Tendinopathy alters mechanical and material properties of the Achilles tendon

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Arya S, Kulig K. Tendinopathy alters mechanical and material properties of the Achilles tendon. J Appl Physiol 108: 670–675, 2010. First published November 5, 2009; doi:10.1152/japplphysiol.00259.2009.—The purpose of this study was to investigate the in vivo material and mechanical properties of the human Achilles tendon in the presence of tendinopathy. Real-time ultrasound imaging and dynamometry were used to assess Achilles tendon stiffness, Young’s modulus, stress, strain, and cross-sectional area (CSA) in 12 individuals with Achilles tendinopathy and 12 age- and gender-matched controls. The results of this study suggest that tendinopathy weakens the mechanical and material properties of the tendon. Tendinopathic tendons had greater CSA, lower tendon stiffness, and lower Young’s modulus. These alterations in mechanical characteristics may put the Achilles tendon at a higher risk to sustain further injury and prolong the time to recovery. Results from this study may be used to design treatment strategies that specifically target these deficits, leading to faster and permanent recovery from tendinopathy.

Young’s modulus; tendon stiffness; ultrasound imaging; dynamometry

THE ACHILLES TENDON is the strongest tendon in the human body, subject to substantial loads reaching up to 12.5 times body weight during running (12). However, the high magnitude of loads on the Achilles tendon and the continual stresses placed on it during locomotion also make it one of the most common tendons to sustain overuse injuries and ruptures. Spontaneous tendon ruptures have been postulated to be end-stage manifestations of subclinical, chronic degenerative changes within the tendon tissue (18, 32). Degenerative disorders of the Achilles tendon, described as tendinopathies, cause considerable morbidity and functional impairment among the athletic and general populations (15, 29). Achilles tendinopathy remains a clinically challenging condition notoriously recalcitrant to current treatment protocols. Despite its high prevalence and therapeutically challenging nature, the pathomechanics of Achilles tendinopathy remain largely unknown.

The morphological changes accompanying Achilles tendinopathy have been well documented (16, 18). Previous studies (3, 11) have shown that tendinopathy results in a tendon with larger cross-sectional area (CSA) with histological evidence of increased ground substance, hypercellularity, and collagen fiber disruption. These changes in tendon morphology and composition may influence the mechanical characteristics (stiffness) and material properties (Young’s modulus) of the tendon. It is well known that the mechanical properties of tendons are directly related to the geometric arrangement of collagen fibers constituting the tendon (31). Therefore, it is reasonable to expect that disruption of the collagen fiber structure and arrangement, accompanied by an increase in mechanically weaker type III collagen as a result of tendinopathy, may alter the tendon’s mechanical and material properties (33).

Although it has been suggested that Achilles tendinopathy results in a mechanically weaker tendon predisposed to ruptures, this hypothesis has not been empirically tested (3). Conversely, a study by Gisslen et al. (6) concluded that tendinopathy does not produce a mechanically weaker tendon. However, “mechanical strength” of the tendon was assessed via performance of a jumping activity, not taking into account possible compensations by other muscle groups, and was limited to results based on a single individual. Thus conclusive evidence of alterations in mechanical characteristics of the tendon in response to pathology remains at best limited.

Altered mechanical characteristics of the tendon in the presence of tendinopathy have important implications for functional performance and predisposition to further injury. The mechanical traits of the Achilles tendon are primarily responsible for its ability to withstand large muscular forces with minimal deformation. Stiffness, an important constituent of tendon mechanical properties, is the ratio of force applied to the tendon and its elongation in response to the force. It has a significant influence on force transmission, muscle power, and energy absorption and release during locomotion (4, 22, 28). An optimal level of tendon stiffness is critical for effective muscle-tendon interactions and for minimizing the energetic costs of locomotion.

Tendon stiffness may be influenced by tendon length and CSA. A shorter tendon with larger CSA is expected to have greater stiffness. However, this relation between tendon morphology and stiffness remains ambiguous (26). Young’s modulus (stiffness normalized to tendon CSA and length) provides a measure of tendon material properties irrespective of its geometric characteristics. This is especially important in the case of pathologic tendons, as pathologic tendons usually present with greater CSA. Thus this study investigated both tendon stiffness and Young’s modulus in individuals with Achilles tendinopathy.

The role of tendon mechanical characteristics in optimizing tendon function, combined with the high incidence of Achilles tendon injuries and their unresponsiveness to current treatments, is a compelling factor underscoring the need to investigate mechanical and material properties of tendons in the presence of pathology. Therefore, this study was done to investigate alterations in in vivo human Achilles tendon stiffness and Young’s modulus in response to tendinopathy. We hypothesized that individuals with chronic Achilles tendinopathy will demonstrate lower tendon stiffness and Young’s modulus compared with age- and gender-matched controls.

METHODS

Subjects

Twenty-four subjects participated in this study. Sample size was determined via power analysis done on preliminary data. An effect
size of 3.94 was calculated for tendon stiffness based on an α-level of 0.05 and power values of 0.8. Subjects were divided into two groups (experimental and age- and gender-matched control group) based on the presence of Achilles tendinopathy confirmed via clinical examination and ultrasound imaging. The physical characteristics for the subjects are given in Table 1. Only male subjects were recruited for this study because of the higher incidence of Achilles tendon injuries in males compared with females and to eliminate any confounding effects of gender on tendon mechanical characteristics (7, 13).

The following criteria were used to determine inclusion of subjects in the experimental group: 1) history of intermittent episodes of Achilles tendon pain lasting more than 6 consecutive weeks within the past 5 yr; 2) more than one episode of tendon pain exacerbation and remission within the past 5 yr; 3) palpable focal thickening of the Achilles tendon in the midsubstance; 4) pain originating from the Achilles tendon on palpation of thickened Achilles tendon; and 5) sonographic evidence of tendinopathy, i.e., focal thickening and hypoechoicity (Fig. 1), consistent with previously reported sonographic characteristics diagnostic of tendinopathy.

Individuals with Achilles tendinopathy were excluded from the study if they reported any of the following: 1) history of previous surgery or tears involving the Achilles tendon; 2) systemic diseases affecting collagenous tissue; 3) insertional Achilles tendinopathy, calcaneal spurs, plantar fasciitis, and other conditions affecting the foot and ankle complex; and 4) active stage of tendinopathy at the time of data collection with moderate to severe pain in the Achilles tendon affecting functional activities like walking.

Control subjects had the same exclusion criteria as subjects in the experimental group. Additionally, they were excluded from the study if they had any past or present Achilles tendon pain. They were matched by age and gender to individuals in the experimental group.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Control (n = 12)</th>
<th>Tendinopathy (n = 12)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>44.83 ± 7.2</td>
<td>47.33 ± 8.3</td>
<td>0.44</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.76 ± 0.09</td>
<td>1.73 ± 0.09</td>
<td>0.71</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>77.97 ± 9.70</td>
<td>86.01 ± 9.62</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Values are means ± SD

Procedure

All data collection was done in a single session at the Musculoskeletal Biomechanics Research Laboratory at the University of Southern California. Before testing, the procedures, risks, and benefits of the study were explained and informed consent, approved by the local institutional review board (University of Southern California, Health Sciences Campus) was obtained from all subjects. Data were collected on the side of tendinopathy for the experimental group and the same side was matched for testing in the control group.

Static ultrasound images. Subjects lay prone on a test table with hip and knee extended and the ankle in neutral position (i.e., 90° angle between the foot and the shank segments). To obtain tendon length, the ultrasound transducer was placed longitudinally over the posterior aspect of the heel. The most distal part of the Achilles tendon attaching to the calcaneous was imaged, and the corresponding point was marked on the skin with a pen. The ultrasound transducer was then moved proximally to view the musculotendinous junction of the medial gastrocnemius muscle. This point was also marked on the skin. The distance between the two points was measured with a caliper. This distance represents the resting length of the tendon.

To obtain CSA measurements, the ultrasound transducer was placed perpendicular to the tendon. Three transverse images were taken at distances of 2, 4, and 6 cm proximal to the tendon insertion on the calcaneous.

Dynamometry. Before performing dynamometric testing, the subjects were instrumented for EMG analysis. The subjects’ skin was shaved and rubbed gently with sandpaper and alcohol to reduce skin impedance. Surface electrodes were taped over the muscle bellies of tibialis anterior and medial gastrocnemius muscles, parallel to the direction of the muscle fibers. The ground electrode was placed on the lateral femoral epicondyle of the contralateral leg. EMG data were recorded simultaneously from the two antagonistic muscles during the ramped isometric plantar-flexion and dorsiflexion contractions.

All subjects performed the isometric contractions in a prone position on the Kin-Com table with hip and knee extended and the foot in neutral position. The axis of rotation of the dynamometer was aligned perpendicular to the tendon. Three transverse images were taken at distances of 2.5, 4.5, and 6.5 cm proximal to the tendon insertion on the calcaneous.

Subjects were asked to perform a slow ramped isometric contraction to maximum isometric plantar flexion over a 5-s period. The rate of the ramp was controlled using a metronome set at 60 bpm. Three submaximal contractions and two maximal contractions were performed before commencing the trial. Thereafter, subjects were instructed to perform three maximal plantarflexion contractions. A 1-min rest period was given between each plantarflexion contraction.

Following the plantar-flexion contractions, subjects were asked to perform a ramped maximal isometric dorsiflexion contraction. This trial was used to account for the coactivation of the antagonist muscle (tibialis anterior muscle) during the plantar-flexion contraction.

Lastly, the ankle joint was rotated passively in the sagittal plane through a 10° rotation from 5° of ankle dorsiflexion to 5° of ankle plantarflexion. Absence of EMG activity was used to ensure a true passive trial. The elongation of the Achilles tendon was measured simultaneously during the passive trial and used to calculate Achilles tendon moment arm.

Tendon elongation. Elongation of the tendon during the isometric plantarflexion contraction and passive ankle joint rotation was recorded simultaneously through dynamic ultrasound imaging at 30 Hz. The ultrasound transducer was positioned manually over the medial gastrocnemius myotendinous junction. A thin wire was used as a skin

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marker and placed between the transducer and the skin. This marker produced a hypoechoic signal on the ultrasound image and was used to ensure no movement of the transducer on the skin during the contraction.

Data Analysis

Tendon cross-sectional area. The ultrasound images were stored digitally as JPEG images and processed on a PC using a public domain NIH Image program (http://rsb.info.nih.gov/nih-image/). A freehand selection tool was used to outline the tendon and measure the CSA at each of the three sites. The average of these three values was taken to represent the CSA of the tendon.

EMG processing. All EMG data were analyzed using MATLAB software (The Mathworks). A zero-lag, 2nd order Butterworth bandpass filter (20–500 Hz) was applied to EMG signals using MATLAB software. The EMG data were further filtered digitally with a notch filter at 60 Hz to eliminate any 60-Hz electrical noise. The filtered EMG signals were full wave rectified and smoothed using a root mean square algorithm over a 300-ms window.

Electrical activity of the tibialis anterior muscle during the plantarflexion trial was normalized to the maximum activity of the tibialis anterior muscle during the maximal isometric dorsiflexion trial. A torque-EMG relationship was established for the tibialis anterior muscle using a linear fit equation applied to the dorsiflexion torque and tibialis anterior EMG activity recorded during the maximal isometric dorsiflexion trial. The slope of the curve-EMG relationship and added to the net plantar flexion torque to get a true estimate of the plantar-flexion torque.

Tendon moment arm. The plantarflexion torque measured from the dynamometer is based on the external moment arm of the dynamometer. To calculate tendon force, the external torque must be divided by the internal moment arm of the Achilles tendon.

The internal moment arm was determined via the tendon excursion method as described by Maganaris and Paul (21). The ankle joint was rotated passively through a 10° rotation (dθ) around the neutral position of the ankle. Absence of medial gastrocnemius and tibialis anterior EMG activity was used to ensure a true passive trial. Tendon position of the ankle. Absence of medial gastrocnemius and tibialis anterior EMG activity recorded during the maximal isometric dorsiflexion trial. The torque contribution of the tibialis anterior muscle during the maximal isometric dorsiflexion trial was determined using the torque-EMG relationship and added to the net plantar flexion torque to get a true estimate of the plantar-flexion torque.

Tendon stiffness, stress, strain, and Young’s modulus. The slope of the resultant tendon force and tendon elongation curve in the last 40% of the linear region was calculated to obtain a measure of tendon stiffness. Tendon strain was calculated by dividing the elongation of the tendon during the maximal plantar-flexion contraction with the resting length of the tendon. Tendon stress was obtained by dividing the calculated tendon force with the average CSA of the tendon. The slope of the stress-strain curve in the last 40% of the linear region was calculated to obtain a measure of Young’s modulus.

Statistical Analysis

Differences in tendon stiffness, strain, stress, and Young’s modulus between the two groups were determined using an independent sample t-test. Test retest reliability was assessed for tendon thickness and elongation measures and peak isometric plantar flexion force on five individuals on 2 separate days 1 wk apart. Repeatability of these measures was evaluated using a two-way random effects intraclass correlation coefficient model, i.e., ICC(2,1). All statistical analyses
RESULTS

Reliability analysis demonstrated high test-retest reliability for Achilles tendon thickness (ICC = 0.99) and tendon elongation (ICC = 0.99) measures using ultrasound imaging and peak isometric force (ICC = 0.80) measured dynamometrically.

Group ensemble curves for force-elongation and stress-strain are shown in Figs. 2 and 3. Stiffness values calculated from the linear slope of the last 40% of force-elongation curve demonstrate a statistically significant difference between the two groups (tendinopathy group: 277.50 ± 81.87 MPa; control: 308.50 ± 50.73 MPa). Similarly, analysis of Young’s Modulus data calculated from the linear slope of the last 40% of the stress-strain curve demonstrates significantly lower values for the tendinopathy group (818.72 ± 217.03 MPa) compared with the controls (1,671.02 ± 277.50 MPa).

Achilles tendon force, elongation, stress, strain, CSA, resting length, stiffness, and Young’s Modulus for both groups are presented in Table 2. Peak tendon force data indicate a non-significant difference between the two groups (tendinopathy group: 1,934.79 ± 308.50 N; controls: 2,258.26 ± 507.96 N), with the tendinopathy group demonstrating 14% lower force values compared with controls. Peak elongation of the Achilles at maximal tendon force was statistically significantly different (tendinopathy group: 12.72 ± 1.06 mm; controls: 11.01 ± 0.8 mm) between the two groups. The tendinopathy group demonstrated significantly higher average CSA measures (92.90 ± 14.11 mm²) compared with controls (56.23 ± 6.18 mm²). Similarly, analysis of Young’s Modulus data calculated from the linear slope of the last 40% of the stress-strain curve demonstrates significantly lower values for the tendinopathy group (818.72 ± 217.03 MPa) compared with the controls (1,671.02 ± 277.50 MPa).

Stiffness, N/mm 375.25
Elongation, mm 11.96
Force, N 2,258.26
Resting length, mm 11.38
Strain, % 0.57
Young’s modulus, MPa 377.25

DISCUSSION

This study provides evidence that tendinopathy weakens the mechanical characteristics and alters the material properties of the human Achilles tendon. Previous investigations (2, 8) of morphological and biomechanical characteristics of degenerated Achilles tendons have primarily been done using an animal model. However, these studies failed to demonstrate any changes in tendon morphology or mechanics despite sub-

jection of the tendon to extensive repetitive loading. Additionally, most in vivo evaluations of tendon mechanics have been limited to adaptations of healthy tendons in response to varying stimuli such as aging, exercise, and immobility (5, 14, 30). Hence, there is still a considerable debate in current literature whether tendinopathy results in a mechanically weaker tendon. This was the first study to evaluate the in vivo biomechanics of degenerated human Achilles tendons and provide evidence for diminished tendon mechanical and material properties in the presence of tendinopathy.

Morphological comparisons of tendinopathic and healthy tendons demonstrated a larger CSA for the degenerated Achilles tendon. This increase in CSA is due to an accumulation of water and increased ground substance as a result of the pathology (19). Typically, a larger tendon is considered mechanically stronger due to its ability to dissipate high stresses (force/area) across the tendon and yield lower strain energy. However, the present study demonstrated that despite having a larger CSA, the degenerated tendon had a lower stiffness and Young’s modulus compared with healthy tendons. This is most likely a result of alterations in tendon tissue composition and structure accompanying the degenerative process, thereby subjecting the tendon to higher strains despite having a larger CSA. Changes in tendon matrix include separation and loss of type I collagen fibers, loss of transverse bands of collagen fibers, increased collagen fiber crimping and ruptures, and increased production of mechanically weaker type III collagen (27). All of these structural and compositional changes may result in a tendon with lower mechanical and material properties.

Table 2. Achilles tendon mechanical, material, and morphological property values for control and tendinopathy groups

<table>
<thead>
<tr>
<th>Property</th>
<th>Control</th>
<th>Tendinopathy</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Force, N</td>
<td>2,258.26 ± 507.96</td>
<td>1,934.79 ± 308.50</td>
<td>0.073</td>
</tr>
<tr>
<td>Elongation, mm</td>
<td>11.01 ± 0.86</td>
<td>12.72 ± 1.06</td>
<td>0.000*</td>
</tr>
<tr>
<td>Stiffness, N/mm</td>
<td>375.25 ± 61.88</td>
<td>300.37 ± 37.60</td>
<td>0.002*</td>
</tr>
<tr>
<td>CSA, mm²</td>
<td>56.23 ± 5.57</td>
<td>92.90 ± 14.11</td>
<td>0.000*</td>
</tr>
<tr>
<td>Resting length, mm</td>
<td>11.96 ± 1.06</td>
<td>12.72 ± 1.06</td>
<td>0.397</td>
</tr>
<tr>
<td>Stress, MPa</td>
<td>40.28 ± 6.62</td>
<td>21.43 ± 5.78</td>
<td>0.000*</td>
</tr>
<tr>
<td>Strain, %</td>
<td>4.36 ± 0.31</td>
<td>5.14 ± 0.57</td>
<td>0.001*</td>
</tr>
<tr>
<td>Young’s modulus, MPa</td>
<td>1,671.02 ± 277.50</td>
<td>818.72 ± 217.03</td>
<td>0.000*</td>
</tr>
</tbody>
</table>

Values are means ± SD and depict group averages of data averaged across 3 isometric plantar-flexion trials for each individual. See METHODS for calculations. CSA, cross-sectional area. *Significant group differences (P ≤ 0.05).
Lower tendon stiffness in case of degenerated tendons, as evidenced by this study, will cause the muscle fascicles to shorten more to take up the excess compliance in the tendons. This may limit the muscle’s ability to function within the optimal region of force-length curve, thereby affecting movement economy. Lower tendon stiffness may also affect the force transmission capabilities of the tendon and thus negatively impact rate of force development and power generation at the limb segments. Over a period of time, these mechanical alterations can compound to have deleterious effects on functional performance. A less stiff tendon is subjected to higher strains, potentially resulting in microscopic disruptions of collagen fibers. Such microtrauma may accumulate over time, making the tendon vulnerable to further injury and potentially predisposing the tendon to the danger of rupture.

The Achilles tendon strain, stress, and Young’s modulus values for the control group calculated in this study are within range of previous studies (23, 25) investigating tendon mechanical properties in healthy tendons using ultrasound imaging. The tendon force and elongation values in this study were relatively lower than those reported by previous studies (1, 22). The lower force and elongation values may be a result of differences in subject position during maximal isometric plantarflexion. This study chose a prone position for testing, while Muraoka et al. (26) and Arampatzis et al. (1) chose sitting as their testing position. Additionally, this study used two foot switches to ensure absence of movement at the foot-shoe and shoe-footplate interface during the isometric contractions, whereas other studies (22, 24) measured foot movement during the isometric contractions and subsequently subtracted the elongation of the tendon due to passive rotation of the foot from the total elongation measured during the active isometric contraction. These authors argued that inability to account for tendon elongation due to ankle rotation during isometric contractions will lead to an overestimation of tendon displacement values. The foot switches used in our study ensured that no movement took place at the heel and may have led to lower plantar flexion moment produced at the ankle joint. The lower force values probably led to lower elongation values as well. The absence of heel movement, combined with the fact that our elongation values were lower than those reported in previous literature, indicates that our experimentation did not overestimate the amount of tendon elongation.

The methodology detailed in this study is well established in previously published literature (1, 21, 23) but is not without certain limitations. Dynamometric measurements of torque during plantarflexion assume that the triceps-surae muscle-tendon complex works completely in the sagittal plane. This assumption may have led to an underestimation of the calculated tendon force. Similarly, ultrasound imaging is also twodimensional and does not take into account any tendon displacement in the transverse plane. Additionally, although all attempts were made to prevent foot motion during the isometric plantarflexion contraction, it is impossible to eliminate motion between the rear and fore foot and motion due to multiple midfoot articulations and soft tissue deformation. However, the amount of motion resulting from these factors is relatively small and consistent between groups. Finally, accurate tendon moment arm values are necessary to estimate tendon force values. This study used the tendon excursion method to estimate moment arms, but changes in moment arm values from rest to maximal isometric contractions could not be taken into account with this methodology (9, 20).

Despite these limitations, the present study provides important evidence regarding the in vivo mechanical properties of tendons under physiologic loads. Tendons are biological structures capable of adapting to changes in mechanical stimuli through alterations in structural and mechanical properties. Recent investigations (17) on the effects of sclerosing injections on the healing of degenerated tendon tissue demonstrate that tendinopathic changes in tendons may be reversible. Furthermore, the beneficial effects of eccentric loading protocols on abatement of symptoms from Achilles tendinopathy have also been demonstrated recently (10). However, the rationale for the benefits gained from such interventions is poorly understood and lacks scientific evidence. Results of this study, delineating the changes in tendon properties as a result of tendinopathy, provide an important first step toward gaining a better understanding of the treatment strategies employed in such conditions. A clear understanding of the mechanical characteristics of tendons in the presence of pathology will enable further investigation into the effect of various current treatment strategies of tendinopathies on the mechanical characteristics of the tendon. Tendons can adapt to mechanical stimuli, thus creating a potential for rehabilitative regimes to address specific mechanical deficits and promote healing.

GRANTS

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DISCLOSURES

No conflicts of interest are declared by the author(s).

REFERENCES