statistical analysis. The Statistical Package for the Social Sciences (version 17; SPSS, Inc., Chicago, IL) was used for all statistical procedures. The age, height, and mass characteristics of the Achilles tendinopathy group were contrasted with those of the control group using a one-way ANOVA.

TABLE 1. Mean ± SE age, height, and mass of control and tendinopathy participants.

	Control	Tendinopathy
Age (yr)	48.2 ± 3.8	49.0 ± 4.5
Height (cm)	181.6 ± 2.0	180.6 ± 2.2
Mass (kg)	97.3 ± 6.9	92.6 ± 5.6

TABLE 2. Mean ± SE Achilles tendon sagittal thickness and echogenicity before exercise.

	Control	Asymptomatic	Symptomatic
Thickness (mm)	4.96 ± 0.42	6.56 ± 0.36*	9.37 ± 0.36***
Echogenicity (U)	119.3 ± 4.5	82.9 ± 3.9*	76.7 ± 3.9*

* Significantly different from control limb ($P < 0.05$).
** Significantly different from asymptomatic limb ($P < 0.05$).

Because of the matching process, the control group was not independent of the tendinopathy group. To overcome this violation of a general linear modeling assumption, a variable "pair" was created to pair the data from the Achilles tendinopathy participant with those of the matched control. Given that the pairs of participants were measured on multiple occasions and therefore not independent, linear mixed models were used for all further analyses, in which pair and time were modeled as random effects. Although two tendinopathy participants were not matched with control participants, this did not affect the ability of the linear mixed models to calculate between-group differences because these differences were calculated on the basis of the mean and associated variance of the groups. Linear mixed models were used to 1) compare tendon thickness and echogenicity at baseline (PRE) across the three tendon categories (control, asymptomatic, and symptomatic), 2) evaluate the effects of time of sonographic examination (IMMPOST or 24 h POST) and tendon category on AP strain and echogenicity, and 3) investigate the effect of tendon category on load duration, maximum and minimum sagittal ankle joint angles, and ankle joint ROM. For each linear mixed model, the underlying assumption, normality of residual variance, was met. Invariably, significant main effects were investigated using custom hypothesis tests within the models, whereas significant interactions were investigated using the pairwise comparisons and 95% confidence intervals. Estimated marginal means and SE calculated by the linear mixed models are presented in the text. The relationships between AP strain and baseline tendon thickness and echogenicity, load duration, and kinematic variables were investigated via scatterplots and simple linear regression.

RESULTS

Demographic details of the two participant groups are presented in Table 1. There was no statistically significant difference in mean age ($P > 0.05$), height ($P > 0.05$), or mass ($P > 0.05$) of the control and Achilles tendinopathy groups.

Table 2 summarizes the sonographic appearance of tendons before eccentric exercise. The symptomatic tendon was thicker than the asymptomatic tendon ($P < 0.05$), which, in

TABLE 3. Mean ± SE load duration and kinematic characteristics of eccentric exercise.

	Control	Asymptomatic	Symptomatic
Load duration (s)	0.93 ± 0.04	0.93 ± 0.03	1.14 ± 0.03***
Peak dorsiflexion (°)	22.1 ± 1.3	13.1 ± 1.2*	12.9 ± 1.1*
Peak plantarflexion (°)	−10.1 ± 1.2	−14.6 ± 1.2	−17.4 ± 1.0*
Ankle ROM (°)	32.2 ± 0.7	27.7 ± 0.7*	30.3 ± 0.6**

* Significantly different from control limb ($P < 0.05$).
** Significantly different from asymptomatic limb ($P < 0.05$).

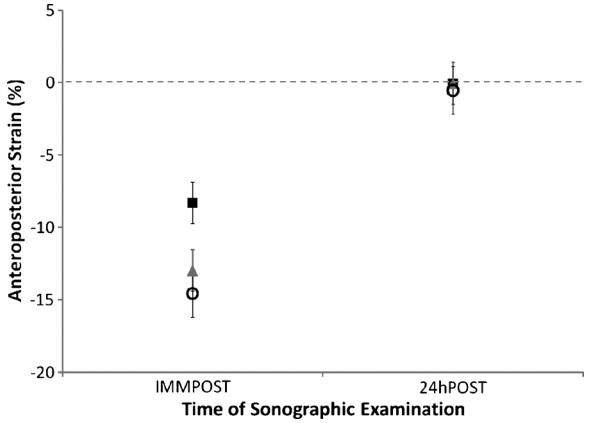

FIGURE 1—Mean AP strain in control (○), asymptomatic (▲), and symptomatic (■;) Achilles tendons after eccentric exercise. ***Error bars*** **represent 95% confidence intervals.**

turn, was thicker than the control tendon ($P < 0.05$). Both the symptomatic and asymptomatic tendons were hypoechoic compared with the control tendon ($P < 0.05$).

Table 3 demonstrates the duration of loading and kinematic characteristics of the limbs during eccentric exercise. The symptomatic limb was characterized by a significantly longer loading duration, higher peak plantarflexion angle, and lower peak dorsiflexion angle compared with the control limb ($P < 0.05$). Ankle joint ROM was not different between the symptomatic and control limbs ($P > 0.05$). The asymptomatic limb demonstrated a similar load duration and peak plantarflexion angle compared with the control limb, although the dorsiflexion angle achieved by the asymptomatic limb was significantly reduced ($P < 0.05$) compared with the control limb, leading to a significantly smaller ROM ($P < 0.05$).

Eccentric exercise resulted in AP strains of −14.6%, −13.0%, and −8.3% for the control, asymptomatic, and symptomatic tendons, respectively (Fig. 1). The symptomatic tendon was characterized by a significantly lower AP strain when compared with the pain-free counterparts ($P < 0.05$). AP strains were not significantly different in the control and asymptomatic tendons ($P > 0.05$). All tendons returned to preexercise dimensions (zero strain) within 24 h (Fig. 1). No tendon category demonstrated a significant change in echogenicity after eccentric exercise. AP strain was not significantly related to tendon thickness ($R^2 = 0.05$, $P > 0.05$) or echogenicity ($R^2 = 0.01$, $P > 0.05$) at baseline, load duration ($R^2 = 0.01$, $P > 0.05$), peak plantarflexion ($R^2 = 0.03$, $P > 0.05$), or peak dorsiflexion ($R^2 = 0.02$, $P > 0.05$).

DISCUSSION

The purpose of this research was to evaluate the sonographic characteristics and acute AP strain response of control (healthy), asymptomatic, and symptomatic Achilles tendons to eccentric exercise. Consistent with previous research (17,26), the symptomatic tendon was thicker (89%) and hypoechoic (36%) compared with the control tendon at baseline. The thickness of the symptomatic tendon (9.37 mm) within the current research, however, was greater than values previously reported (range = 5.6–7.6 mm) (12,17). The reason for the discrepancy in symptomatic tendon thickness between studies is difficult to determine. Although it could be hypothesized that greater thickness may be related to greater symptom severity and/or duration, the average Victorian Institute of Sport Assessment-Achilles score reported by participants in the current study (62) was similar to that of a previous study (56) in which a lower symptomatic tendon thickness (5.6 ± 1.1 mm) was reported (17). Another potential reason for the discrepancy in symptomatic tendon thickness may be related to differences in measurement procedures or measurement error; however, the thickness of the control tendon (4.96 mm) in the current study was comparable to that reported for a "normal" tendon within the literature (4.4 and 5.2 mm) (12,17), suggesting this was not the case.

Consistent with previous research (9), all tendons demonstrated an immediate decrease in tendon thickness in response to eccentric exercise. However, the symptomatic tendon was characterized by a reduced AP strain response compared with the asymptomatic and control tendons. Although the reduced AP strain suggests that the symptomatic tendon may respond abnormally to tensile load, the difference in AP strain response between tendons must be considered with respect to the Achilles tendon loading conditions. The duration of loading was significantly longer in the symptomatic limb and was accompanied by an increase in ankle joint plantarflexion and concomitant decrease in dorsiflexion, compared with the control limb. It is possible, therefore, that these factors combined to alter the total loading impulse experienced by the symptomatic tendon during the exercise. However, as the tendon is a viscoelastic structure, the greater load duration experienced by the symptomatic tendon would be expected to result in a greater, rather than reduced, AP strain, as has been shown *in vitro* (16). Although a reduced AP strain in the symptomatic tendon may have resulted from the shift in ankle joint ROM toward the plantar-flexed end of the range, on the basis of previous research (19), we estimate that a 7° increase in plantarflexion (as noted in the symptomatic limb) would result in a 2.5-mm increase in the Achilles tendon moment arm and only lower the tendon force by about 3.9%. The duration of loading for the symptomatic limb, in contrast, was 18.4% greater than that for the control limb. Therefore, the total loading impulse is estimated to be greater for the symptomatic limb, and the AP strain response of the symptomatic tendon would be expected to be greater than that of the control tendon, which was not the case. Interestingly, neither load duration nor any kinematic parameter was significantly correlated with AP strain, suggesting that an altered loading pattern was not responsible for the reduced strain response observed in the symptomatic tendon.

We hypothesize, therefore, that the reduced AP strain characteristic of the symptomatic tendon reflects the change in

tendon structure associated with tendinopathy. It has been previously hypothesized that a negative AP strain response of tendon to tensile load largely represents movement of fluid out of the tendon due to a reduction in the interfibrillar space associated with the straightening and extension of collagen fibrils (9,16,31,32). Collagen fibril thinning and disorganization, typically observed in tendinopathic tissue (14,18), may reduce the realignment and extension of collagen fibrils under load, thereby minimizing fluid movement. Similarly, the elevated glycosaminoglycan levels commonly associated with tendinopathy (18,24) likely further reduce fluid movement within the tendon by increasing the proportion of water bound to macromolecules and reducing the amount of free water available to be moved by collagen realignment (4). Whereas there is evidence that load-induced fluid movement does not necessarily influence the diffusion of small solutes, such as glucose, in the tendon (10), studies evaluating convective transport in cartilage and the vertebral disc suggest that fluid shifts induced by mechanical loading enhance the penetration of large solutes (in the order 40 kDa) (6,11). Given that many growth factors associated with the regulation of collagen synthesis are of this size (14,27), load-induced fluid movement likely plays an important role in remodeling and homeostatic processes. Consequently, altered fluid movement may represent a previously unidentified mechanism underlying tendinopathy. It is recommended that future research be directed toward exploring fluid movement, convective transport, and molecular perfusion in the development and progression of tendinopathy.

In the current study, the asymptomatic tendon was also observed to be significantly thicker (32%) and hypoechoic (30%) compared with the control tendon. The observation of bilateral thickening in unilateral tendinopathy raises the possibility that degenerative change in tendinopathy may be a bilateral or systemic process, as has been proposed in other "overuse" soft tissue injuries, such as plantar fasciitis (28,30). In support of this concept, degenerative change associated with tendinopathy has been hypothesized to proceed asymptomatically in the human tendon (13), and prospective studies have shown that as many as 45% of thickened Achilles tendons progress to develop clinical symptoms within 12 months (7), whereas 40% of individuals with unilateral Achilles tendinopathy developed symptoms in the contralateral limb (22). More recently, variations in the *COL5A1* and tenascin-C genes, both of which encode for structural components of the tendon, have been associated with Achilles tendinopathy (20,21), highlighting the potential for systemic involvement in degenerative tendinopathy. Alternatively, thickening and hypoechoic changes in symptomatic and asymptomatic tendons may represent a bilateral process. Animal models of tendinopathy, in which only one limb is exposed to repetitive loading, have also observed degenerative changes and thickening of the tendon bilaterally, suggesting that central neural mechanisms may be involved in the development of tendinopathic characteristics in the nonexercised contralateral limb (2). A further explanation for the observed change in the sonographic appearance of the asymptomatic tendon is that thickening represents a reaction to increased loading as a result of an antalgic gait response. Although a higher loading rate due to a stiffer landing strategy has been linked to patella tendinopathy (3), the current research did not investigate the kinetics or kinematics of the participants' gait. Moreover, given the cross-sectional nature of the current study, it is unclear as to whether the sonographic thickening and hypoechogenicity observed bilaterally with Achilles tendinopathy preceded or followed the onset of symptoms. Further, prognostic research into the onset and natural progression of Achilles tendinopathy is required to ascertain the relationship between clinical symptoms and sonographic signs of Achilles tendinopathy.

Although the asymptomatic tendon demonstrated sonographic signs of degenerative change (thickening and hypoechogenicity), the AP strain response did not differ from that of the control tendon. Given that AP strain was not significantly correlated with tendon thickness or echogenicity at baseline, it would seem, therefore, that tendon degeneration may need to advance to a level associated with symptoms before the strain response is affected. This finding is consistent with the proposed "iceberg theory," wherein tendon degeneration occurs along a continuum with the clinical symptom of pain occurring only with end-stage degenerative change of the tendon substance (8). Moreover, the lack of correlation between AP strain and measures of thickness and echogenicity at baseline suggests that baseline measures of tendon thickness and echogenicity are poor predictors of a tendon's dynamic response to exercise. Given that measures of tendon thickness and echogenicity have also been shown to be poor predictors of clinical outcome (5,7), it is possible that more dynamic measures of the response of a tendon to a given stimulus (in this case, the AP strain response to exercise) may represent a more sensitive indicator of clinical outcome. Further research designed to prospectively monitor the AP strain response of the Achilles tendon with the resolution of clinical symptoms is required to determine the clinical utility of measures of AP strain.

The current research is limited by many factors that should be considered when interpreting the results. First, the investigation used an eccentric loading protocol that is commonly used in a clinical setting, and as such, no attempt was made to standardize the peak plantarflexion and dorsiflexion angles or the load duration across participants. Whereas only minor variations in the effective Achilles tendon moment arm were noted between limbs in the current study, there were larger discrepancies in the duration of loading. Future research may benefit from using prescribed ankle joint angles and duration of load to ensure a more consistent loading impulse. Second, tendon strains were determined in one dimension only. Although animal models have suggested that the Achilles tendon is transversely isotropic (15), the current study did not monitor the mediolateral strain response of the tendon. Therefore, it is unclear

if the mediolateral strain response is similar to the observed AP strain response.

This is the first study to show that, in comparison with a healthy tendon, symptomatic Achilles tendinopathy is associated with a reduced AP strain response to eccentric exercise. This finding suggests that structural changes associated with symptomatic tendinopathy inhibit fluid movement within the tendon matrix. Given that altered fluid movement may disrupt homeostatic processes and represent a plausible mechanism underlying the progression of tendinopathy, the AP strain response of the tendon to exercise may be a more sensitive indicator of clinical progression in tendinopathy than traditional measures of tendon thickness and echogenicity. Nevertheless, bilateral signs of altered tendon structure in unilaterally symptomatic cases lend further support to the notion that Achilles tendinopathy may be systemic or bilateral in nature.

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