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Micromechanical poroelastic finite element and shear-lag models of tendon predict large strain dependent Poisson's ratios and fluid expulsion under tensile loading

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ABSTRACT

As tendons are loaded, they reduce in volume and exude fluid to the surrounding medium. Experimental studies have shown that tendon stretching results in a Poisson's ratio greater than 0.5, with a maximum value at small strains followed by a nonlinear decay. Here we present a computational model that attributes this macroscopic observation to the microscopic mechanism of the load transfer between fibrils under stretch. We develop a finite element model based on the mechanical role of the interfibrillar-linking elements, such as thin fibrils that bridge the aligned fibrils or macromolecules such as glycosaminoglycans (GAGs) in the interfibrillar sliding and verify it with a theoretical shear-lag model. We showed the existence of a previously unappreciated structure–function mechanism whereby the Poisson's ratio in tendon is affected by the strain applied and interfibrillar-linker properties, and together these features predict tendon volume shrinkage under tensile loading. During loading, the interfibrillar-linkers pulled fibrils toward each other and squeezed the matrix, leading to the Poisson's ratio larger than 0.5 and fluid expulsion. In addition, the rotation of the interfibrillar-linkers with respect to the fibrils at large strains caused a reduction in the volume shrinkage and eventual nonlinear decay in Poisson's ratio at large strains. Our model also predicts a fluid flow that has a radial pattern toward the surrounding medium, with the larger fluid velocities in proportion to the interfibrillar sliding.

1. Introduction

Tendons function as mechanical, load-bearing structures that allow motion by transmitting forces from muscle to bone. The composition and organizational structure of tendon are optimized to allow its mechanical response for a range of stresses and strains. Under tensile loading, tendon exhibits shrinkage of volume [1] and fluid exudation to the surrounding medium [2,3]. By defining the Poisson's ratio as $(\nu_{eff} = (1 - \Delta V/(\varepsilon V_0))/2)$, where $(\Delta V/V_0 < 0)$ is the negative relative volume change at a tensile strain (ε), the Poisson's ratio for tendon is expected to be larger than 0.5. Indeed, reported Poisson's ratio for tendon is 1.65 ± 0.35 for human hip joint ligament [4], 2.98 ± 2.59 for sheep flexor tendon [5], and 0.7 ± 0.52 for rat tail tendon fascicles [6]. Yet, mechanical models replicating this experimental behavior have been limited. Understanding the micromechanical response of tendon is therefore important to fully describe its material behavior from the macro to microstructural levels.

Tendons are composed of a dense extracellular matrix consisting primarily of collagenous and noncollagenous components. Modeling tendon as a distribution of fibrils embedded in a poroelastic matrix where the matrix adopts a Poisson's ratio within the range of the homogenous isotropic materials (i.e. 0–0.5), predicts that tendons swell during tensile loading by absorbing fluid from the surrounding medium. This behavior is in contrast to the above experimental results [1–3] and to overcome this contradiction in previous poroelastic studies, the measured macroscopic Poisson's ratio for the whole tendon (2.5 for sheep flexor [7] and 1.7 for rat tail [8]) was input as the microscopic Poisson's ratio for the extracellular matrix (ECM). These large Poisson's ratios for the ECM lead to the shrinkage of the matrix under tensile loading, and as a result, such models are capable of explaining the fluid exudation, although a concrete justification to equate the tendon and ECM Poisson's ratios has not been presented. In addition these models are unable to predict the nonlinear variation of the

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Poisson's ratio with strain; experiments have shown that tendons exhibit a large Poisson's ratio (~6) at low strains that decays at high strains [9].

The biphasic behavior of tendon components implies that both collagen fibrils and the non-collagenous matrix play a role in stress transfer during uniaxial loading [10–17]. For example, proteoglycans (PGs) interact with and bind to type I collagen fibrils [18,19] at discrete sites with their protein cores, and their associated glycosaminoglycans (GAGs) extend into the interfibrillar matrix [19–22]. Evidence for binding of the GAG chains to certain domains on like molecules or to each other [23–26] suggests that GAGs may act as interfibrillar-links that contribute to fibril–fibril communication. Although recent models [27] have suggested that the contributions of PG-associated GAGs are greatest in the context of short, discrete fibrils likely during tendon *development* and *healing*, experimental studies in *mature uninjured* tendons have shown that, enzymatic digestion of GAGs does not induce changes in mechanical stiffness [15,16,28–30]. Still, the potential interfibrillar-linking role of secondary collagen fibers such as type VI and XII or other molecules such as elastin [31,32] must be considered.

In addition to the macromolecules, variation in the morphology of the fibrils is a potential alternative mechanism that influences the pathway of the load transfer between the fibrils. Collagen fibrils are predominantly aligned in parallel along the direction of loading, in the form of the well-organized bundles [33,34]. Among all the fibrils, electron microscopy has identified smaller diameter fibrils that traverse and bifurcate with larger diameter fibrils [17,35,36]. Experimental studies have shown that under tensile loading, there is discrepancy between the strains measured in the fibrils and applied to the tissue and this strain is compensated by interfibrillar sliding [37–39]. These thin fibrils bridging between aligned fibrils may regulate the interfibrillar sliding and contribute to the force transmission mechanism between fibrils [17]. Here we present a computational model to show that the potential interfibrillar-linking contribution of the bridging fibrils or macromolecules such as GAGs in combination with the existing interfibrillar sliding, remarkably leads to the fluid exudation and the Poisson's ratio larger than 0.5 under tensile loading.

Therefore, the objective of this study is to develop a micromechanical poroelastic model to (1) explain the experimental observation of large Poisson's ratios and its variation with strain and (2) quantify fluid flow directionality and velocity along fibrils. Our model is based on the force transmission between the fibrils through interfibrillar-linking elements which are modeled as elastic springs. These interfibrillar-linkers can represent thin fibrils that are bridging between the aligned fibrils or GAGs and other potential interfibrillar-linking elements such as collagen type VI and XII. Given the uncertainty in the current literature about the frequency and stiffness of the bridging fibrils, we perform a parametric study on the elastic stiffness and density of these interfibrillar-linkers. To produce the interfibrillar sliding, fibrils in our model are modeled as discontinuous elements embedded in the ECM. In this setting, under tensile loading, the relative displacement between the adjacent fibrils can represent the interfibrillar strain as observed in the experimental results. The importance of the current model is in part to show that while the Poisson's ratio of the tendon constituents such as collagen fibril and matrix can be within the range of the homogenous isotropic materials (i.e. 0–0.5), yet the macroscopic Poisson's ratio is larger than 0.5. We used a two-prong approach, incorporating both a three-dimensional finite element model to predict the tendon Poisson's ratio and the fluid flow direction and velocity, as well as a simple shear lag model to explain the micromechanical mechanism behind the observed Poisson's ratio variation with strain.

2. Methods and materials

Our finite element method (FEM) tendon model is comprised of (i) a staggered distribution of collagen fibrils, (ii) interfibrillar-linking elements between the fibrils which can represent bridging fibrils or GAGs, and (iii) the ECM that envelopes all of the components (Fig. 1). In this model, fibrils are assumed to be 1-D elastic elements (with Young's modulus, E_f and Poisson ratio, ν_f) with length (L), radius (R_f) and center-to-center distance of (d_f). The interfibrillar-linkers are also modeled as elastic springs with stiffness (K) and spacing (d) along the length of the fibrils (Table 1).

The third component, the ECM, is modeled as a biphasic porous material that is saturated with fluid and is coherently bonded to the fibrils. By applying mechanical loading to the ECM, a fluid pressure gradient is created resulting in movement of the fluid. Darcy's Law was applied to connect the fluid flow velocity to the pressure gradient in the ECM:

$$\vec{V} = -\frac{k}{n}\nabla P \tag{1}$$

In Eq. (1), $\vec{V}$ is the fluid velocity field (m/s), P is the fluid pressure (Pa), k is the ECM permeability (m^4/Ns), and n is the matrix porosity, defined as the volume fraction of the pores in the matrix.

The fluid flow is related to the deformation of the material through the continuity equation

$$\frac{\partial \varepsilon_{vol}}{\partial t} + \nabla \cdot (-k\nabla P) = 0 \tag{2}$$

where ε_{vol} is the volumetric strain of the matrix.

In addition, the mechanical equilibrium equation should also be solved for the matrix

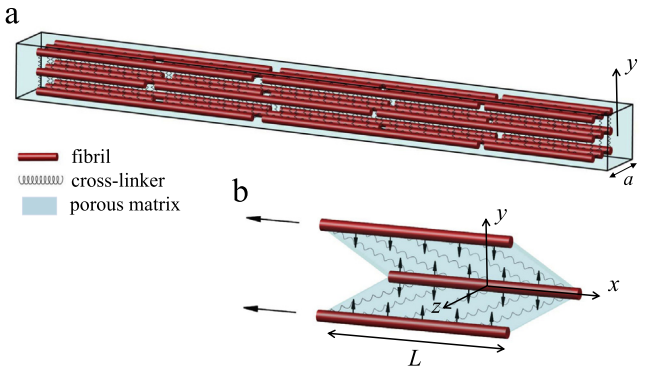

Fig. 1. (a) The finite element model consists of staggered fibrils (red rods) interconnected with interfibrillar-linkers (springs) placed in a porous medium (cyan). The coordinate system is placed at the center of the model with x-axis directed along the fibril orientation, and y-axis perpendicular to it. (b) Under tensile loading, interfibrillar-linkers exert a compressive force to the biphasic medium (vertical arrows) and cause the fluid to flow radially outward from the encapsulated matrix. The aspect ratio in the figure is not in scale.

Table 1
Definitions and values for symbols used in the model.

Symbol	Definition	Value	References
L	Fibril length	100 μm	[25]
E_f	Fibril Young's modulus	1.5 GPa	[25,42]
ν_f, ν_m	Fibril and matrix Poisson's ratio	0.3	[42]
G_m	Matrix shear modulus	0.1 MPa	[5,8,40,41]
d_f	Fibril center-to-center spacing	300 nm	[25]
R_f	Fibril radius	100 nm	[25]
k	Matrix permeability	3.08e−14 m^4/Ns	[8]
n	Matrix porosity	2/3	[8,43]