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An anisotropic multiphysics damage model with application to annulus fibrosus

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Abstract

An anisotropic multiphysics damage model is developed to characterize the couplings among multiple physical fields within soft tissues and the tissue damage based on thermodynamic principles. This anisotropic multiphysics damage model integrates the continuum mixture theory and a continuum damage model, and the anisotropic damage is considered by evolution of internal damage variables governing the anisotropic mechanical behaviors of tissues. The energy dissipation associated with the transport of fluid and ions is generally related to tissue damage. The anisotropic multiphysics damage model is applied to simulate a case of annulus fibrosus (AF) damage in an isolated intervertebral disc under compression, to understand the damage initiation and propagation. It is found that, for this case (with 1000 N/s of compression rate and neglected ground matrix damage), the damage initiated in the outer and middle posterior regions of AF at about 700 N of disc compression. The region-dependent yield stretch ratio predicted by this model is consistent with experimental findings. A sensitive study on the damage parameters is also presented. This study provides an additional insight into AF damage in the isolated disc under mechanical compression.

Keywords

intervertebral disc; continuum damage; multiple physical fields; finite element analysis

1. Introduction

Abnormal mechanical loading is a major factor that can cause the degeneration of intervertebral discs (IVDs). One possible mechanism is that abnormal mechanical loading causes the damage in annulus fibrosus (AF, the outer ring of IVD), thus, altering the biomechanics of IVD. This is evidenced by the strong association between the damage of

Conflict of Interest

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AF and the disc degeneration by clinical observation, postmortem studies, and animal studies (Dullerud and Johansen, 1995; Masuda et al., 2005; Osti et al., 1990; Sharma et al., 2009; Yu et al., 1989). The AF is a hydrated soft tissue of multi-laminated structure which consists of obliquely oriented collagen fiber bundles in each lamella, with alternatively oriented fiber bundles in adjacent lamellae. The AF is subjected to complicated mechanical loading conditions during daily activities, such as compression and flexion, resulting in different patterns in AF damage. One of the typical damage pattern is the herniation of disc, which usually occurs in the posterior, or posterolateral AF regions (Maezawa and Muro, 1992; Ninomiya and Muro, 1992; Veres et al., 2009).

Many in vitro experiments have been conducted to study the association of different loading regimes with the catastrophic damage of AF. Studies have shown that axial compression on motion segments (vertebra-disc-vertebra) would cause fracture of the vertebra or cartilaginous endplate (CEP) rather than the damage of AF (Brown et al., 1957; Lin et al., 1978; Roaf, 1960; Yoganandan et al., 1988), while compression combined with flexion (compression-flexion) would cause damage in the posterior AF (Adams and Hutton, 1982a, b; Aultman et al., 2005; McNally et al., 1993; Tampier et al., 2007). Studies also have shown that loading rates affect the damage of AF (Veres et al., 2010).

Numerical analyses of disc damage have also been conducted. In some previous computational studies, the AF was modeled as a fiber reinforced composite material, and the damage was evaluated with a maximum stress or strain criterion (Natarajan et al., 1994; Shirazi-Adl, 1989). It was reported that compression alone is unlikely to cause the damage of AF (Natarajan et al., 1994), whereas the load mimicking the symmetric lifting (combination of compression, shear, and flexion) combined with lateral bending or twisting is more likely to cause the damage of AF (Shirazi-Adl, 1989). Qasim et al. developed a computational model to study the fatigue damage of AF under dynamic loading conditions based on an S-N curve [stress (S) against the number of cycles to failure (N)] of AF, with which they found that the damage initiates at the posterior inner AF adjacent to the cartilage-endplate (CEP) (Qasim et al., 2012, 2014). However, these computational models are limited in describing the nonlinear couplings among solid deformation, fluid transport, and the electro-osmotic effect in the charged and hydrated disc tissues. These couplings among multiple physical fields have been shown to govern the mechanical behaviors of disc tissues (Iatridis et al., 2003).

Thus, the objectives of this study are: 1) to develop an anisotropic multiphysics damage model to account for the effect of nonlinear couplings among the multiple physical fields and tissue damage, and 2) to study the initiation and propagation of damage in the AF of isolated discs under compression. The anisotropic multiphysics damage model integrates the continuum mixture theory and continuum damage model. The continuum mixture theory has been widely used to describe the coupling of multiple physical fields within soft tissues as well as IVD (Ateshian, 2007; Gao et al., 2016; Gu et al., 1998; Lai et al., 1991; Zhu et al., 2014). Continuum damage model is developed based on irreversible thermodynamics with internal damage variables to represent the status of damage (Kachanov, 1958; Rabotnov, 1980). The continuum damage model has been widely used for modeling the damage of soft tissues (e.g., arterial walls)(Balzani et al., 2006; Famaey et al., 2013).

2. Model development

2.1 Anisotropic multiphysics model

An anisotropic multiphysics model was adopted to describe the couplings of multiple physical fields within the IVD (Gao et al, 2016). The IVD was modeled as a mixture consisting of solid (s), fluid (w), and solute phases (+ and – for cations and anions, respectively) based on the mechano-electrochemical continuum mixture theory (Ateshian, 2007; Gu et al., 1998; Huyghe and Janssen, 1999; Lai et al., 1991; Sun, 2002). The governing equations are briefly introduced in this paper.

The mass balance equation for the mixture can be written as:

$$\nabla \cdot \left(\sum_{\alpha} \phi^{\alpha} \mathbf{v}^{\alpha} \right) = 0, \quad \alpha = s, w, +, - \quad (1)$$

where ϕ^{α} and $\mathbf{v}^{\alpha}$ are the volume fraction and velocity of species α at current state, respectively. The above equation is derived with the assumptions that the mixture is saturated, that is, $\sum_{\alpha} \phi^{\alpha} = 1$; and each phase is intrinsically incompressible.

The balance equation of linear momentum for each phase α can be written as:

$$\nabla \cdot \boldsymbol{\sigma}^{\alpha} + \boldsymbol{\pi}^{\alpha} = 0, \quad (2)$$

where $\boldsymbol{\sigma}^{\alpha}$ is the stress of phase α , and $\boldsymbol{\pi}^{\alpha}$ is the momentum supplied to phase α by other phases.

The electroneutrality condition is assumed to be satisfied for every point in the disc, which gives,

$$\nabla \cdot \sum_{\beta} \frac{z^{\beta} \phi^{\beta}}{\tilde{V}^{\beta}} (\mathbf{v}^{\beta} - \mathbf{v}^s) = 0, \quad \beta = w, +, - \quad (3)$$

where z^{β} is the valence of phase β ($z^w = 0$), and $\tilde{V}^{\beta}$ is the partial molar volume of phase β .

Since the human body temperature keeps constant, it is reasonable to assume that all the processes occur under isothermal conditions. The entropy inequality for per mixture volume at current state can be written as:

$$\sum_{\alpha} \left[-\phi^{\alpha} \frac{D^{\alpha} A^{\alpha}}{Dt} + \boldsymbol{\sigma}^{\alpha} : \nabla \mathbf{v}^{\alpha} - \boldsymbol{\pi}^{\alpha} \cdot \mathbf{v}^{\alpha} \right] \geq 0, \quad (4)$$

where A^α is the Helmholtz free energy of phase α , and $\frac{D^\alpha}{Dt}$ is the material derivative of species α . This entropy inequality should hold under the constraints of Equations (1) and (3), therefore,

$$\sum_{\alpha} \left[-\phi^{\alpha} \frac{D^{\alpha} A^{\alpha}}{Dt} + \boldsymbol{\sigma}^{\alpha} : \nabla \mathbf{v}^{\alpha} - \boldsymbol{\pi}^{\alpha} \cdot \mathbf{v}^{\alpha} \right] + p \nabla \cdot \left(\sum_{\alpha} \phi^{\alpha} \mathbf{v}^{\alpha} \right) + \lambda \left[\nabla \cdot \sum_{\beta} \frac{z^{\beta} \phi^{\beta}}{\tilde{V}^{\beta}} (\mathbf{v}^{\beta} - \mathbf{v}^s) \right] \geq 0, \quad (5)$$

where p and λ are two Lagrange multipliers.

Transforming the above equation to per initial mixture volume by multiplying it with the volume ratio (J) between current volume and volume at reference state, we get

$$\begin{aligned} & -\frac{D^s W}{Dt} + J \boldsymbol{\sigma}^e : \nabla \mathbf{v}^s \\ & + J \sum_{\beta} \left\{ \left[\boldsymbol{\sigma}^{\beta} + \phi^{\beta} \left(p - A^{\beta} + \frac{\lambda z^{\beta}}{\tilde{V}^{\beta}} \right) \mathbf{I} \right] : \nabla (\mathbf{v}^{\beta} - \mathbf{v}^s) \right\} \\ & + J \sum_{\beta} \left\{ (\mathbf{v}^{\beta} - \mathbf{v}^s) \cdot \left[-\nabla (\phi^{\beta} A^{\beta}) + \left(p + \frac{\lambda z^{\beta}}{\tilde{V}^{\beta}} \right) \nabla \phi^{\beta} - \boldsymbol{\pi}^{\beta} \right] \right\} \geq 0 \end{aligned} \quad (6)$$

where $W = J \sum_{\alpha} \phi^{\alpha} A^{\alpha}$ is the Helmholtz free energy per initial mixture volume, and $\boldsymbol{\sigma}^e = \sum_{\alpha} \boldsymbol{\sigma}^{\alpha} + p \mathbf{I}$.

For an intact mixture, the energy function W is a function of solid phase deformation, the volume fraction of each phase $\Phi^{\alpha} (= J \phi^{\alpha})$, and the relative velocities to the solid phase $\mathbf{v}^{\beta s}$ [$= \mathbf{F}^{-1}(\mathbf{v}^{\beta} - \mathbf{v}^s)$ where $\mathbf{F}$ is the deformation gradient tensor]. Since the AF is a soft tissue reinforced by two families of fibers ($\theta = 1, 2$), the deformation of solid phase may be characterized by the right Cauchy-Green deformation tensor $\mathbf{C}$, and fiber structure tensors $\mathbf{N}_0^{\theta}$ ($= \mathbf{n}_0^{\theta} \otimes \mathbf{n}_0^{\theta}$ where $\mathbf{n}_0^{\theta}$ is a unit vector representing the fiber direction). The deformation of solid phase also can be represented by a set of invariants I_m , where $I_m = f(\mathbf{C})$ (Holzapfel, 2000). In order to consider the damage of AF, another set of independent variables, d^i , are introduced to characterizes the damage of AF due to the damage of component i . Thus, the energy function W can be written as,

$$W = W(I_m, \Phi^{\alpha}, \mathbf{v}^{\beta s}, d^i). \quad (7)$$

By using the chain rule on the time differentiation of W , equation (6) can be rewritten as,

$$\begin{aligned}
& (J\sigma^e - 2\mathbf{F} \sum_m \frac{\partial W}{\partial I_m} \frac{\partial I_m}{\partial \mathbf{C}} \mathbf{F}^T) : \nabla \mathbf{v}^s \\
& + \sum_{\beta} \left\{ -\frac{\partial W}{\partial \mathbf{v}^{\beta s}} \frac{D^s \mathbf{v}^{\beta s}}{Dt} + J \left[\sigma^{\beta} + \phi^{\beta} (\tilde{\mu}^{\beta} - A^{\beta}) \mathbf{I} \right] : \nabla (\mathbf{v}^{\beta} - \mathbf{v}^s) \right\} \\
& + J \sum_{\beta} \left\{ (\mathbf{v}^{\beta} - \mathbf{v}^s) \cdot \left[-\nabla (\phi^{\beta} A^{\beta}) + \tilde{\mu}^{\beta} \nabla \phi^{\beta} - \boldsymbol{\pi}^{\beta} \right] \right\} \\
& - \sum_i \frac{\partial W}{\partial d^i} \frac{D^s d^i}{Dt} \geq 0
\end{aligned} \quad (8)$$

where $\tilde{\mu}^{\beta} = \frac{\partial W}{\partial \Phi^{\beta}} + p + \frac{\lambda z^{\beta}}{\tilde{V}^{\beta}}$ is the electrochemical potential of phase β . The above inequality must be true for any values of the independent variables, which leads to,

$$\sigma^e = \frac{2}{J} \mathbf{F} \sum_m \frac{\partial W}{\partial I_m} \frac{\partial I_m}{\partial \mathbf{C}} \mathbf{F}^T, \quad (9)$$

$$\frac{\partial W}{\partial \mathbf{v}^{\beta s}} = 0, \quad (10)$$

$$\sigma^{\beta} = \phi^{\beta} (A^{\beta} - \tilde{\mu}^{\beta}) \mathbf{I}, \quad (11)$$

$$J \sum_{\beta} \left\{ (\mathbf{v}^{\beta} - \mathbf{v}^s) \cdot \left[-\nabla (\phi^{\beta} A^{\beta}) + \tilde{\mu}^{\beta} \nabla \phi^{\beta} - \boldsymbol{\pi}^{\beta} \right] \right\} - \sum_i \frac{\partial W}{\partial d^i} \frac{D^s d^i}{Dt} \geq 0. \quad (12a)$$

Based on Equations (2), (11), Equation (12a) can be simplified as:

$$J \sum_{\beta} [(\mathbf{v}^{\beta} - \mathbf{v}^s) \cdot (-\phi^{\beta} \nabla \tilde{\mu}^{\beta})] - \sum_i \frac{\partial W}{\partial d^i} \frac{D^s d^i}{Dt} \geq 0 \quad (12b)$$

Equation (12b) shows that the energy dissipation caused by the transport of fluid and ion phases is, in general, related to the damage of the mixture. The coupling between the kinetics of transport and damage could be represented by a constitutive model based on experimental studies. However, to our best knowledge, there are no such experimental results available for soft tissues. In the following analysis, we assume that the energy dissipations associated with transport and damage are decoupled with each other. This assumption dismisses the explicit coupling between the kinetics of transport and damage, but does not dismiss the implicit coupling. This is because the damage would affect the

deformation which could affect the transport properties. Thus, based on this assumption, Equation (12b) can be reduced to:

$$-\sum_{\beta}(\mathbf{v}^{\beta}-\mathbf{v}^s) \cdot \phi^{\beta} \nabla \tilde{\mu}^{\beta} \geq 0, \quad (13)$$

$$-\sum_i \frac{\partial W}{\partial d^i} \frac{D^s d^i}{Dt} \geq 0. \quad (14)$$

2.2 Continuum damage model

As shown above, the strain energy function does not depend on the relatively velocity, but depends on the other independent variables including solid phase deformation, volume fractions of each phase, and damage variables. In this study, the AF is modeled as an anisotropic material with two families of fiber bundles embedded in an isotropic ground matrix. Thus, the strain energy function for an intact AF (W_{AF}) can be written as (Ehlers and Eipper, 1999; Holzapfel et al., 2000),

$$W_{AF}=W_m(\mathbf{C}, \phi_0^w)+\sum_{\theta=1,2} W_f^{\theta}(\mathbf{C}, \mathbf{n}_0^{\theta}), \quad (15)$$

with

$$W_m=\frac{\mu}{2}(I_1-3)-\mu \ln J+\lambda(\phi_0^w)^2\left[\frac{J-1}{\phi_0^w}-\ln\left(\frac{J-1}{\phi_0^w}+1\right)\right], \quad (16)$$

and

$$W_f^{\theta}=\frac{k_1}{2k_2}\{H(I_f^{\theta}-1)\exp[k_2(I_f^{\theta}-1)^2]-1\} \quad (17)$$

where W_m is the strain energy density function of the intact ground matrix, W_f^{θ} is the strain energy density function of intact fibers, respectively, with $\theta=1,2$ representing each family of fibers, ϕ_0^w is the water volume fraction at the reference state, μ and λ are the two Lamé constants, I_1 is the first invariant of $\mathbf{C}$, k_1 and k_2 are the material parameters, $H(\bullet)$ is the Heaviside step function, and $I_f^{\theta}=\mathbf{C}:(\mathbf{n}_0^{\theta} \otimes \mathbf{n}_0^{\theta})$ is the square of the stretch ratio of fiber in the direction of $\mathbf{n}_0^{\theta}$ ($\pm 30^\circ$ with respect to the horizontal plane).

Three damage variables, d^m and d^θ ($\theta = 1, 2$), are introduced to the strain energy density function to model the damage of ground matrix and two families of fiber bundles, respectively, and they are in the range from 0 (no damage) to 1 (complete damage). The strain energy density function for the damaged AF may be expressed as (Balzani et al., 2006; Famaey et al., 2013),

$$W_{AF} = (1 - d^m)W_m + \sum_{\theta=1,2} (1 - d^\theta)W_f^\theta. \quad (18)$$

In this study, we neglect the damage of ground matrix (i.e., $d^m = 0$) because: (1) the contribution of fiber bundles to the tensile modulus of AF is much larger than that of the ground matrix, and (2) the yield strain of fiber bundles under tension is much smaller than that of the ground matrix (Ebara et al., 1996; Fujita et al., 1997). A possible evolution of the damage variable for fiber bundles is given by (Balzani et al., 2006; Famaey et al., 2013),

$$d^\theta = d_{\max} \left[1 - \exp \left(-\frac{\beta^\theta}{\gamma} \right) \right], \quad (19)$$

where $d_{\max} \in [0, 1]$ and γ are two damage parameters, and β^θ is the internal damage variable which is a function of W_f^θ . In this study, we assumed that the damage is irreversible and unhealable. Thus, the parameter β^θ is defined as (Balzani et al., 2006; Famaey et al., 2013),

$$\beta^\theta = \max_{0 \leq \tau \leq t} \langle W_f^\theta(\tau) - \hat{W}_f \rangle, \quad (20)$$

where $\hat{W}_f$ is the threshold value for the strain energy density above which damage will occur. The bracket, $\langle \bullet \rangle$, defined as $\langle \bullet \rangle = 0.5[(\bullet) + |\bullet|]$, is used to filter out negative values. It is easy to check that the choice of strain energy function and the damage variables [see equations. (18) – (20)] meets the thermodynamic constraint [equation (14)].

2.3 Setup of numerical simulations

In the following section, numerical examples are given to show the applicability of the anisotropic multiphysics damage model described above to simulate the damage of AF. The numerical simulations are performed by incorporating the damage model into a previously developed and validated anisotropic multiphysics framework which could characterize the couplings of multiple physical fields in an intact IVD (Gao et al., 2016).

In this study, the value of $\hat{W}_f$ in equation (20) is assumed to be 100 kJ/m^3 , and independent of the anatomic region of the non-degenerated AF (Ebara et al., 1996). There are few experimental results on the values of $d_{\max}$ and γ in equation (19) available for AF. These

two parameters govern the stress-stretch curve after the damage initiated. In this study, the values of $d_{\max}$ and γ are approximately chosen to be 0.5 and 50 kJ/m³, respectively, which is based on a displacement-load curve obtained with the tensile experiment on animal IVDs (Pezowicz, 2010).

In this study, an overall damage variable, D , is defined to evaluate the damage of both families of fiber bundles by,

$$D = \frac{1}{2} \sum_{\theta=1,2} d^{\theta}. \quad (21)$$

The damage region is defined as wherever $D \geq 0.1$ (the value of 0.1 rather than 0 is chosen to avoid possible numerical errors) in this study. A volume fraction of damage region is defined to evaluate the extent of the damage in the AF, which is defined as the volume ratio of the damage region to the whole AF region.

A quarter of the 3D vertebra-disc-vertebra segment (L4–L5) is used for numerical simulation on the assumption that the segment is approximately symmetric along the middle sagittal and middle axial planes, see Fig. 1. The mechanical, transport, and electrochemical properties of AF, NP, and CEP could be found in (Gao et al., 2016). The values of (electro)chemical potentials for fluid and solutes in the blood serum are assigned at the interfaces between the CEP and the vertebra and at the periphery of AF. The vertebrae are included in order to apply the mechanical loading boundary conditions, and they are modeled as rigid bodies. The initiation and propagation of damage under compression is simulated in this study. The disc is vertically compressed at a rate of 1000 N/s to 2000 N.

3. Results

The damage initiates under about 700 N compression, then the volume fraction of damage region increases nonlinearly with the linear increase of the compressive load, which reaches to about 6% when the compressive load increases to 2000 N, see Fig. 2. The effects of $\pm 10\%$ change in damage parameters ($\hat{W}_f$, $d_{\max}$, and γ) on the simulated volume fraction of damage region were investigated. It was found that the damage region was more sensitive to the value of $\hat{W}_f$ than those of $d_{\max}$ and γ , see Fig. 2.

The spatial distributions of overall damage at different levels of compressive load are shown in Fig. 3a–3c. The damages occur in the outer posterior AF, middle posterior AF (adjacent to the vertebra), and the junction of posterior AF and CEP at 700 N compressive load (Fig. 3a). Additional damages occur in outer anterior and outer lateral AF when the compressive load increases to about 1000 N (Fig. 3b), and these damage regions expand with increasing the compressive load (Fig. 3c).

The spatial distributions of stretch ratio at different levels of compressive load for one family of fiber bundles are shown in Fig. 3d–3f, and those for another family of fiber bundles are shown in Fig. 3g–3i. It is found that: (1) the damage in the outer AF occurs when the fiber

stretch ratio reaches about 1.10 (Fig. 3a and 3d); (2) the damage in the middle-to-outer AF occurs when the fiber stretch ratio reaches about 1.12 (Fig. 3b, 3e, and 3h); (3) the damage in the inner AF occurs when the fiber stretch ratio reaches about 1.17 (Fig. 3a and 3g, Fig. 3c and 3f).

4. Discussion

An anisotropic multiphysics damage model is developed based on the continuum mixture theory and the classic continuum damage model. In this model, the damage is characterized by a damage variable which is a non-observable variable. A recent study showed that the damage can be described by observable variables in a reactive damage framework, whereby the damage is characterized by the amount of bonds in the solid phase that have broken (Nims et al., 2016). It has been also showed that the classic continuum damage model can be reproduced from the reactive damage framework as the non-observable damage variable in classic continuum damage model representing the mass fraction of broken bonds in the reactive damage framework (Nims et al., 2016). Therefore, the anisotropic multiphase continuum damage model developed in the present study could be interpreted as a macroscopic model for tissues with the solid phase consisting of undamaged and damaged constituents, and the evolution of the damage variable represents the mass exchange between undamaged and damaged constituents within the solid phase (Nims et al., 2016).

In the present study, uniform yield strain energy density is chosen as the failure criterion measure in simulating the damage of AF based on experimental findings, whereas, in previous studies (Natarajan et al., 1994; Shirazi-Adl, 1989), uniform yield stress or strain was chosen as the failure criterion measure. However, experimental studies have shown that the yield stress and strain depend on the anatomic region of AF (Ebara et al., 1996; Skaggs et al., 1994). Our simulation results show that the yield stretch ratio ranges from 1.10 to 1.17 from the outer AF to inner AF (Fig. 3) which is consistent with the experimental results from uniaxial tensile tests (Ebara et al., 1996; Skaggs et al., 1994). This consistency indicates the value of $\bar{W}_f$ is reasonably chosen.

The values of $d_{\max}$ and γ are approximately chosen based on the experimental data (Pezowicz, 2010). They are not necessary to be constants but can evolve along with the growth of damage (Balzani et al., 2012). When the value of $d_{\max}$ evolves to 1, the condition corresponds to the completely rupture of AF. In this study we focus on simulating the initiation of damage in AF rather than the rupture of AF, thus it is reasonable to set the value of $d_{\max}$ as 0.5.

The numerical results are obtained with an isolated disc, thus the relationship between damage volume fraction and compressive load does not represent those under in vivo condition. This is because: (1) only an isolated disc rather than a full motion segment is used in this study so that the confined effect provided by the longitudinal ligaments and the load-sharing effect provided by the facet joint are not accounted; and (2) this study only considered the damage of fiber bundles in the AF, whereas the damage of ground matrix and the delamination of lamellae have not yet been included in the current model due to the lack of failure criterion and experimental data.

Previous experimental studies have shown that pure axial compression on a motion segment is more likely to cause the failure in CEPs/vertebrae, as well as in the AF (Brown et al., 1957; Lin et al., 1978; McNally et al., 1993; Roaf, 1960; Yoganandan et al., 1988). These in vitro experimental studies focused on the catastrophic damage of the motion segment, and the failure loads were over 4000 N. In our current study, only the initiation of damage in the AF of an isolated disc is studied. The maximum compressive load simulated in this study is 2000 N, which is much smaller than the failure load of CEPs and vertebrae.

In summary, we present an anisotropic multiphysics damage model for modeling the initiation and propagation of damage in the AF based on thermodynamic principles. This anisotropic multiphysics damage model integrates the continuum mixture theory and a continuum damage model. The damage criterion (based on strain energy density) used in the current study could capture the anatomic region dependent damage behaviors of AF. The simulation result is more sensitive to the value of $\hat{W}_f$ than the values of other damage parameters. More experimental studies are needed to evaluate the damage variables of AF.

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Fig. 1.
Schematic of a quarter of vertebra-disc-vertebra segment used in the numerical simulation.
Length scale is in mm.

Fig. 2.

Volume fraction of damage region in AF under compression with a constant loading rate of 1000 N/s. The solid black curve represents the simulated results using the chosen damage parameters, whereas other solid and dash curves represent the simulated results with varying the damage parameters by $\pm 10\%$.

Fig. 3.

Damage distribution [represented by damage variable defined in equation (21)] in AF compressed at a rate of 1000 N/s to: (a) 700 N, (b) 1000 N, and (c) 2000 N; the spatial distribution of fiber stretch ratio for one family of fiber bundles at: (d) 700 N, (e) 1000 N, and (f) 2000 N; the spatial distribution of stretch ratio for the other family of fiber bundles at: (g) 700 N, (h) 1000 N, and (i) 2000 N.