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Incorporating Plasticity of the Interfibrillar Matrix in Shear Lag Models is Necessary to Replicate the Multiscale Mechanics of Tendon Fascicles

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Abstract

Despite current knowledge of tendon structure, the fundamental deformation mechanisms underlying tendon mechanics and failure are unknown. We recently showed that a shear lag model, which explicitly assumed plastic interfibrillar load transfer between discontinuous fibrils, could explain the multiscale fascicle mechanics, suggesting that fascicle yielding is due to plastic deformation of the interfibrillar matrix. However, it is unclear whether alternative physical mechanisms, such as elastic interfibrillar deformation or fibril yielding, also contribute to fascicle mechanical behavior. The objective of the current work was to determine if plasticity of the interfibrillar matrix is uniquely capable of explaining the multiscale mechanics of tendon fascicles including the tissue post-yield behavior. This was examined by comparing the predictions of a continuous fibril model and three separate shear lag models incorporating an elastic, plastic, or elastoplastic interfibrillar matrix with multiscale experimental data. The predicted effects of fibril yielding on each of these models were also considered. The results demonstrated that neither the continuous fibril model nor the elastic shear lag model can successfully predict the experimental data, even if fibril yielding is included. Only the plastic or elastoplastic shear lag models were capable of reproducing the multiscale tendon fascicle mechanics. Differences between these two models were small, although the elastoplastic model did improve the fit of the experimental data at low applied tissue strains. These findings suggest that while interfibrillar elasticity contributes to the initial stress response, plastic deformation of the interfibrillar matrix is responsible for tendon fascicle post-yield behavior. This information sheds light on the physical processes underlying tendon failure, which is essential to improve our understanding of tissue pathology and guide the development of successful repair.

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1. Introduction

From an engineering perspective, tendons are hydrated composite biomaterials with a complicated hierarchical structure spanning multiple length scales (Fig. 1) (Kastelic et al., 1978). At the macroscopic level, tendons are separated into subunits called fascicles, which are dense collections of highly aligned collagen fibrils interspersed with cells (Rowe, 1985). The fibrils are separated by an interfibrillar matrix composed of proteoglycans, elastin, collagen VI, and other macromolecules (Thorpe et al., 2013a). However, the primary components of tendon are collagen I molecules, which are contained within the fibrils in a semi-crystalline organization and connected through naturally occurring inter-molecular crosslinks (Bailey et al., 1998; Hulmes et al., 1995; Orgel et al., 2006). Despite the detailed understanding of tendon hierarchical structure, it is unknown how each of the tissue components across the various length scales contributes to the macroscale tissue response. Identifying these structure-function relationships is crucial for understanding tendon pathology leading to tissue failure and for developing repair strategies using either cell-mediated regeneration or tissue engineered replacements.

A central question is whether collagen fibrils are continuous structures that extend across the entire tendon length. Despite difficulties finding fibril ends in tendon and the observation of fibril branching (Kadler et al., 2000; Provenzano and Vanderby, 2006), multiscale experimental testing of tendon fascicles suggests that fibrils are discontinuous and transmit load through shearing of the interfibrillar matrix (Bruehlmann et al., 2004; Gupta et al., 2010; Puxkandl et al., 2002; Rigozzi et al., 2011; Szczesny and Elliott, 2014). We recently showed that a shear lag model, which explicitly assumes interfibrillar load transfer between discontinuous fibrils, can explain the multiscale fascicle mechanics, strongly suggesting that fibrils are indeed discontinuous (Szczesny and Elliott, 2014). Furthermore, that study proposed that the fascicle post-yield behavior is produced by plastic deformation of the interfibrillar matrix. However, it is unclear whether alternative physical mechanisms, including elastic interfibrillar deformation and fibril yielding, also contribute to fascicle mechanical behavior.

Shear lag models have been widely used to represent several biological tissues composed of discontinuous reinforcing elements embedded in a softer matrix (Ahmadzadeh et al., 2014; Buehler, 2008, 2006; Gao et al., 2003; Jäger and Fratzl, 2000; Zhang et al., 2010). The fundamental assumption of all shear lag models is that the externally applied load is transferred to the reinforcing elements through shear stresses acting at the interface with the surrounding matrix. The classic shear lag model first proposed by Cox (1952) has been applied to tendon (Torp et al., 1975). However, this formulation assumed that the matrix is isotropic linear elastic and that the shear forces generated in the matrix are not directly coupled to the relative sliding between fibrils (Hull and Clyne, 1996). More recent models have relaxed these restrictions and coupled interfibrillar shear stresses to specific tissue components, but they still assume elasticity of the interfibrillar matrix (Ahmadzadeh et al., 2013; Bar-On and Wagner, 2013; Fessel and Snedeker, 2011). Alternative formulations have been developed incorporating a plastic, elastoplastic, and viscous interfibrillar matrix (Ahmadzadeh et al., 2014; Ciarletta and Ben Amar, 2009; Gao et al., 2008; Goh et al., 2007; Mijailovich et al., 1993; Szczesny and Elliott, 2014). However, there has not been an

evaluation of how well the various forms of shear lag models predict the multiscale mechanical behavior of tendon fascicles.

The objective of the current work was to determine if plasticity of the interfibrillar matrix is uniquely capable of explaining the multiscale mechanics of tendon fascicles including the tissue post-yield behavior. This was examined by comparing the predictions of a continuous fibril model and three separate shear lag models incorporating an elastic, plastic, or elastoplastic interfibrillar matrix with published multiscale experimental data (Szczeny et al., 2014). The potential effects of fibril yielding on each of these models were also considered. The results demonstrated that only the plastic or elastoplastic shear lag models were capable of reproducing the experimental data and the post-yield behavior in particular, which suggests that plastic deformation of the interfibrillar matrix is responsible for yielding of tendon fascicles. This information sheds light on the physical processes underlying tendon failure, which is essential to improve our understanding of tissue pathology and guide the development of successful repair.

The general principles of the shear lag models as well as the specific formulations for the elastic and plastic models are presented in Section 2. In order to compare the shear lag models to experimental data, the derived equations were modified to account for fibril crimping (Section 2.4), which is responsible for the initial “toe-region” in the stress-strain behavior of tendon fascicles (Diamant et al., 1972). In Section 3, the multiscale behavior of both the elastic and plastic models are compared along with their predictions of the experimental data and the potential effects of fibril yielding. Finally, Section 4 presents the derivation and performance of the elastoplastic shear lag model.

2. Shear Lag Model Formulations

2.1. General Features

For the current study of tendon fascicles, the fascicle structure is simplified as a staggered array of discontinuous fibrils embedded in an extrafibrillar matrix (Szczeny and Elliott, 2014; Ahmadzadeh et al., 2013; Goh et al., 2007), which is represented by a periodic unit cell that is half the fibril length (L) (Fig. 2). At any point x where there is relative sliding between the fibrils, an interfibrillar shear stress ($\tau(x)$) acts equally over the entire fibril circumference, leading to the following equilibrium equation:

$$\frac{d\sigma_1(x)}{dx} = \frac{2\tau(x)}{r} = -\frac{d\sigma_2(x)}{dx} \quad (1)$$

where $\sigma_i(x)$ is the stress in the i -th fibril and r is the fibril radius. Note that the ends of the fibrils are unloaded, P is the stress at fibril midpoint, and, based on symmetry, $\sigma_2(x) = \sigma_1(L/2 - x)$. It is assumed that the fibrils are linear elastic with a modulus E_f and that the axial displacements of the fibrils are uniform throughout their cross-section (i.e., no intra-fibrillar shear). The only difference between the shear lag models presented in this paper is the form taken by the interfibrillar shear stress ($\tau(x)$). Once this is specified, expressions for the fibril stress and displacements ($u_f(x)$) are determined using the boundary condition $u_1(L/2) = -u_2(0) = U/2$ with the strain applied to the unit cell (ϵ_{uc}) equal to $2U/L$.

2.2. Elastic Interfibrillar Matrix

The classic shear lag model first proposed by Cox (1952) assumes that the interfibrillar matrix is isotropic linear elastic and that the matrix strain far from the fibrils is equal to the macroscale strain applied to the tissue composite (Hull and Clyne, 1996). This second assumption may not be valid given the high density and close packing of fibrils in tendon (Parry and Craig, 1984). Therefore, recent applications of shear lag models to tendon have described the interfibrillar shear stress as a function of the relative sliding between adjacent fibrils (Ahmadzadeh et al., 2013; Mijailovich et al., 1993), which can be expressed as

$$\tau(x) = G \cdot \frac{u_1(x) - u_2(x)}{h} \quad (2)$$

where G is the shear modulus of the interfibrillar matrix and h is the distance between the fibril surfaces. Although more complicated formulations have been developed based on assumptions for the organization and behavior of specific tissue components (Ahmadzadeh et al., 2013; Ciarletta and Ben Amar, 2009; Fessel and Snedeker, 2011; Goh et al., 2007; Zhang et al., 2010), Eq. (2) captures the fundamental mechanisms underlying elastic shear lag models. Assuming hexagonal lateral fibril packing, the interfibrillar distance can be related to the fibril volume fraction (ϕ) and radius (Hull and Clyne, 1996):

$$h = 2r \left(\frac{\sqrt{\pi}}{\sqrt{2\phi} \sqrt[4]{3}} - 1 \right) \quad (3)$$

Solving the governing equilibrium equation (Eq. (1)) using the boundary conditions described in Section 2.1 produces the following expressions for the fibril stress, interfibrillar shear stress, and fibril displacements:

$$\sigma_1(x) = C \varepsilon_{uc} \left(e^{x/L_c} - e^{(L/2-x)/L_c} + e^{L/2L_c} - 1 \right) \quad (4)$$

$$\tau(x) = C \varepsilon_{uc} \cdot \frac{r}{2L_c} \left(e^{x/L_c} + e^{(L/2-x)/L_c} \right) \quad (5)$$

$$u_1(x) = \frac{C \varepsilon_{uc}}{E_f} \left[L_c e^{x/L_c} + L_c e^{(L/2-x)/L_c} + \left(1 - e^{L/2L_c} \right) \left(\frac{L}{4} - x \right) \right] \quad (6)$$

$$u_2(x) = -\frac{C \varepsilon_{uc}}{E_f} \left[L_c e^{x/L_c} + L_c e^{(L/2-x)/L_c} - \left(1 - e^{L/2L_c} \right) \left(\frac{L}{4} - x \right) \right] \quad (7)$$

$$C = \frac{E_f L}{4} \left[\left(L_c + \frac{L}{4} \right) e^{L/2L_c} + \left(L_c - \frac{L}{4} \right) \right]^{-1} \quad (8)$$

$$L_c = \sqrt{\frac{E_f r h}{4G}} \quad (9)$$

where L_c is the characteristic length over which load is transferred between fibrils. The average stress in the unit cell (σ_{uc}) is given by the stress averaged over the two fibrils, which, due to symmetry, reduces to

$$\sigma_{uc} = \frac{\sigma_1(x) + \sigma_2(x)}{2} = C \varepsilon_{uc} \left(e^{L/2L_c} - 1 \right) = P/2. \quad (10)$$

Note that the expression for σ_{uc} is equal to the fibril stress at the midpoint of the unit cell ($\sigma_1(L/4) = \sigma_2(L/4)$).

2.3. Plastic Interfibrillar Matrix

Various shear lag models have incorporated plasticity of the interfibrillar matrix (Ciarletta and Ben Amar, 2009; Gao et al., 2008; Goh et al., 2007; Mijailovich et al., 1993). Here we used a simple perfectly plastic constitutive behavior to represent the fundamental features of plasticity of the interfibrillar matrix (Szczeny and Elliott, 2014). Specifically, relative sliding between fibrils was assumed to produce a constant interfibrillar shear stress:

$$\tau(x) = \begin{cases} \tau_p & u_1(x) \neq u_2(x) \\ 0 & u_1(x) = u_2(x) \end{cases} \quad (11)$$

By enforcing compatibility along the fibril length, direct integration leads to the following piecewise solutions for the fibril stress and displacements:

$$\sigma_1(x) = \begin{cases} 2\tau_p x / r & 0 \leq x \leq L_s \\ 2\tau_p L_s / r = P/2 & L_s \leq x \leq L/2 - L_s \\ 2\tau_p (x - L/2 + 2L_s) / r & L/2 - L_s \leq x \leq L/2 \end{cases} \quad (12)$$

$$u_1(x) = \begin{cases} \tau_p (x^2 + L_s^2 - L_s L/2) / r E_f & 0 \leq x \leq L_s \\ \tau_p L_s (2x - L/2) / r E_f & L_s \leq x \leq L/2 - L_s \\ \tau_p [x^2 + L_s^2 + (L/2)^2 + (4L_s - L)x - 3L_s L/2] / r E_f & L/2 - L_s \leq x \leq L/2 \end{cases} \quad (13)$$

$$u_2(x) = \begin{cases} -\tau_p (x^2 + L_s^2 - 4L_s x + L_s L/2) / r E_f & 0 \leq x \leq L_s \\ \tau_p L_s (2x - L/2) / r E_f & L_s \leq x \leq L/2 - L_s \\ -\tau_p [x^2 + L_s^2 + (L/2)^2 - Lx - L_s L/2] / r E_f & L/2 - L_s \leq x \leq L/2 \end{cases} \quad (14)$$

$$L_s = -L/4 + \sqrt{(L/4)^2 + (r E_f L / 4 \tau_p) \varepsilon_{uc}} \quad (15)$$

where L_s is the length from the fibril ends over which there is relative sliding between fibrils. As in Eq. (10), due to symmetry, the stress averaged over the two fibrils is equal to

the fibril stress at the midpoint of the unit cell ($\sigma_{uc} = \sigma_l(L/4) = P/2$). Therefore, the average stress in the unit cell is

$$\sigma_{uc} = \frac{\tau_p}{r} \left[\sqrt{L^2/4 + (rE_f L/\tau_p) \varepsilon_{uc}} - L/2 \right]. \quad (16)$$

2.4. Fibril Uncrimping

In order to compare the shear lag models to experimental data, the equations described above were modified to account for fibril crimping, which is responsible for the initial non-linearity (i.e., toe-region) in the macroscale tissue behavior (Diamant et al., 1972). The fibrils, and hence each unit cell, were assumed to be initially crimped and bear load only after the applied tissue stretch (λ) reaches a specific uncrimping stretch (λ_c). Since fibrils are not uniformly crimped within a fascicle (Kastelic et al., 1978; Rowe, 1985), a gamma probability distribution was used to represent the spread of tissue stretches required to uncrimp the unit cells ($\gamma(\lambda_c - 1)$) (Sacks, 2003; Szczesny et al., 2012), which was parameterized by the mean uncrimping stretch ($\bar{\lambda}_c$) and standard deviation of uncrimping (λ_c^{SD}). The macroscale tissue stress (σ_T) was then calculated through the weighted integral of the expressions for σ_{uc} (Eq. (10,16)) multiplied by the fibril volume fraction

$$\sigma_T(\lambda) = \phi \int_1^\lambda \sigma_{uc} \gamma(\lambda_c - 1) d\lambda_c \quad (17)$$

where ε_{uc} in Eq. (10,16) is substituted with $\lambda/\lambda_c - 1$, which is the expression for the strain of the unit cell after uncrimping normalized by its uncrimping stretch.

A similar correction for uncrimping must be made in order to compare the fibril strains predicted by the model to the strains measured experimentally. Since the fibril strains were measured using optical microscopy (Szczesny et al., 2014), the apparent (experimentally observed) fibril strains are not simply the strains for which the fibrils bear load, but are the cumulative elongation of the fibrils normalized by their original crimped length. Therefore, the equivalent apparent strain (ε_{app}) of the uncrimped fibrils predicted by the shear lag models is

$$\varepsilon_{app} = (1 + \sigma_{uc}/E_f) \lambda_c - 1 \quad \text{where} \quad \lambda \geq \lambda_c. \quad (18)$$

Note that the term σ_{uc}/E_f represents the true fibril strain (averaged over the fibril length) due to the application of load following uncrimping. Prior to uncrimping (i.e., $\lambda < \lambda_c$), the apparent fibril strain is equal to the tissue strain ($\lambda - 1$). Again using a gamma distribution for uncrimping stretches, the apparent fibril:tissue strain ratio (η_{app}) predicted by the models is

$$\eta_{app}(\lambda) = [1 - \Gamma(\lambda - 1)] + (\lambda - 1)^{-1} \int_1^{\lambda} \varepsilon_{app} \gamma(\lambda_c - 1) d\lambda_c \quad (19)$$

where $\Gamma(\lambda - 1)$ is the gamma cumulative distribution function. Note that the first term in brackets represents the fibrils that are still crimped and, therefore, have the same apparent strain as the tissue. These techniques have previously been validated to accurately represent fibril uncrimping in tendon fascicles (Szczesny and Elliott, 2014). For the comparison of the model predictions with experimental data, the fibril radius and volume fraction were assumed to be 85 nm and 0.7, respectively (Parry and Craig, 1984; Screen et al., 2005), and the remaining model parameters (E_f , τ_p , L , $\bar{\lambda}_c$, λ_c^{SD}) were based on a previous fit of the plastic model using Eq. (17) to average macroscale fascicle mechanics (Szczesny et al., 2014) (Table 2). The same parameter values were used for the elastic model (Table 2), with the value of G (~1 Pa) chosen to give a linear-region tissue response that was similar to the prediction of the plastic model.

3. Comparison of Elastic and Plastic Models

3.1. General Behavior

Before investigating how well the elastic and plastic models replicate fascicle multiscale mechanics, it is instructive to compare their general behavior. In particular, consider the length over which load is transferred between fibrils through interfibrillar shear. For the elastic model, this is described by L_c , which, according to Eq. (9), is a fixed value determined by material and geometric constants. This means that the amount of interfibrillar sliding in the elastic model is the same regardless of the strain applied to the tissue. Furthermore, while the magnitudes of the fibril stress, interfibrillar shear stress, and fibril displacements scale with ε_{uc} (Eq. 4–7), their distributions are independent of the applied tissue strain since L_c remains constant (Fig. 3). In contrast, the expression for the load transfer length in the plastic model (L_s) is dependent on the unit cell strain and, therefore, the applied tissue strain (Eq. (15)). This indicates that in the plastic model the amount of interfibrillar sliding, and hence the behavior of the model, changes with the applied tissue strain. At low strains, L_s is less than L_c , which means that there is less interfibrillar sliding (i.e., smaller region where $u_1 - u_2$) in the plastic model than in the elastic model (Fig. 3C–D). This is because the interfibrillar shear stress in the plastic model is greater than the maximum interfibrillar shear stress in the elastic model (Fig. 3B). Furthermore, less interfibrillar sliding means that more strain is transmitted to the fibrils, resulting in a higher average fibril stress in the plastic model compared to the elastic model (Fig. 3A).

As more strain is applied to the tissue, the interfibrillar sliding in the plastic model increases but remains constant in the elastic model. When L_s and L_c are comparable in magnitude, the amount of interfibrillar sliding and point at which the interfibrillar shear approaches zero is approximately the same in both models (Fig. 3F–H). At this point, the fibril stress distribution over the unit cell is also very similar between the elastic and plastic models (Fig. 3E). However, if the applied strain continues to increase, then ultimately the interfibrillar shear stress will act over the entire fibril length in the plastic model ($L_s = L/4$). At this point ($\varepsilon_{uc} = 3\tau_p L/4rE_f = \varepsilon_p$) the fibrils are fully loaded with interfibrillar shear and sliding

occurring at all points along the fibril length (Fig. 3I–L). Any tissue strain beyond this point will simply cause the fibrils to slide without further elongation. In contrast, as expected from Eq. (4–7) for the elastic model, the distributions of the fibril stress, interfibrillar shear stress, and fibril displacements are simply scaled by the applied strain, with no change in the distribution shape.

3.2. Consequences on Multiscale Behavior

The difference in load transfer lengths between the plastic and elastic shear lag models has important consequences on the resulting macroscale tissue behavior. Ignoring any uncrimping nonlinearities in the tissue stress response (these are included in Section 3.3), the macroscale tissue modulus predicted by each model can be determined by differentiating the expressions for σ_{uc} (Eq. (10,16)) with respect to ε_{uc} and multiplying by the fibril volume fraction:

$$E_e = \phi C \left(e^{L/2L_c} - 1 \right) = \frac{\phi E_f L}{4} \frac{\left(e^{L/2L_c} - 1 \right)}{\left(L_c + L/4 \right) e^{L/2L_c} + \left(L_c - L/4 \right)} \quad (20)$$

$$E_p = \phi E_f L \cdot \left[L^2 + (4r E_f L / \tau_p) \varepsilon_{uc} \right]^{-1/2} \quad (21)$$

where E_e and E_p are the tissue moduli for the elastic and plastic models, respectively. Similar to the load transfer lengths, the tissue modulus for the elastic model is constant and independent of the applied tissue strain. In contrast, the tissue modulus for the plastic model is a function of ε_{uc} , which is equal to the applied tissue strain in the case of no fibril crimping. Initially, when ε_{uc} is zero, the modulus is equal to E_f , which is equivalent to the case where the fibrils are continuous and extend across the entire tissue length. As the applied tissue strain increases, the modulus continuously decreases until $\varepsilon_{uc} = \varepsilon_p$. At this point, the tissue stress (i.e., σ_{uc}) ultimately reaches a maximum value of $\tau_p L / 2r$ and, as described earlier, any increase in the tissue strain beyond this point simply causes the fibrils to slide without increasing the fibril stress, which produces a perfectly plastic response at the macroscale tissue level.

Similar differences between the plastic and elastic shear lag models are also observed at the fibrillar length scale. Dividing the expressions for σ_{uc} for each model (Eq. (10,16)) by E_f produces the average fibril strain and further dividing by ε_{uc} results in the fibril:tissue strain ratios:

$$\eta_e = \frac{C}{E_f} \left(e^{L/2L_c} - 1 \right) = \frac{L}{4} \frac{\left(e^{L/2L_c} - 1 \right)}{\left(L_c + L/4 \right) e^{L/2L_c} + \left(L_c - L/4 \right)} \quad (22)$$

$$\eta_p = \frac{\tau_p}{r E_f} \left[\sqrt{\left(\frac{L}{2\varepsilon_{uc}} \right)^2 + \left(\frac{r E_f L}{\tau_p \varepsilon_{uc}} \right)} - \frac{L}{2\varepsilon_{uc}} \right] \quad (23)$$

where η_e and η_p are the fibril:tissue strain ratios for the elastic and plastic shear lag models, respectively. Similar to the macroscale tissue modulus, for the elastic model, the fibril:tissue strain ratio is constant while, for the plastic model, the strain ratio decreases with applied tissue strain. In fact, it is exactly this behavior at the fibrillar level that produces the drop in the tissue modulus with applied strain and ultimately leads to perfectly plastic macroscale behavior. The significance of the decreasing fibril:tissue strain ratio inherent in the plastic shear lag model will be evident when both models are applied to multiscale fascicle experimental data in the next section.

3.3. Prediction of Tendon Fascicle Multiscale Mechanics

In this section the multiscale behavior predicted by the elastic and plastic shear lag models (Eq. (17,19)) are compared to published experimental data of tendon fascicles (Szczeny et al., 2014). A model with continuous fibrils that span the entire fascicle length is used as a reference, where $\sigma_{uc} = E_f \cdot \varepsilon_{uc}$. The plots of the tissue behavior demonstrate that the continuous model cannot reproduce the macroscale stress response (Fig. 4A) or the fibril:tissue strain ratio (Fig. 4B). In particular, since the fibrils in this model are continuous, then, by definition, the fibril:tissue strain ratio is fixed at one, which does not agree with the experimental observations. This suggests that the fibrils are discontinuous and that a shear lag model is more appropriate. However, the elastic shear lag model does not perform substantially better. As expected from Eq. (10) and (20), the elastic model produces a linear macroscale tissue response after initial fibril uncrimping, which cannot replicate the observed strain-softening of the tendon fascicle (Fig. 4A). In addition, after the initial drop due to fibril uncrimping, the elastic model predicts a constant fibril:tissue strain ratio (Eq. (22)), which does not match the experimentally measured decrease in the fibril:tissue strain ratio with increased tissue strain (Fig. 4B). In contrast, the plastic shear lag model can reproduce the macroscale tissue behavior including the post-yield response (Fig. 4A). Furthermore, this model successfully predicts the general relationship between the fibril and tissue strains (Fig. 4B). This suggests not only that the fibrils are discontinuous but that plastic deformation of the interfibrillar matrix is responsible for the fascicle multiscale mechanics.

It is possible that alternative processes other than interfibrillar plasticity (e.g. fibril yielding) could explain the tissue strain-softening behavior and potentially improve the performance of the continuous model or the elastic shear lag model. To investigate this possibility, the fibril modulus was reduced by 30% and the model simulations were repeated. Although this does not strictly represent a progressive drop in fibril modulus, in the absence of clear experimental data for fibril yielding, it does provide an estimate of how each model would be affected by such behavior. As expected, for the continuous model and both shear lag models, the tissue stress is lower with the reduced fibril modulus (Fig. 4C). Therefore, it is conceivable that a progressive drop in the fibril modulus could improve the fit of the tissue post-yield behavior for the continuous and elastic models. However, the reduced fibril modulus increases the fibril:tissue strain ratio for the elastic shear lag model, while the fibril:tissue strain ratio for the continuous model remains at one (Fig. 4D). This demonstrates that fibril yielding does not improve the prediction of the fibril strains for either the continuous model or the elastic shear lag model. For the plastic model, the

increased fibril strains due to fibril yielding possibly improves the prediction of the fibril:tissue strain ratio at higher tissue strains. This suggests that while plasticity of the interfibrillar matrix is necessary to explain the fascicle post-yield behavior, fibril yielding may contribute as well.

4. Combined Elastoplastic Shear Lag Model

4.1. Model Formulation

Although the plastic shear lag model described above can reproduce the general fascicle multiscale behavior (Fig. 4), it is likely that the interfibrillar matrix does have an elastic response at low stress values. Additionally, it is more likely that the interfibrillar shear stress gradually increases with interfibrillar sliding up to a maximum value before becoming plastic instead of the binary response described in Eq. (11). Therefore, in this section, an elastoplastic shear lag model is presented with an interfibrillar matrix that is initially elastic and then behaves perfectly plastically upon reaching a shear stress τ_p .

The constitutive behavior of the elastoplastic interfibrillar matrix can be described by bounding the elastic expression in Eq. (2), such that $|\tau(x)| \leq \tau_p$. Alternatively, if we define L_p as the length from the fibril ends where the interfibrillar shear stress has become perfectly plastic, then we can use the following spatial definition for elastoplastic constitutive behavior:

$$\tau(x) = \begin{cases} \tau_p & 0 \leq x \leq L_p \\ G \cdot \frac{u_1(x) - u_2(x)}{h} & L_p \leq x \leq L/2 - L_p \\ \tau_p & L/2 - L_p \leq x \leq L/2 \end{cases} \quad (24)$$

By enforcing compatibility along the fibril length, applying the boundary conditions described in Section 2.1, and ensuring the interfibrillar shear stress is continuous (i.e., $u_1(L_p) - u_2(L_p) = \tau_p h / G$), direct integration of Eq. (1) leads to the following piecewise solutions for the fibril stress, interfibrillar shear stress, and fibril displacements:

$$\sigma_1(x) = \begin{cases} 2\tau_p x / r & 0 \leq x \leq L_p \\ C_1 \left[e^{x/L_c} - e^{(L/2-x)/L_c} + e^{(L/2-L_p)/L_c} - e^{L_p/L_c} \right] + 2\tau_p L_s / r & L_p \leq x \leq L/2 - L_p \\ 2\tau_p (x - L/2) / r + P & L/2 - L_p \leq x \leq L/2 \end{cases} \quad (25)$$

$$\tau(x) = \begin{cases} \tau_p & 0 \leq x \leq L_p \\ \frac{C_1 r}{2L_c} \left[e^{x/L_c} + e^{(L/2-x)/L_c} \right] & L_p \leq x \leq L/2 - L_p \\ \tau_p & L/2 - L_p \leq x \leq L/2 \end{cases} \quad (26)$$

$$u_1(x) = \begin{cases} \tau_p x^2 / r E_f + C_2 & 0 \leq x \leq L_s \\ \frac{C_1}{E_f} \left[L_c \left(e^{(L/2-x)/L_c} + e^{x/L_c} \right) + (x - L/4) \left(e^{(L/2-L_p)/L_c} - e^{L_p/L_c} \right) \right] + \frac{2\tau_p L_p}{r E_f} (x - L/4) & L_s \leq x \leq L/2 - L_s \\ \tau_p (x^2 - Lx) / r E_f + Px / E_f + C_3 & L/2 - L_s \leq x \leq L/2 \end{cases} \quad (27)$$

$$u_2(x) = \begin{cases} -\tau_p x^2 / r E_f + P x / E_f + C_4 & 0 \leq x \leq L_s \\ -\frac{C_1}{E_f} \left[L_c \left(e^{(L/2-x)/L_c} + e^{x/L_c} \right) - (x-L/4) \left(e^{(L/2-L_p)/L_c} - e^{L_p/L_c} \right) \right] + \frac{2\tau_p L_p}{r E_f} (x-L/4) & L_s \leq x \leq L/2-L_s \\ -\tau_p (x^2 - Lx) / r E_f + C_5 & L/2-L_s \leq x \leq L/2 \end{cases} \quad (28)$$

where L_c is given by Eq. (9) and

$$P = \frac{\tau_p h E_f}{G L_c} \left(\frac{e^{(L/2-L_p)/L_c} - e^{L_p/L_c}}{e^{(L/2-L_p)/L_c} + e^{L_p/L_c}} \right) + \frac{4\tau_p L_p}{r} \quad (29)$$

$$C_1 = \frac{\tau_p h E_f}{2G L_c (e^{(L/2-L_p)/L_c} + e^{L_p/L_c})} \quad (30)$$

$$C_2 = \frac{\tau_p h}{2G} \left[1 + \frac{1}{L_c} (L_p - L/4) \left(\frac{e^{(L/2-L_p)/L_c} - e^{L_p/L_c}}{e^{(L/2-L_p)/L_c} + e^{L_p/L_c}} \right) \right] + \frac{\tau_p}{r E_f} (L_p^2 - L L_p / 2) \quad (31)$$

$$C_3 = \frac{\tau_p h}{2G} \left[1 - \frac{1}{L_c} (3L/4 - L_p) \left(\frac{e^{(L/2-L_p)/L_c} - e^{L_p/L_c}}{e^{(L/2-L_p)/L_c} + e^{L_p/L_c}} \right) \right] + \frac{\tau_p}{r E_f} (L^2/4 - 3L L_p / 2 + L_p^2) \quad (32)$$

$$C_4 = -\frac{\tau_p h}{2G} \left[1 + \frac{1}{L_c} (L_p + L/4) \left(\frac{e^{(L/2-L_p)/L_c} - e^{L_p/L_c}}{e^{(L/2-L_p)/L_c} + e^{L_p/L_c}} \right) \right] - \frac{\tau_p}{r E_f} (L_p^2 + L L_p / 2) \quad (33)$$

$$C_5 = -\frac{\tau_p h}{2G} \left[1 - \frac{1}{L_c} (L/4 - L_p) \left(\frac{e^{(L/2-L_p)/L_c} - e^{L_p/L_c}}{e^{(L/2-L_p)/L_c} + e^{L_p/L_c}} \right) \right] - \frac{\tau_p}{r E_f} (L^2/4 - L L_p / 2 + L_p^2) \quad (34)$$

For an applied unit cell strain of ε_{uc} , L_p is determined by solving the equation

$$\varepsilon_{uc} L / 4 + C_4 = 0. \quad (35)$$

Again, based on symmetry, the stress applied to the unit cell averaged over the two fibrils is $\sigma_{uc} = P/2$, which can be determined from Eq. (29).

4.2. General Behavior

Initially, the elastoplastic model behavior matches the elastic shear lag model since the interfibrillar matrix has an elastic response while $\tau(x) < \tau_p$ (Fig. 5A–C). The unit cell strain at which the interfibrillar shear stress initially becomes plastic at the fibril ends (i.e., $\tau(0) = \tau(L/2) = \tau_p$) is determined from Eq. (35) with $L_p = 0$ and is given by

$$\varepsilon_{p,i} = \frac{2\tau_p h}{GL} \left[1 + \frac{L}{4L_c} \left(\frac{e^{L/2L_c} - 1}{e^{L/2L_c} + 1} \right) \right]. \quad (36)$$

As the applied strain increases, the region of plastic interfibrillar shear stress and interfibrillar sliding enlarges towards the center of the unit cell (Fig. 5D–F). Ultimately, at a unit cell strain of $\varepsilon_{p,f}$ the interfibrillar shear stress has become plastic along the full fibril length (i.e., $L_p = L/4$; Fig. 5G–I):

$$\varepsilon_{p,f} = \frac{2\tau_p h}{GL} + \frac{3\tau_p L}{4rE_f}. \quad (37)$$

Note that the second term is the expression for ε_p in the plastic shear lag model (Section 3.1). Since all the terms in Eq. (37) are positive values, this means that the initial elasticity of the interfibrillar matrix increases the tissue strain at which the fibrils are maximally loaded and the macroscale tissue response becomes perfectly plastic.

4.3. Prediction of Tendon Fascicle Multiscale Mechanics

The combined elastoplastic shear lag model was compared to the plastic model described above (using the same model parameters as in Table 2 and Section 3.3) in order to evaluate the importance of adding elasticity within the interfibrillar matrix to the plastic shear lag model. At the macroscopic level, the elastoplastic model produced a tissue response that was very similar to the plastic model (Fig. 6A). However, the effect of the interfibrillar matrix elasticity was more pronounced on the fibril:tissue strain ratio, producing a small improvement in the model predictions at low tissue strains (Fig. 6B). At strains above $\varepsilon_{p,f}$, the two models converged since the response of the interfibrillar matrix in the elastoplastic model has become fully plastic at this point. These simulations demonstrate that, while the initial elasticity of the interfibrillar matrix may contribute to the tissue response at low strains, matrix plasticity is responsible for the post-yield behavior.

5. Discussion

Recently we proposed that the multiscale mechanics of tendon fascicles can be explained by a shear lag model incorporating a perfectly plastic interfibrillar matrix that transmits load between discontinuous fibrils (Szczeny and Elliott, 2014). The objective of this study was to evaluate whether alternative models based on an elastic interfibrillar matrix could provide equally successful explanations of the fascicle mechanics. Comparing the predictions of these models with the plastic shear lag model demonstrated that the plastic model has a unique ability to reproduce the tissue behavior at multiple length scales. A key feature of the plastic shear lag model is that interfibrillar sliding increases with greater applied tissue strain (Eq. 15), which is consistent with previous microscale measurements (Screen et al., 2004; Szczeny and Elliott, 2014; Szczeny et al., 2014; Thorpe et al., 2013b). This increased interfibrillar sliding reduces the amount of strain that is transmitted to the fibrils during testing and results in a decrease in the tissue modulus, which is consistent with the observed multiscale experimental data of tendon fascicles (Fig. 4A–B). In contrast, none of these

behaviors are replicated by the elastic shear lag model. An elastoplastic shear lag model, in which the interfibrillar matrix is initially elastic before becoming plastic, does improve the prediction of the fibril:tissue strain ratio at low applied tissue strains (Fig. 6B); however, the improvement is small despite the addition of an extra model parameter. Therefore, these data suggest that, while elasticity of the interfibrillar matrix contributes to the initial tissue response to loading, interfibrillar plasticity is indeed the dominant deformation mechanism underlying fascicle post-yield behavior.

The unique ability of the plastic shear lag model to replicate tendon fascicle mechanics is consistent with previous suggestions that fascicle failure is due to plasticity within the interfibrillar matrix rather than fibril rupture. Tensile testing of individual fibrils produces failure strains and stresses that are substantially higher than the values obtained from tendon fascicles, even after accounting for the fibril volume fraction (Hansen et al., 2009; Shen et al., 2010; Svensson et al., 2013), which suggests that fascicle failure is not due to fibril rupture. In addition, tendon fascicles stretched beyond their yield point contain fibrils with localized plastic deformation that do not show signs of rupture until after a substantial amount of fatigue loading (>800 cycles) (Fung et al., 2009; Veres et al., 2013). Fascicle behavior during long-term creep and notch tension testing suggests that damage processes are localized to the interfibrillar matrix (Ker, 2007; Wang and Ker, 1995). This agrees with findings suggesting dissipative deformation mechanisms within the interfibrillar matrix (Gupta et al., 2007, 2005; Gutsman et al., 2005; Svensson et al., 2010), possibly due to the breakage and reformation of weak non-covalent bonds (Liu et al., 2005). Such energy dissipation through plastic deformation of the interfibrillar matrix would serve as a toughening mechanism for tendon and help prevent complete rupture due to localized tissue defects.

While fibrils may not rupture, it is possible that fibril yielding also contributes to the strain-softening behavior observed in the post-yield behavior of tendon fascicles. Fascicles subjected to sub-rupture tensile testing contain kinked fibrils, which may indicate some form of fibril yielding (Fung et al., 2009; Thorpe et al., 2014; Veres et al., 2013). However, despite numerous experimental studies of the tensile properties of individual fibrils, little data exists concerning fibril yielding and post-yield behavior (Hansen et al., 2009; Shen et al., 2010; Svensson et al., 2013; van der Rijt et al., 2006; Yang et al., 2012). Furthermore, molecular dynamics simulations predict fibril yielding due to molecular sliding within fibrils but only at large fibril strains (>25%) (Buehler, 2008, 2006). Therefore, we simulated the effects of fibril yielding by evaluating the model performance with a reduced (but still constant) fibril modulus. Although this does not simulate true fibril yielding, it does provide an upper bound estimate for the effects since the fibril modulus is reduced throughout testing rather than after reaching some particular yield strain.

We found that fibril yielding, while potentially explaining the macroscale strain-softening, does not improve the prediction of the fibril:tissue strain ratio for the continuous fibril model or the elastic shear lag model (Fig. 4C–D). It is possible that fibril yielding could bring the fibril:tissue strain ratio predicted by the plastic model closer to the experimental values at high tissue strains, but it is unclear if the fibrils would reach their yield point in this model. At a tissue strain of 8%, the maximum fibril strain averaged over the fibril length is only

2.31% in the plastic model, which demonstrates how interfibrillar sliding can protect fibrils from yielding and complete rupture. This agrees with X-ray diffraction studies of fascicles from 6–12 month-old rats (which were also used for the experimental data in Fig. 4 and 6 (Szczesny et al., 2014)) that found that sliding between fibrils preceded molecular slippage within fibrils, and likely, fibril yielding (Folkhard et al., 1987). Hence, plastic interfibrillar sliding is a more likely explanation of the multiscale data than fibril yielding.

The use of relatively simple shear lag models in this study provided a clear comparison of how the capabilities and limitations of each model were dictated by the choice of the constitutive behavior for the interfibrillar matrix. Other shear lag models have been developed that assume more complex properties or organization of specific tissue components (e.g. proteoglycans) to generate elastic, plastic, and viscous behaviors of the interfibrillar matrix (Ahmadzadeh et al., 2013; Ciarletta and Ben Amar, 2009; Fessel and Snedeker, 2011; Gao et al., 2008; Goh et al., 2007; Mijailovich et al., 1993; Zhang et al., 2010). These models offer interesting explanations for tissue behavior, although the experimental data necessary to qualify their underlying assumptions are limited (Fessel and Snedeker, 2011; Lujan et al., 2007; Rigozzi et al., 2013; Svensson et al., 2011). We attempted to avoid this issue and clarify interpretations of the models' capabilities by using formulations based on fundamental principles common to nearly all shear lag models. For example, despite its simplicity, the elastic shear lag model presented in this study captures the basic features of other elastic models, including the constant values for L_c , the tissue modulus, and the fibril:tissue strain ratio (Ahmadzadeh et al., 2013; Bar-On and Wagner, 2013; Gao et al., 2003; Hull and Clyne, 1996; Jäger and Fratzl, 2000). The conclusions of this study can guide the future development and application of multiscale models that encapsulate multiple phenomena across the full tendon hierarchical structure, including nonlinear strain-stiffening fibril behavior as well as viscous and poroelastic effects (Gautieri et al., 2011; Reese, 2013; Shen et al., 2011; Yang et al., 2012; Yin and Elliott, 2004).

Since the main purpose of this study was to compare the general ability of various shear lag models to reproduce the multiscale mechanics of tendon fascicles, an optimization and sensitivity analysis of the model parameters were not performed. Still, it is worth considering the effects and physiological significance of the chosen values for the model parameters, especially the fibril length. A length of 10 mm was chosen in this study since it is the approximate midpoint between the lower bound of 1 mm, suggested by previous measurements of fibril lengths in fetal tendon (Birk et al., 1995), and the upper bound of 25 mm, based on the initial grip-to-grip distance used in the experimental testing (Szczesny et al., 2014). While fibril lengths may not be the same in different tendons or different animals, 10 mm is not an unreasonable assumption for the fibril length in rat tail tendon fascicles, which are over 100 mm long (Legerlotz et al., 2010). Furthermore, while different fibril lengths change the values of the interfibrillar shear properties (i.e., τ_p for the plastic model and G for the elastic model), the qualitative behavior of the models themselves is unaltered (Szczesny and Elliott, 2014). Regarding the interfibrillar shear properties, there are limited data in the literature to compare with the model parameters. Nevertheless, macroscale testing of human ligament produced a shear modulus of less than 1 kPa (Bonifasi-Lista et al., 2005), which is comparable to the plastic interfibrillar shear stress (τ_p). Finally, while ~ 1

P_a is a relatively low modulus value, the large shear strains that occur in the interfibrillar matrix during fibril sliding produce high shear stresses, causing the interfibrillar matrix in the elastoplastic model to reach the plastic limit ($\epsilon_{p,i}$) at approximately 1% tissue strain. Therefore, while these parameter values may vary across tendons, tissues, and species, the representative values used here are reasonable for rat tail tendon fascicles and for the study objective of comparing various model formulations against multiscale fascicle mechanics.

One limitation of this study is that only a single experimental dataset of rat tail tendon fascicles was used to evaluate the performance of the models. While rat tail tendon fascicles are a widely used tissue model for tendon (Folkhard et al., 1987; Hansen et al., 2009; Kastelic et al., 1978; Li et al., 2013; Reese, 2013; Screen et al., 2004; Svensson et al., 2013; Torp et al., 1975), fascicles from other tendons may behave differently. For example, the plateau in the macroscale stress observed in rat tail fascicles (Fig. 4A and (Szczeny and Elliott, 2014)) also occurs in the common digital extensor tendon (Screen et al., 2013) but not in the Achilles tendon or superficial digital flexor tendon, at least up to 10% strain (Kahn et al., 2010; Screen et al., 2013). Furthermore, multiscale tissue mechanics are known to vary with tissue type, age, and cross-linking (Folkhard et al., 1987; Han et al., 2013; Li et al., 2013; Puxkandl et al., 2002; Svensson et al., 2013; Thorpe et al., 2013b; Torp et al., 1975). Nevertheless, fibril:tissue strain ratios less than one have also been observed in several tendons and tissues (Han et al., 2013; Liao et al., 2005; Rigozzi et al., 2011; Thorpe et al., 2013b), which suggests that discontinuous fibrils are not unique to rat tail tendon. Future investigations can apply the modeling principles presented in this study to other collagenous tissues in order to compare the microscale deformation mechanisms and determine whether the interfibrillar matrix in these tissues also behaves plastically.

6. Conclusions

This study demonstrated that incorporating plasticity of the interfibrillar matrix in shear lag models is necessary to reproduce the multiscale tendon fascicle mechanics. While elasticity of the interfibrillar matrix may contribute to the initial tissue behavior, only plastic interfibrillar deformation could replicate the macroscale strain-softening as well as the increasing interfibrillar sliding and decreasing fibril:tissue strain ratio observed at microscopic length scales. This suggests that plastic deformation of the interfibrillar matrix is responsible for the post-yield behavior of tendon fascicles. Identifying the mechanisms that reduce the tissue's ability to bear load is necessary to understand the initiation and progression of tendon pathology ultimately leading to complete rupture. This knowledge can then be used to restore proper tendon function by repairing the underlying structural defects through tissue regeneration. Finally, determining the fundamental structure-function relationships in tendon is essential for designing tissue engineered replacements that effectively reproduce native tissue behavior.

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- Various models were applied to tendon fascicle multiscale mechanics.
- Fibril yielding could not explain microscale post-yield behavior.
- Only shear lag models assuming plasticity of interfibrillar matrix were successful.
- Suggests fascicle yielding is due to plastic deformation of interfibrillar matrix.

Figure 1. Tendon hierarchical structure (adapted from Kastelic et al. (1978)). Collagen molecules are organized into extracellular structures called fibrils, which are the primary tensile load bearing elements in tendon. The fibrils are highly aligned and tightly packed into fascicles, which are the final macroscopic subunits in tendon. Within each fascicle are cells and an interfibrillar matrix surrounding the fibrils containing proteoglycans, elastin, and other macromolecules.

Figure 2.

The construction of the shear lag models consists of a periodic array of discontinuous and staggered fibrils. Tensile load was transferred between fibrils through an interfibrillar shear stress ($\tau(x)$).

Figure 3.

Comparison of the fibril stress (σ), interfibrillar shear stress (τ), and fibril displacements (u) along the unit cell length in the elastic and plastic shear lag models with increasing applied tissue strain (from row 1 to 3). (Row 1: A–D) Initially, when L_s is less than L_c , there is less interfibrillar sliding (i.e., smaller region where $u_1 - u_2$) in the plastic model compared to the elastic model ($\varepsilon_{uc} = 0.5\%$). (Row 2: E–H) As the applied tissue strain increases, L_s becomes similar to L_c and the amount of interfibrillar sliding is approximately the same between the two models. The fibril stress distribution and point at which the interfibrillar shear stress approaches zero are also very similar between the two models ($\varepsilon_{uc} = 1.4\%$). (Row 3: I–L) At higher applied strains, L_s continues to increase until $L_s = L/4$, where the interfibrillar sliding and shear stress occur across the entire fibril length, maximally loading the fibrils. In contrast, the elastic model produces identical distributions of stresses and displacements since the characteristic length over which loads are transmitted between fibrils (L_c) remains constant. Model parameters: $E_f = 700$ MPa, $r = 85$ nm, $\varphi = 0.7$, $\tau_p = 0.275$ kPa, $G = 1$ Pa, $L = 10$ mm.

Figure 4.

Model predictions of tendon fascicle multiscale mechanics (mean $\pm$ standard deviation) (Szczeny et al., 2014). (A,B) The poor performance of a continuous fibril model (dashed line) in reproducing (A) the tissue stress response and (B) the fibril:tissue strain ratio suggests that fibrils are indeed discontinuous. The elastic shear lag model (dotted line) provides only marginally improved results since, after the fibrils are uncrimped, it predicts a constant tissue modulus and a constant fibril:tissue strain ratio. However, the plastic shear lag model (solid line) can accurately replicate the macroscale strain-softening as well as the continuous drop in the fibril:tissue strain ratio, suggesting that plasticity of the interfibrillar matrix explains tendon fascicle mechanics. Model parameters: $E_f = 700$ MPa, $r = 85$ nm, $\varphi = 0.7$, $\tau_p = 0.275$ kPa, $G = 0.5$ Pa, $L = 10$ mm, $\bar{\lambda}_c = 1.003$, and $\lambda_c^{SD} = 0.0017$. (C,D) The effects of fibril yielding were approximated by a 30% reduction in the fibril modulus (red lines). (C) While fibril yielding may enable the continuous and elastic models to replicate the macroscale strain-softening, (D) it does not improve the prediction of the fibril:tissue strain ratio. However, for the plastic model, fibril yielding may improve the agreement with the experimental fibril:tissue strain ratios at higher applied tissue strains.

Figure 5.

Distributions of the fibril stress (σ), interfibrillar shear stress (τ), and fibril displacements (u) along the unit cell length for the elastoplastic shear lag model. (A–C) At strains below $\varepsilon_{p,i}$, the model behaves identically to the elastic shear lag model since $\tau(x) < \tau_p$. (D–F) As the applied strain increases, the interfibrillar shear stress becomes plastic at the fibril ends (L_p) and the region of interfibrillar sliding (i.e., $u_1 - u_2$) enlarges towards the center of the unit cell. (G–I) Ultimately, at a strain of $\varepsilon_{p,f}$ the interfibrillar shear stress has become plastic along the full fibril length ($L_p = L/4$). Model parameters: $E_f = 700$ MPa, $r = 85$ nm, $\phi = 0.7$, $\tau_p = 0.275$ kPa, $G = 0.5$ Pa, $L = 10$ mm.

Figure 6.

Effect of initial elasticity of the interfibrillar matrix on model predictions of tendon fascicle multi-scale mechanics (mean $\pm$ standard deviation) (Szczesny et al., 2014). (A) At the macroscopic level, the elastoplastic model produced a tissue response that was very similar to the plastic model. (B) While the elastoplastic model improves the prediction of the fibril:tissue strain ratio at low applied tissue strains, the post-yield behavior is still dominated by plasticity of the interfibrillar matrix. Model parameters: $E_f = 700$ MPa, $r = 85$ nm, $\varphi = 0.7$, $\tau_p = 0.275$ kPa, $G = 0.5$ Pa, $L = 10$ mm, $\bar{\lambda}_c = 1.003$, and $\lambda_c^{SD} = 0.0017$.

Table 1**Nomenclature****General Terms**

E_f	Fibril modulus
L	Fibril length
P	Fibril stress at fibril midpoint
r	Fibril radius
U	Total elongation of unit cell
u_i	Displacement of i -th fibril
x	Position along unit cell
Γ	Gamma cumulative distribution function
γ	Gamma probability distribution function
ε_{uc}	Unit cell strain
ε_{app}	Apparent fibril strain
η_{app}	Apparent fibril:tissue strain ratio
λ	Tissue stretch
λ_c	Tissue stretch required to uncrimp unit cell
$\overline{\lambda_c}$	Mean uncrimping stretch
λ_c^{SD}	Standard deviation of uncrimping stretches
σ_i	Fibril stress of i -th fibril
σ_{uc}	Average stress in unit cell
σ_T	Macroscale tissue stress
τ	Interfibrillar shear stress
	Fibril volume fraction

Elastic Model

E_e	Macroscale tissue modulus
G	Shear modulus of interfibrillar matrix
h	Surface-to-surface distance between fibrils
L_c	Characteristic load transfer length
η_e	Fibril:tissue strain ratio

Plastic Model

E_p	Macroscale tissue modulus
L_s	Length over which interfibrillar sliding occurs
ε_p	Unit cell strain where interfibrillar sliding occurs over entire fibril length
η_p	Fibril:tissue strain ratio
τ_p	Interfibrillar shear stress due to interfibrillar sliding

Elastoplastic Model

L_p	Length over which interfibrillar shear stress is plastic
$\varepsilon_{p,i}$	Unit cell strain at which interfibrillar shear first becomes plastic

$\varepsilon_{p,f}$	Unit cell strain where interfibrillar shear is plastic over entire fibril length
τ_p	Plastic limit for interfibrillar shear stress

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Table 2

Model Parameter Values

E_f	700 MPa
L	10 mm
r	85 nm
	0.7
G	0.5 – 1 Pa
τ_p	0.275 kPa
$\overline{\lambda_c}$	1.003
λ_c^{SD}	0.0017

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