MECHANICAL CHARACTERIZATION OF SUBRUPTURE FATIGUE DAMAGE IN TENDONS

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INTRODUCTION
Despite the high prevalence of tendon injuries, the etiology of tendon rupture is poorly understood. Degeneration due to cumulative microtrauma resulting from overuse has been widely discussed as a major contributor to tendon failure. However, existing biomechanical models of fatigue have largely focused on the relationship between the applied stress and the number of cycles to failure (Wang et al, 1995), identifying stiffness loss as an index of damage. The purpose of the present study was to assess alternate parameters as indices of mechanistic, sub-rupture changes that may underlie the tendon fatigue process.

METHODS
Flexor digitorum longus (N=18) tendons from adult female Sprague-Dawley rats were dissected immediately following euthanasia and underwent fatigue loading in a PBS bath maintained at 39°C. The ends of the tendons were clamped using sandpaper-covered plates. The upper and lower plates were secured to custom grips that were aligned with the test actuator (Instron 8872). Tensile force was measured using a 50-lb load cell attached between the grips and the actuator.

Each sample was allowed to equilibrate to the PBS bath, preloaded to establish its initial length, and loaded cyclically according to a novel damage accumulation protocol (Lee et al, 2006) which consisted of, sequentially, a preconditioning period, Diagnostic test I (D1), Damage Induction phase, Diagnostic test II (D2), Recovery phase, and Diagnostic test III (D3) (Fig. 1). The tendon was cyclically loaded at 20% maximum load for 300 cycles in the preconditioning period and for 120 cycles each in D1, D2 and D3. In the Damage Induction phase, six tendons were loaded at ~50% maximum load to a mechanical endpoint of 20% decline in secant stiffness, while the remaining twelve tendons were loaded to endpoint grip-to-grip strain of 15% or less. The tendon was unloaded for 4500 sec in the Recovery phase to allow transient effects of the cyclic loading to dissipate (Jepsen and Davy, 1997). Secant stiffness (S1, S2, S3), elongation at peak cyclic load (ε1, ε2, ε3) relative to the initial length, and hysteresis (E1, E2, E3) were determined as the mean of the last five cycles of D1 to characterize the tendon in the pre-fatigue state, D2 in the post-fatigue state, and D3 in the post-recovery state, respectively.

RESULTS AND DISCUSSION
Tendons were grouped based on their endpoint strain observed during Damage Induction. In tendons that were loaded to ‘high’ (>11% strain, N=6) and ‘mid’ (9%-11% strain, N=5) fatigue levels, hysteresis (E1→E2) (p≤0.07) increased and stiffness
(S₁→S₂) (p≤0.07) decreased following Damage Induction (Table 1), and did not change significantly following Recovery (E₂→E₃, S₂→S₃). In tendons that were loaded to a ‘low’ level of fatigue (<9% strain), both hysteresis and stiffness did not change significantly from one diagnostic test to the next. Increases in tendon elongation (ε₁→ε₂) were consistent with their respective fatigue levels (from low to high), but did not show a significant amount of recovery (ε₂→ε₃) at any fatigue level.

In the tendons that were loaded to an endpoint of 20% secant stiffness loss, post-fatigue stiffness declined by 21.2±4.0% (S₁→S₂) (p<0.001), indicating that the dynamic assessment of stiffness during Damage Induction in the loading protocol accurately characterized the state of tissue fatigue of the tendon. Changes in strain and stiffness among these tendons during the Damage Induction phase exhibited consistent patterns (Fig. 2). Stiffness remained constant before it declined rapidly, while strain concurrently showed a gradual followed by more rapid rate of increase.

Mechanical characterization of the tendons during Damage Induction and all diagnostic tests is consistent with a fatigue mechanism by which loading is supported by shorter fibers initially and redistributes to the lesser-loaded longer fibers as the loading exceeds the shorter fibers’ capacity. These events, which do not appear to cause stiffness loss, are characterized by increases in elongation. Continued loading leads to the initiation and accumulation of microtears, resulting in stiffness loss, which is also seen in bone and other composite material resulting from the formation of small voids during fatigue. Such damage in tendons altered the viscous response, as reflected by the coupling of changes between stiffness and hysteresis at increasing levels of fatigue. Histological evaluation on these tendons is underway to examine the morphological manifestations of mechanical fatigue and damage.

**Table 1**: Changes in stiffness (S), elongation (ε) and hysteresis (E) from D₁→D₂ (mean±SD). * p≤0.05; # p=0.07.

<table>
<thead>
<tr>
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<th>‘low’ fatigue</th>
<th>‘mid’ fatigue</th>
<th>‘high’ fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td>S₁→S₂ (%)</td>
<td>-6.6±6.6</td>
<td>-13.8±14.5#</td>
<td>-11.1±8.4*</td>
</tr>
<tr>
<td>ε₁→ε₂ (×10⁻³)</td>
<td>+12.3±8.1*</td>
<td>+34.0±18.4*</td>
<td>+49.3±30.7*</td>
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<td>E₁→E₂ (%)</td>
<td>+2.2±5.7</td>
<td>+26.0±29.8#</td>
<td>+30.0±27.1*</td>
</tr>
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</table>

**Figure 2**: Typical stiffness and strain behavior seen in Damage Induction.

**SUMMARY/CONCLUSIONS**

The evaluation of mechanical parameters at various stages of tendon fatigue showed that a low level of fatigue is characterized by changes in the tendon’s elongation without compromise in the stiffness. The varying responses of elongation, stiffness and hysteresis at increasing levels of fatigue reflect the mechanistic changes that may underlie the process by which the tendon degenerates from subrupture loads.

**REFERENCES**


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