Swelling Affects Failure Mechanics of the Annulus Fibrosus

Minhao Zhou, Grace D. O'Connell
University of California, Berkeley, Berkeley, CA
g.oconnell@berkeley.edu

Disclosures: The authors have nothing to disclose.

INTRODUCTION: During daily activities, the avascular fiber-reinforced annulus fibrosus (AF) experiences large tensile stresses that can cause tears or degenerative remodeling [1, 2]. Understanding AF failure mechanics is important for preventing damage accumulation and for developing biological tissue repair strategies. The AF is comprised mostly of collagen fibers embedded within a highly hydrated proteoglycan-rich extrafibrillar matrix, which provides the tissue with its excellent capacity for absorbing water (>75%/ww) [3]. Previous studies showed that tissue swelling alters interfibrillar spacing and decreases tissue stiffness by ~50% [4, 5]. Finite element models are powerful tools that allow researchers to understand the role of sub-tissue anatomy on tissue-level mechanics, which is difficult to measure experimentally. However, there is a lack of structurally based models that can explicitly describe fiber-matrix interactions and tissue-swelling behaviors, because the majority of tissue-level models are developed based on homogenization and cannot be used to elucidate the roles of fiber-matrix interactions [6]. Therefore, the objective of this study was to investigate the effects of tissue swelling and damage on AF mechanics (sub-failure & failure mechanics) using a structurally based model, where the fibers and matrix are described as two distinct materials that occupy separate space in the model [7]. We hypothesized that swelling would pre-strain the tissue, especially the collagen fibers, prior to tensile loading, causing early tissue failure. Tissue-level models were based on properties of the outer AF; however, the methods used are applicable to other fiber-reinforced tissues.

METHODS: An AF 'separate model' was created with 4 lamellae (0.2 mm/lamella) and fibers were modeled as full-length cylinders oriented at ± 30º welded to the matrix (Fig. 1A; ~2x10^6 tetrahedral elements; tissue dimensions: 4.8, 2, and 0.8 mm for length, width, and thickness) [8]. Modeled tissue geometry was developed based on our previous experimental work, which ensured tissue failure at the mid-length to measure true failure properties (Fig. 1A, 25% of cross sectional area remained at mid-length) [9, 10]. Triphasic mixture theory (***triphasic model)*** was used to describe tissue swelling. The matrix was modeled as a compressible hyperelastic material (Neo-Hookean material) with parameters curve fit to data presented in [11]. Collagen fibers were modeled as a compressible hyperelastic material (Holmes-Mow material with exponential-linear fiber description) with parameters curve fit to data presented in [10]. The fixed charge density represents the glycosaminoglycan content and was set to -100 mmol/L for the matrix and 0 mmol/L for fibers (*i.e.,* no active swelling). Fluid (*i.e.,* water) and ion phases (Na^+ and Cl^-) were included to simulate the swelling response (ion free diffusivity = 0.00147 mm^2/s, ion diffusivity within tissue = 0.0008 mm^2/s). Tissue permeability was strain-dependent (Holmes-Mow permeability: k_0 = 0.0064 mm^4/(Ns) for matrix and 0.0032 mm^4/(Ns) for fibers; M = 4.8; α = 2). Reactive damage mechanics was applied to simulate tissue failure behaviors and maximum Lagrangian strain served as the failure criterion. Quintic cumulative distribution functions were used to describe damage evolution ($\mu_{min,EFM}$ = 1.0, $\mu_{max,EFM}$ = 1.7, $\mu_{min,fiber}$ = 0.5, and $\mu_{max,fiber}$ = 0.8) [9, 11]. A ***hyperelastic model*** (no tissue swelling) was developed to serve as the control. Steady-state free swelling was performed under 0.15 M saline, followed by uniaxial tension to 20% global engineering strain. Tissue swelling and change in fiber diameter was measured for model validation. Then, the linear-region modulus was calculated at ~17.5% global engineering strain and bulk tissue failure properties were evaluated.

RESULTS: The bulk tissue volume increased by more than 30% with swelling, and a 12% increase in fiber diameter was observed in the fibers due to swelling of the matrix (Fig. 1B). Damage was initiated within collagen fibers that were exposed on the tissue surface, while the majority of damage at failure accumulated in both the fibers and matrix at the mid-length (Fig. 1C). Tissue swelling had a slight impact on the linear-region modulus (Fig. 1D - black vs. red solid lines; triphasic model: 15.5 MPa; hyperelastic model: 16.9 MPa). Inclusion of the damage description decreased the linear-region modulus by ~50% (Fig. 1D - solid vs. dashed lines; triphasic: 51% decrease to 7.9 MPa, hyperelastic: 56% decrease to 9.5 MPa). Including swelling behavior with the damage description resulted in earlier predictions of damage initiation (Fig. 1D - '+'; triphasic vs. hyperelastic: 9 vs. 12% global strain) and failure (Fig. 1D - '*'; 1.2 vs. 1.3 MPa, 20.5 vs. 22.5%, respectively). Lastly, swelling induced residual strains within the tissue, prior to tension (Fig. 1E - blue circles do not pass through the origin), which also decreased the overall matrix modulus (Fig. 1E - slope of blue vs. red: 0.08 vs. 0.11 MPa).

DISCUSSION: We investigated the effects of tissue swelling and damage on AF mechanics using a structurally based finite element model. To do this, triphasic and hyperelastic models were developed by curve fitting material parameters to bulk tissue data reported in the literature [9, 11]. Tissue damage was initiated in collagen fibers located on the tissue surface and propagated to the adjacent matrix, which is consistent with a recent tissue damage mechanics study that used a combined particle/continuum approach [12]. Interestingly, bulk tissue swelling in the triphasic model caused a ~12% increase in the collagen fiber diameter (Fig. 1B), suggesting transverse fiber deformations are largely due to mechanical stresses from matrix swelling. Our simulated response agrees well with previous observations in the literature that reported an increase in fiber diameter; however, we did not observe a decrease in interfibrillar spacing; changes in fibril spacing may be altered with a higher fixed charge density [4, 5]. Tissue swelling increased tissue volume, resulting in residual stresses and strains, without external mechanical loading; hence, the triphasic model experienced a lower stress and strain at failure (Fig. 1D). These findings suggest that an increase in water content in fiber-reinforced materials (*e.g.,* higher glycosaminoglycan content or extended bed rest) may make the tissues more susceptible to failure under mechanical loading. Future work will investigate whether tissues with greater fixed charge density, such as the inner AF, will increase swelling-induced residual stresses and decrease the stress and strain at failure under mechanical loads. In conclusion, tissue swelling and, therefore, glycosaminoglycan content plays a crucial role in failure mechanics of fiber-reinforced tissues [13].

SIGNIFICANCE: The findings from this study demonstrate the importance of tissue swelling on annulus fibrosus failure mechanics, which will be important for understanding annular tears with aging and degeneration, as well as designing biological repair strategies for fiber-reinforced tissues.

REFERENCES: [1] Vernon-Roberts+, Spine, 2007; [2] O'Connell+, Biores, 2015; [3] Bezci+, JBME, 2015; [4] Screen+, Acta Biomat, 2006; [5] Han+, Ann Biomed Eng, 2012; [6] Yin+, JBM, 2005; [7] Yang+, SB^{3}C, 2016; [8] Marchand+, Spine, 1990; [9] Holzapfel+, BMMB, 2005; [10] Werbner&Zhou, JBME, 2017; [11] Fujita+, JOR, 1997; [12] Rausch+, BMMB, 2016; [13] Werbner+, SB^{3}C 2016.

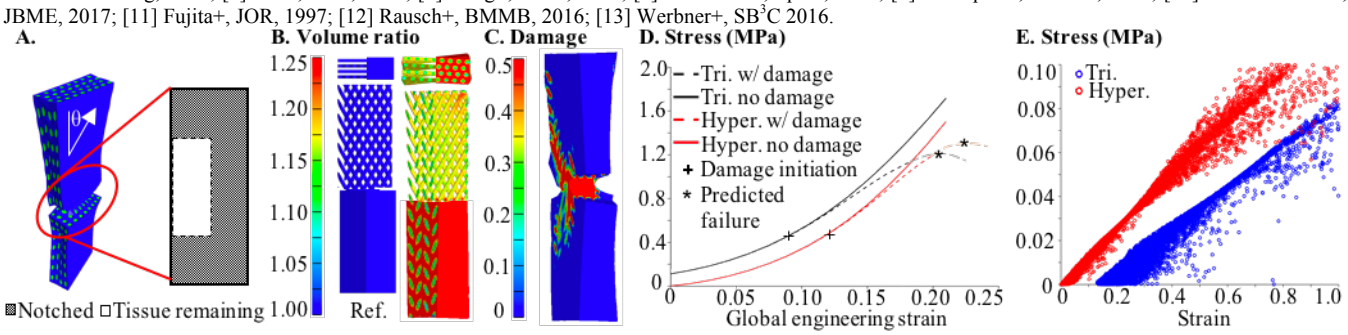

Fig. 1 (A) Schematic of the separate AF model in tension (blue: matrix; green: fibers). *Zoomed:* mid-length notch geometry. (B) Tissue relative volume with free-swelling (Ref.: reference configuration). (C) Tissue failure in tension. (D) Stress-strain response (Tri: triphasic; Hyper: hyperelastic; dashed lines of lighter colors: predicted damage curves). (E) Stress-strain response of the extrafibrillar matrix under uniaxial tension.