

Effects of Creep and Cyclic Loading on the Mechanical Properties and Failure of Human Achilles Tendons

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Abstract—The Achilles tendon is one of the most frequently injured tendons in humans, and yet the mechanisms underlying its injury are not well understood. This study examines the *ex vivo* mechanical behavior of excised human Achilles tendons to elucidate the relationships between mechanical loading and Achilles tendon injury. Eighteen tendons underwent creep testing at constant stresses from 35 to 75 MPa. Another 25 tendons underwent sinusoidal cyclic loading at 1 Hz between a minimum stress of 10 MPa and maximum stresses of 30–80 MPa. For the creep specimens, there was no significant relationship between applied stress and time to failure, but time to failure decreased exponentially with increasing initial strain (strain when target stress is first reached) and decreasing failure strain. For the cyclically loaded specimens, secant modulus decreased and cyclic energy dissipation increased over time. Time and cycles to failure decreased exponentially with increasing applied stress, increasing initial strain (peak strain from first loading cycle), and decreasing failure strain. For both creep and cyclic loading, initial strain was the best predictor of time or cycles to failure, supporting the hypothesis that strain is the primary mechanical parameter governing tendon damage accumulation and injury. The cyclically loaded specimens failed faster than would be expected if only time-dependent damage occurred, suggesting that repetitive loading also contributes to Achilles tendon injuries. © 2003 Biomedical Engineering Society. [DOI: 10.1114/1.1569267]

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INTRODUCTION

The Achilles tendon is among the most frequently injured tendons in the human body.^{7,9} It often experiences overuse injuries such as tendinitis and tendinosis.⁵ These injuries occur when repetitive loading creates damage in the tendon faster than it can be repaired.⁵ Repetitive loading may also contribute to traumatic injuries such as ruptures and partial tears. In most cases, ruptures occur only after damage has accumulated in the tendon, causing degeneration which predisposes the ten-

don to failure.⁹ Achilles tendon injuries therefore result from functional demands that create excessive damage through sustained or repetitive loading. In this study, we examine the effects of prolonged (creep) and repetitive (cyclic) loading on the mechanical properties and failure of human Achilles tendons *ex vivo*.

Two sets of investigators have previously studied the *ex vivo* behavior of tendons under creep and/or cyclic loading. Wang *et al.*^{20,21} studied creep and cyclic loading of wallaby tail tendons, and Schechtman and Bader^{17,18} examined cyclic loading of human extensor digitorum longus (EDL) tendons. Time and cycles to failure decreased exponentially with increasing stress for the wallaby tail tendons under both creep²⁰ and cyclic²¹ loading. Cycles to failure also decreased exponentially with increasing stress for the cyclically loaded human EDL tendons.^{17,18}

Both sets of investigators found stiffness or modulus ratios to be useful measures of damage accumulation in tendons. These ratios are calculated as the stiffness or modulus following a period of loading divided by the stiffness or modulus when loading was first imposed. Wang and colleagues defined their ratio in terms of the secant slope of the force versus displacement curve for individual loading cycles.^{20,21} For the creep tests, loading cycles were interposed at specified intervals during the test to allow for stiffness measurements.²⁰ The stiffness ratio decreased at an increasing rate as the cyclic tests progressed. During the creep tests, the stiffness ratio was proportional to the yield stress of damaged tendons and therefore served as a useful indicator of diminished tendon strength. Schechtman and Bader defined damage ratios in terms of the dynamic and storage moduli determined by their dynamic characterization tests.¹⁸ They found a linear relationship between damage ratios for these moduli and both the tangent moduli and ultimate tensile stresses of partially damaged tendons.

While it is well accepted that cyclic mechanical loading contributes to tendon injury, the specific mechanical stimuli driving damage accumulation and injury have not

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yet been determined. We have previously shown that tensile strains may be the stimulus that most directly influences normal tendon development and functional adaptation.^{24,25} We hypothesize that tensile strains similarly govern damage accumulation in tendons and, consequently, tendon injury. Schechtman and Bader found that tendons cycled to failure all failed at approximately the same strain regardless of load magnitude and frequency.¹⁷ They also found that partially damaged tendons exhibited much less variation in failure strain than in modulus or failure stress. Wang and Ker observed a linear relationship between the stiffness ratio during creep and the strain sustained beyond that from the initial loading.²⁰ Strains may therefore govern changes in mechanical properties during creep and cyclic loading as well as the failure characteristics of both normal and damaged tendons.

In this study, we examine the mechanical behavior of excised human Achilles tendons undergoing creep and cyclic loading *ex vivo*. We examine time-dependent changes in the tendon's modulus and energy dissipation characteristics. We also look at possible predictors of time or cycles to failure and consider the relative contributions of time-dependent and cycle-dependent damage. Finally, we evaluate the hypothesis that tensile strain is the primary mechanical parameter governing Achilles tendon damage accumulation and injury.

METHODS

Forty-three fresh-frozen human Achilles tendons from 41 donors were procured from the International Institute for the Advancement of Medicine (Scranton, PA) and the Northern California Transplant Bank (San Rafael, CA). Each specimen was carefully dissected from its calcaneal insertion, and all soft tissue including the paratenon was removed from around the tendon.

High-frequency ultrasound was used to measure the cross-sectional area of each tendon. Previous studies have shown that this method of measurement has good accuracy with average error less than 5%.¹⁹ For each specimen, transverse ultrasound images were recorded at locations 2, 4, and 6 cm proximal to the tendon's insertion. National Institutes of Health Image was used to measure the tendon's cross-sectional area on each image, and the smallest area for each specimen (A_{\min}) was used in stress calculations.

Mechanical testing was performed using a servo-hydraulic materials testing system (MTS, Eden Prairie, MN) (Fig. 1). The ends of each specimen were gripped using insulated liquid nitrogen-cooled freeze clamps maintained at -15°C . Pilot tests showed that this temperature allows the insulated clamps to remain frozen without freezing the tendon between the clamps. At the distal end of the tendon, the specimen was gripped over

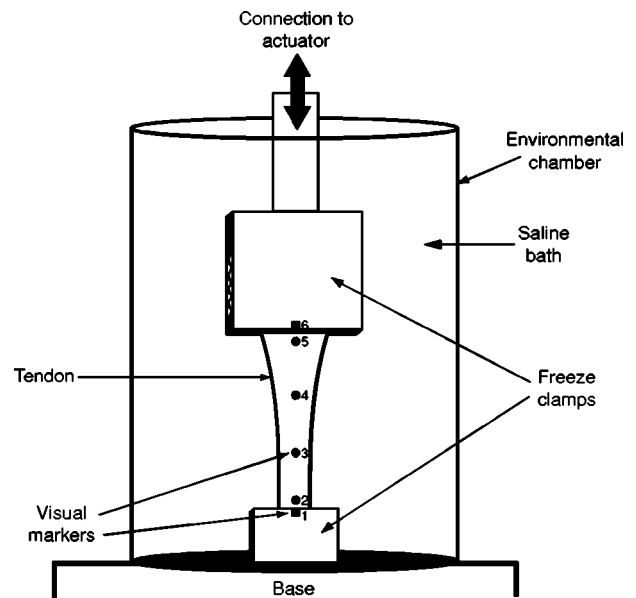


FIGURE 1. Experimental setup.

approximately 1 cm of the insertional region. At the proximal end, the specimen was gripped so that the initial grip-to-grip gage length (L_0) was approximately 10 cm. Throughout testing, the specimen was immersed in phosphate-buffered saline maintained at 37°C and circulated throughout the environmental chamber. Loads (F) and crosshead displacements (d) were recorded during testing by a computer attached to the materials testing system. Stress was calculated as applied load divided by the tendon's cross-sectional area ($\sigma = F/A_{\min}$). Nominal strain was calculated as displacement divided by initial gage length ($\varepsilon = d/L_0$).

Prior to the specimen's immersion in saline, four circular marks were made on the surface of the tendon using black India ink. The marks were approximately 3 mm in diameter and were placed along the centerline of the tendon adjacent to the clamps and at two evenly spaced intermediate points. Markers were also present at the edges of the clamps. The six visual markers were numbered 1–6 starting at the most distal marker (Fig. 1). A charge coupled device camera recorded movement of the markers during testing. The contrast between the markers and the light colored tendon and clamps allowed subsequent identification and location of the markers using MATLAB (Mathworks, Natick, MA) with an accuracy of approximately ± 0.25 mm (accuracy varies slightly depending on the field of view imaged for each specimen). These data were used to examine strains in subregions of the tendon. The regions are identified by their bounding markers, e.g., Region 1–2 represents the region between markers 1 and 2.

Creep testing was performed on 18 specimens, while 25 specimens underwent cyclic tests (Table 1). For the

TABLE 1. Specimen and donor information.

	Number of specimens	Age (yr) Mean \pm SD	Gender		Cross-sectional area (mm ²) Mean \pm SD
			% Male	% Female	
Creep	18	60 \pm 10	61	39	75 \pm 11
Cyclic	25	75 \pm 12	44	56	63 \pm 13

creep tests, two specimens were tested at each of the following stress levels: 35, 40, 45, 50, 55, 60, 65, 70, and 75 MPa. The stress was held constant at the specified level following a 2 s ramp increase in stress to that level. For the cyclic tests, sinusoidal loading was imposed at 1 Hz between a minimum stress of 10 MPa and a constant maximum stress. The maximum stresses were distributed across the 30–80 MPa range without preselection of specific stress levels.

For the cyclic tests, a secant modulus was calculated from the stress versus strain curve for each cycle. This modulus was defined as the slope of a line passing through the point with the lowest stress and the point with the highest stress during the loading phase of the cycle. The secant modulus from the first cycle was used to calculate normalized modulus values for each specimen. Cyclic energy dissipation was defined as the area between the loading and unloading stress versus strain curves and was calculated using the trapezoidal method.

For both the creep and cyclic tests, STATVIEW (SAS Institute Inc., Cary, NC) was used to perform linear regression of log (time or cycles to failure) versus peak stress, initial strain, and failure strain. For the creep tests, initial strain was defined as the strain when the target stress was first reached. For the cyclic tests, initial strain was defined as the peak strain from the first loading cycle. The creep data were used to predict the lifetime that would be expected for the cyclically loaded specimens if only time-dependent damage occurred (see the Appendix). These predictions were compared with the cyclic loading data to determine the contribution of time-dependent damage to tendon failure in the cyclic loading tests.

RESULTS

The creep specimens exhibited normal creep behavior with primary, secondary, and tertiary creep regions [Fig. 2(a)]. The cyclically loaded specimens exhibited similar behavior with an expanding cyclic strain range superimposed on a creep curve [Fig. 2(b)]. During the cyclic tests, the stress versus strain curves for individual loading cycles became less linear and less steep with increasing area of the hysteresis loop [Fig. 2(c)]. These changes reflect progressive extension of the toe region, reduction of modulus, and increasing cyclic energy dissipation (Fig. 3).

The most distal region (Region 1–2) experienced much higher strains than the other regions. Subfailure strains in this region were generally >20% and sometimes as high as 40%–50%. In the other regions, sub-

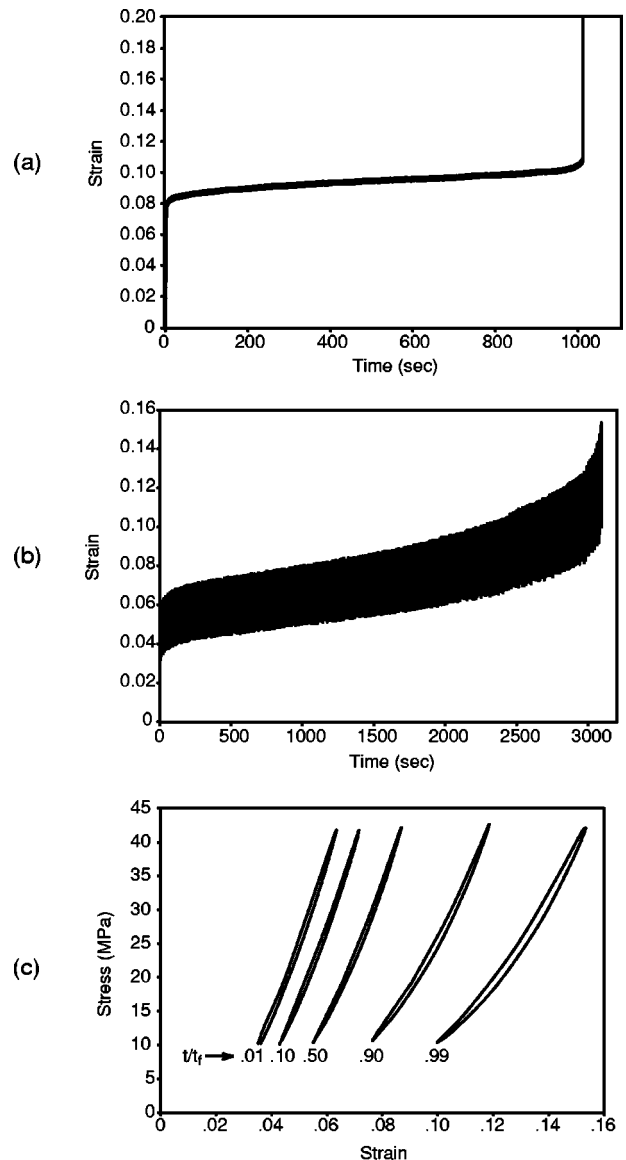


FIGURE 2. Typical results for: (a) time-dependent strains during a creep test, (b) time-dependent strains during a cyclic test, and (c) stress vs. strain cycles during a cyclic test. The cycles shown represent times (t) corresponding to 1%, 10%, 50%, 90%, and 99% of the specimen's lifetime (t_f).

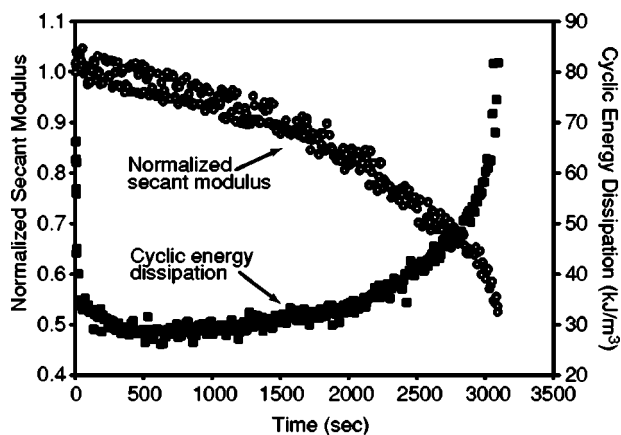


FIGURE 3. Normalized secant modulus and cyclic energy dissipation for a typical specimen undergoing cyclic testing.

failure strains were generally <10%. Several specimens had slightly higher strains in the 10%–15% range in the most proximal region (Region 5–6). Despite the high strains in Region 1–2, failure did not occur in this region.

Strains increased initially in all regions when loading was first applied. Following this initial increase, a gradual increase or no increase in strains was usually observed in all regions until the specimen was close to failure. As failure approached, most specimens experienced rapidly increasing strains in Region 2–3 and/or Region 3–4. Strains generally did not increase through failure in the other regions.

All failures occurred in the tendon midsubstance away from the clamps (Regions 2–3 and 3–4). It appeared that most of the failures began in the distal 4 cm of the tendon, although it was difficult to determine the exact location at which failure was initiated. Four creep specimens failed before reaching their preassigned stresses. For these specimens, results are given using the maximum stress achieved before failure. One creep specimen loaded to 35 MPa exceeded the available supply of liquid nitrogen without failing. This specimen is shown on the plot of applied stress versus time to failure [Fig. 4(a)] but was excluded from the data analyses.

For the creep tests, applied stress did not predict time to failure ($p=0.29$) [Fig. 4(a)]. Creep lifetime was best predicted by initial strain ($p=0.0008$) [Fig. 4(b)]. Failure strain also showed a significant relationship with creep lifetime ($p=0.03$) [Fig. 4(c)].

For the cyclic tests, there was a significant relationship between peak stress and time or cycles to failure ($p=0.002$) [Fig. 5(a)]. This relationship was consistent with the results of monotonic failure tests on ten additional specimens (unpublished data) assuming that the monotonic failures occur after half a cycle of loading [Fig. 5(a)]. As with the creep tests, initial peak strain was

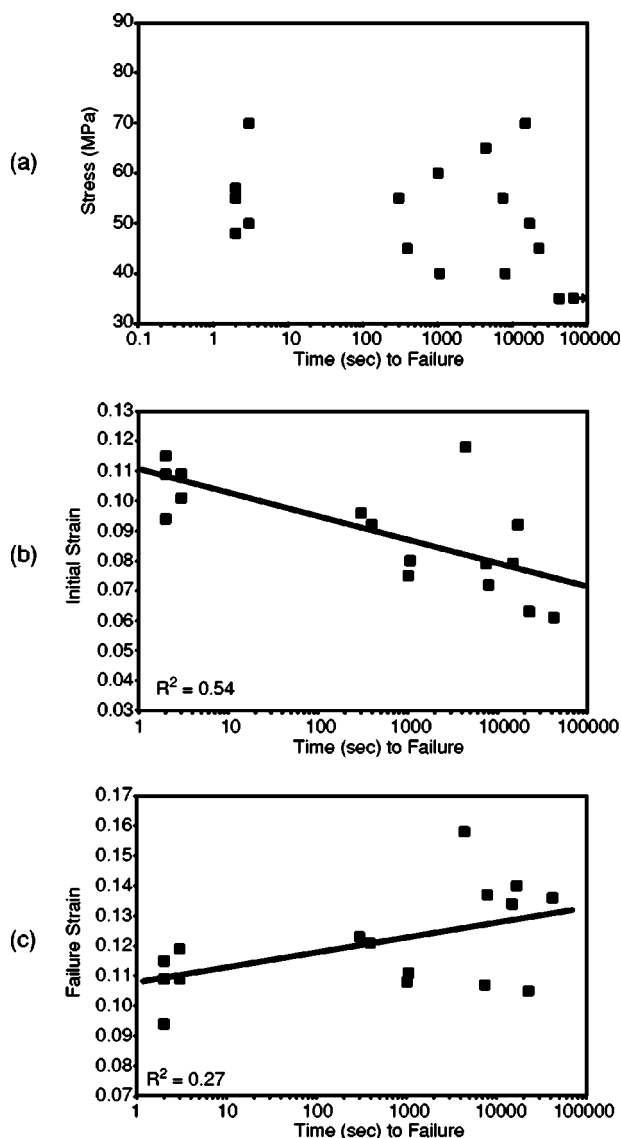


FIGURE 4. Creep lifetime results for: (a) applied stress, (b) initial strain, and (c) failure strain. Initial strain was the best predictor of creep lifetime. There was no significant relationship between applied stress and creep lifetime. Data point with arrow represents specimen that did not fail. This specimen was excluded from data analyses.

the best predictor of cyclic lifetime ($p<0.0001$) [Fig. 5(b)]. There was also a significant relationship between failure strain and cyclic lifetime ($p=0.02$) [Fig. 5(c)].

The cyclic loading results differed from what would be predicted if failure were due to time-dependent mechanisms alone (see the Appendix). Lifetime under cyclic loading is shorter than predicted with only time-dependent damage [Fig. 5(b)] suggesting that the cyclic nature of the loading accelerates failure.

DISCUSSION

This study examined the creep and cyclic behavior of a clinically important tendon, the human Achilles tendon.

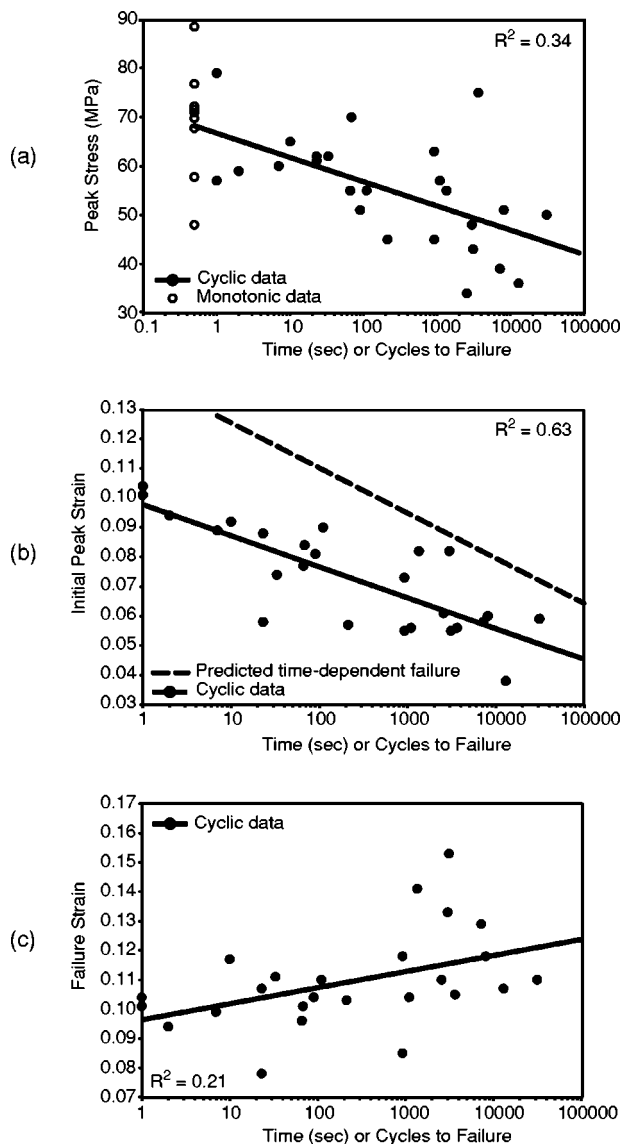


FIGURE 5. Cyclic lifetime results for (a) peak stress, (b) initial peak strain, and (c) failure strain. Initial peak strain was the best predictor of cyclic lifetime. Cyclic lifetime was shorter than predicted for exclusively time-dependent damage.

This tendon poses a greater challenge for mechanical testing than the tendons previously tested by other investigators, namely, the human extensor digitorum longus^{17,18} and wallaby tail tendons.^{20,21} First, only two Achilles tendons are available from each donor compared with a potential of eight EDL and 60 wallaby tail tendons. Because of the high variability of tendons among individuals, this restriction likely increased the scatter of our data, although it may also make our results more applicable to the general population. In addition, because of the Achilles tendon's large size and low length-to-width ratio, it is difficult to position the tendon exactly in line with the axis of loading to assure uniform

loading and measurement of maximal mechanical properties. The Achilles tendon also has a less uniform cross section than the other tendons, making the stresses and strains variable along the length of the tendon.

Most of our results are presented using nominal strains based on the entire length of tendon tested (~10 cm). This was done for two reasons. First, the strain distributions varied among tendons, and it was difficult to determine the location at which failure began. Second, since accuracy of the measurement system is constant with respect to displacement, the global strains are more accurate than regional strains calculated using much smaller initial lengths. The regional strains are used primarily to understand the patterns of strain distribution along the length of the tendon. Strains usually increased significantly in the distal 4 cm of the tendon immediately prior to failure. This region has a smaller cross-sectional area than the rest of the tendon, and it corresponds with the location of most *in vivo* ruptures.⁶ Much higher axial strains were observed in the insertional region. While these strains could have been affected by the gripping technique, other studies using bone-tendon preparations have reported similar results.^{22,26} It appears that, for reasons yet unknown, the insertional region is able to withstand higher strains than the rest of the tendon without failing.

As in the studies by Wang *et al.*²¹ and Schechtman and Bader,¹⁷ we found that tendon lifetime under cyclic loading decreases exponentially as the applied stress increases. We also found that both creep and cyclic lifetime decrease exponentially with increases in the initial strain. This suggests that low strains create relatively little damage consistent with the finding of Provenzano *et al.*¹⁶ that strains below 5% caused no lasting structural damage in rat medial collateral ligaments.

Because the cyclic loading applied to tendons during mechanical testing does not have a mean stress of zero as required for pure fatigue, tendon "fatigue" tests can include both time-dependent creep as well as cycle-dependent fatigue damage components. Comparison of our creep and cyclic loading results suggests that significant damage occurred through both mechanisms. Unfortunately, we did not control for donor age and gender, which differed between the creep and cyclic loading groups. It is therefore possible that the older age or higher proportion of females (with smaller tendon cross-sectional areas) in the cyclic loading group contributed to that group's accelerated failure. However, using similar methods Wang *et al.* also found that both creep and fatigue damage mechanisms affect tendon lifetime.²¹ While idealized creep and cyclic loading may not reflect the full complexity of *in vivo* loading, investigation of these simple loading patterns provides insight into the likely contributions of prolonged and repetitive loading in tendon damage accumulation and injury.

In many ways, the human Achilles tendon behaves like other tendons. We reported previously that this tendon has material properties consistent with the properties of other tendons.²⁶ In this study, we observed creep and cyclic behavior similar to the behavior reported for wallaby tail tendons.^{20,21} While immersion in saline causes swelling, which increases tendon viscoelasticity,⁴ the general behavior should be similar for this common testing environment and the liquid paraffin environment used in the wallaby tail tendon studies.^{20,21} We also observed a progressive and accelerating decrease in secant modulus during cyclic testing similar to that reported for wallaby tail tendons.^{20,21} As the modulus decreased, cyclic energy dissipation increased. This behavior has been reported previously for bone,¹⁵ but to our knowledge it has not been addressed previously for tendon. The decreased modulus and increased cyclic energy dissipation may serve as indicators of tendon damage.

Although the human Achilles tendon has material properties similar to other tendons, it likely experiences higher *in vivo* stresses. While most tendons experience peak stresses below 30 MPa, peak stresses in the human Achilles tendon may approach 70 MPa during isometric triceps surae contraction.^{10,12,13} Even higher stresses may be achieved with dynamic and eccentric loading, as indicated by buckle transducer studies that have reported Achilles tendon stresses up to 111 MPa during running.¹¹ These stresses exceed the failure stresses of many of our specimens, possibly due to younger subject age or methodological issues, but it is clear that the Achilles tendon experiences high *in vivo* stresses. In the absence of a correspondingly high modulus, the Achilles tendon would be expected to experience high strains. In this study, we observed initial strains in the 4%–12% range for applied stresses of 30–80 MPa. Recent ultrasound studies have also measured Achilles tendon strains over 5% for stresses around 40 MPa during maximal isometric contractions of the triceps surae.^{13,14} These strains are much higher than the 1.5%–3.0% strains expected for most tendons and ligaments.^{1,12,23} Higher strains likely create more damage in the tendon and may account for the high incidence of Achilles tendon injuries.

We have previously shown that tensile strain may be the mechanical stimulus that most directly influences normal tendon development and functional adaptation.^{24,25} We hypothesize that strains similarly influence tendon injury. Because physiologic and failure strains are well conserved during development and functional adaptation and across species and anatomic locations,²⁴ it seems probable that strains govern tendon development, adaptation, and injury. This hypothesis is supported by our finding that initial strain is the best predictor of time to failure for both creep and cyclic loading. Higher strains create more damage, leading to earlier failure. They should also, however, stimulate

functional adaptation which reduces strain magnitude by stiffening and strengthening the tendon. There may be limits to the adaptive capabilities of a tendon, however, due to physiologic and anatomic constraints on tendon size and collagen packing.²⁴ A secondary adaptive response may occur in the Achilles tendon in which extended lifetime is achieved by increasing failure strain.

Wang and colleagues^{20,21} and Schechtman and Bader¹⁸ both proposed indices of tendon damage based on stiffness or modulus ratios. They showed that these ratios change in proportion to the reductions in modulus, yield stress, and ultimate stress of partially damaged tendons. In light of our hypothesis that tendon adaptation and injury are most directly influenced by strains, however, we propose that strains may provide a more direct measure of tendon damage. Increased strains, in fact, reflect reductions in modulus as indicated by the linear relationship between stiffness ratio and “extra strain” reported by Wang and Ker.²⁰ Since noninvasive measurement of *in vivo* strains is now possible,^{12,14} strains may be used as a future indicator of tendon damage and risk of injury.

Clinically, tendons are most susceptible to injury when the cumulative damage from mechanical loading exceeds the reparative ability of the tendon.^{5,7,8} This is the case for acute ruptures as well as chronic overuse injuries since ruptures are almost always preceded by tendon degeneration.^{8,9} The current study helps elucidate the mechanisms of Achilles tendon injury by examining the damage created under well-defined, nonphysiologic loading protocols. An understanding of the damage response to these simple loading protocols provides a basis for understanding the effects of more complex physiologic loading. Additional studies would be needed to determine how variable load magnitudes, different loading rates, and rest periods affect the damage response. Additional studies are also needed to elucidate the role of biologic repair processes that this study has not attempted to address. To fully understand the mechanisms underlying Achilles tendon injury, all of these factors must be considered.

In summary, we have investigated the mechanical behavior of human Achilles tendons subject to creep and cyclic loading *ex vivo*. This tendon is of great clinical interest because it is frequently injured. The sustained and cyclic natures of *in vivo* loading both contribute to damage and subsequent injury of the tendon. The reduction in modulus and increase in cyclic energy dissipation during testing can serve as indicators of tendon damage. With initial strain being the best predictor of time or cycles to failure, however, we propose that strain is the mechanical parameter that most directly influences tendon damage accumulation and injury. Because the Achilles tendon is subject to high *in vivo* strains, it experiences a high incidence of injury.

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APPENDIX

Failure of a cyclically loaded structure can occur through time-dependent mechanisms, cycle-dependent mechanisms, or a combination of both. Since creep tests eliminate any contribution from cycle-dependent mechanisms, the results from creep tests can be used to predict time to failure for arbitrary loading profiles assuming that only time-dependent damage occurs.^{2,3}

We found in this study that creep lifetime can be related to initial strain through an exponential [Fig. 4(b)] such that the creep lifetime is

$$T_C = A e^{-B\varepsilon_{\text{init}}}, \quad (\text{A1})$$

where $\varepsilon_{\text{init}}$ is the initial strain and A and B are experimentally determined constants. We define a creep damage parameter D_C which is zero for undamaged tissue and one at failure ($0 \leq D_C \leq 1$). If damage occurs at a constant rate, the rate of damage accumulation during creep is

$$\frac{dD_C}{dt} = \frac{1}{A e^{-B\varepsilon_{\text{init}}}}. \quad (\text{A2})$$

Because our cyclic loading history is defined in terms of stress rather than strain, we must express the damage equation in terms of stress. We therefore define an initial modulus for each specimen:

$$E^* = \sigma_{\text{max}} / \varepsilon_{\text{init}}, \quad (\text{A3})$$

where σ_{max} is the peak cyclic or creep stress and $\varepsilon_{\text{init}}$ is the initial strain for the specimen. The rate of damage accumulation during creep is then

$$\frac{dD_C}{dt} = \frac{1}{A e^{-B\sigma_{\text{max}}/E^*}}. \quad (\text{A4})$$

For an arbitrary stress history, the cumulative creep damage can be calculated as

$$D_C(t) = \int_0^t \frac{dt}{A e^{-B\sigma/E^*}}. \quad (\text{A5})$$

At failure, $D_C = 1$ so that the time-dependent failure lifetime t_c can be determined from the equation

$$\int_0^{t_c} \frac{dt}{A e^{-B\sigma/E^*}} = 1. \quad (\text{A6})$$

The cyclic loading used in this study followed a sinusoidal profile with a frequency of 1 Hz. The time-dependent stress history can therefore be described as

$$\sigma(t) = 1/2(\sigma_{\text{max}} + \sigma_{\text{min}}) + 1/2(\sigma_{\text{max}} - \sigma_{\text{min}})\sin(2\pi t), \quad (\text{A7})$$

where σ_{min} is the minimum and σ_{max} is the maximum cyclic stress. In this study, the minimum stress was set at 10 MPa, and the maximum stress varied between 30 and 80 MPa. Dividing through Eq. (7) by E^* gives

$$\begin{aligned} \sigma/E^* &= 1/2(\varepsilon_{\text{init}} + \sigma_{\text{min}}/E^*) \\ &+ 1/2(\varepsilon_{\text{init}} - \sigma_{\text{min}}/E^*)\sin(2\pi t). \end{aligned} \quad (\text{A8})$$

The time-dependent failure lifetime can be determined by substituting Eq. (8) into Eq. (6) with $A = 5.29 \times 10^8$ s and $B = 158$ as determined from the regression in Fig. 4(b). The resulting equation was solved using the trapezoidal numerical integration function “trapz” in MATLAB (Mathworks, Natick, MA) to predict the time-dependent failure lifetime t_c for each specimen [Fig. 5(b)]. This lifetime is compared with results from the cyclic tests to determine the relative contributions of time-dependent and cycle-dependent damage [Fig. 5(b)].

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