Hydration Mitigates Rapid Changes in Microscopic Strains of the Annulus Fibrosus During Tensile Loading

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INTRODUCTION: There are conflicting data in the literature with respect to the role of hydration on intervertebral disc injuries. Specifically, the effect of hydration on annulus fibrosus (AF) tissue-level failure mechanics is not well understood. Clinically, injuries and annular tears are associated with aging and degeneration [1], but fully hydrated discs also experience a greater risk of *in vitro* injury, due to an increase in stress during bending [2-3]. Finite element models are powerful tools for predicting stress-strain distributions in complex, fiber-reinforced tissues. For example, our previous work developed a model to accurately predict AF failure location during uniaxial tension [4]. More recently, we developed and validated a structure-based tissue-level model of the AF to study stress distributions between fibers and the surrounding matrix [5]. In this study, we applied the structure-based model to evaluate the effect of hydration on AF failure mechanics. We tested the hypothesis that a sufficient tissue hydration protects tissue from failure, but overhydration would increase the likelihood of premature tissue-level failure by initiating damage in fiber bundles.

METHODS: AF models were developed with four lamellae (0.2 mm/lamella) to represent rectangular specimens commonly used in uniaxial tensile tests (dimensions: 8.0, 2.0, 0.8 mm for length, width, thickness, respectively). Fibers were welded to the extrafibrillar matrix (EFM) and oriented at ±45° and ±30° to represent the fiber architecture of the inner and outer AF (IAF, OAF), respectively. A midlength notch was included to ensure tissue midlength failure [4]. Triphasic material descriptions were applied to model tissue swelling behaviors. The tissue solid volume fraction was 30% with strain-dependent permeability. The EFM and fibers were described as compressible hyperelastic materials (EFM: Neo-Hookean material; fibers: Holmes-Mow material with an exponential-linear fiber description). The fixed charge density was -250 mmol/L in the IAF, -125 mmol/L in the OAF [6], and 0 mmol/L in fibers [7]. A quintic function was used to describe tissue damage accumulation using the maximum Lagrangian strain criterion. Material and damage parameters were determined based on data in [8]. Swelling of IAF and OAF specimens were simulated under 2.0, 0.15, or 0.075 M saline conditions to represent dehydrated, hydrated, or overhydrated tissue specimens. Microscopic strain (ε_{micro}) distributions in tissue subcomponents (*i.e.*, EFM and fibers) were evaluated. The swelling was followed by a 20% uniaxial tension. To calculate stress-stretch response during the applied tension, the point prior to tension and after swelling was defined as the reference configuration to replicate the reference configuration used experimentally. At the point of predicted bulk tissue failure, failure strain (ε_f) was calculated and ε_{micro} distribution was evaluated. Damage accumulation in tissue subcomponents was also assessed.

RESULTS: Tissue volume of dehydrated, hydrated, and overhydrated specimens increased with swelling by 10, 70, and 120% for IAF specimens, and 7, 50, and 70% for OAF specimens, respectively. ε_{micro} followed a normal distribution and increased with an increase in hydration. For dehydrated IAF specimens, the average ε_{micro} in the EFM and fibers was below 5% (Fig. 1A - left column - light orange), and no tissue damage was observed prior to tension (Fig. 1B - top - orange lines). An increase in water absorption greatly increased the mean of ε_{micro} distribution in the EFM and fibers (Fig. 1A - light green or blue). However, greater hydration of IAF specimens resulted in >15% fiber damage accumulation prior to tension (Fig. 1B - dashed green or blue lines). Similarly, for OAF specimens, greater hydration increased the mean of ε_{micro} distribution in the EFM and fibers (Fig. 1A - right column - light colored bins). However, no damage was observed in either subcomponent prior to tension across all hydration levels (Fig. 1B - bottom, all lines started at the origin). Increased hydration decreased bulk AF modulus (Fig. 1C) and helped maintain the mean, spread, and peak of ε_{micro} distributions in both the EFM and fibers throughout uniaxial tension (Fig. 1A - light versus dark bins). Bulk tissue failure strain was almost identical across all hydration levels for specimens from the same anatomical location (Fig. 1C - 'X'; $\varepsilon_{f,\,IAF} \approx 15\%$; $\varepsilon_{f,\,OAF} \approx 11\%$), except for the overhydrated IAF specimen, which experienced premature bulk tissue failure ($\varepsilon_f < 5\%$) due to the excessive damage accumulation during swelling (Fig. 1B - top - dashed blue line).

DISCUSSION: We investigated the effects of hydration on AF failure mechanics using a structure-based finite element model. Swelling, through more water absorption, reduced bulk tissue modulus, which was consistent with the literature (Fig. 1C) [9]. Tissue damage was initiated in the fibers and propagated to the adjacent EFM, which confirmed our hypothesis and was consistent with a recent computational study that evaluated damage mechanics of fiber-reinforced tissues [10]. Prior to uniaxial tension, microscopic strains in hydrated specimens were at least four times greater than strains in dehydrated specimens, increasing the amount of damaged elements prior to mechanical loading. Interestingly, damage accumulation and greater microscopic strains due to swelling did not result in earlier bulk tissue failure during mechanical loading. That is, failure strain of OAF specimens was not dependent on the initial hydration condition (Fig. 1C - 'X's). Hydrated specimens had a smaller shift in peak microscopic strains during tension, where the average microscopic strain in dehydrated samples increased by 400-500%, but hydrated samples had less than a 50% shift in microscopic strains during loading (Fig. 1A - lengths of the arrows). Taken together, these findings suggest that sufficient tissue hydration acts to protect fiber-reinforced tissues from catastrophic failure by mitigating rapid changes in microscopic strains under mechanical loading and preventing overloading the microstructure. However, overhydration induced significantly larger strains, especially in the IAF, making the tissue more susceptible to early failure during mechanical loading. Future work will evaluate the effects of hydration on AF failure mechanics for more complex loading conditions (*e.g.*, biaxial loading).

SIGNIFICANCE: These findings demonstrate that a sufficient tissue hydration level is crucial for preventing bulk tissue failure, while overhydration may make the tissue more susceptible to mechanical failure, due to an increase in local stresses and strains during swelling. These findings are important for understanding mechanisms for herniated discs and annular tears with aging and degeneration, as well as for designing biological repair strategies.

REFERENCES: [1] Adams+, Spine, 2006; [2] Adams+, Spine, 1986; [3] Wade+, Spine, 2014; [4] Werbner+, JBME, 2017; [5] Zhou+, ORS, 2018; [6] Urban+, BBA, 1979; [7] Huyghe+, BMMB, 2003; [8] Holzapfel+, BMMB, 2005; [9] Han+, Ann Biomed Eng, 2012; [10] Rausch+, BMMB, 2016.

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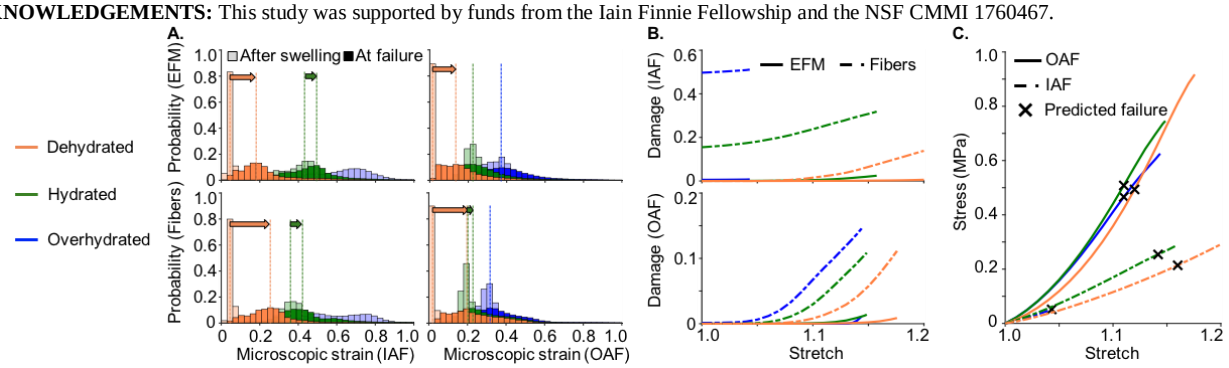

Fig. 1 (A) Microscopic strain distribution in EFM (top) and fibers (bottom) after swelling (light bars) and at failure (dark bars). The arrow represents the shift in average microscopic strain during tension. Specimens without an arrow had a negligible shift in the average strain during tension. Strain distribution of overhydrated IAF was not shown due to premature failure. (B) Damage accumulation for IAF and OAF during tension. (C) Stress-stretch response.