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Effects of flow-dependent and flow-independent viscoelastic mechanisms on the stress relaxation of articular cartilage

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Abstract

Articular cartilage is a bearing material that lines the ends of the bones of synovial joints. The solid phase of articular cartilage is chiefly composed of complex macromolecules including collagen and proteoglycans. The fluid phase is presented by interstitial fluid filling in the solid phase's pores. The rheological behavior of articular cartilage depends upon the intrinsic interaction between the solid matrix's deformation and the interstitial fluid's motion. Thus, the viscoelastic properties of articular cartilage arise from (1) the diffusional drag of relative velocity between the interstitial fluid and the solid matrix, or flow-dependent mechanism, and (2) the intrinsic viscoelastic properties of the solid matrix, or flow-independent mechanism. This study aimed to assess both mechanisms' contribution to the stress relaxation of articular cartilage.

The mathematical model of confined compression of articular cartilage was developed using the linear biphasic theory of Mow et al. [1]. The stress-time curves were computed using the quasi-linear viscoelastic model of Fung. The assessment procedure was considered based on the experimental data of Soltz and Ateshian [2].

Our findings envisage that the linear biphasic theory of Mow et al. failed in predicting stress relaxation, that is, the flow-dependent viscoelastic mechanism is not able solely (coincidence 41.4 % of the theoretical and experimental data) to cover the stress relaxation mechanism after stepwise loading. The interrelation between the intrinsic viscoelasticity and the permeability of the solid matrix is discussed.

Keywords: Articular cartilage, stress relaxation, viscoelastic mechanisms

1. Introduction

Articular cartilage is composed mainly of an extracellular (solid) matrix and interstitial fluid. The extracellular matrix includes mostly type II collagen fibers, proteoglycan monomers and aggregates, and non-collagenous proteins. The interstitial fluid is composed mainly of water with associated mobile electrolytes. It has been shown that articular cartilage exhibits characteristic viscoelastic creep or stress relaxation responses under compressive stress or compressive strain, respectively [1,3,4]. Interactions between the articular cartilage's solid and fluid phases are important mechanisms that contribute to the observed viscoelastic behavior. The solid phase of articular cartilage is composed of structurally complex macromolecules including collagen and proteoglycans. The intertwist, friction and electrostatic interactions between those components give rise to the intrinsic or flow-independent viscoelastic response. In addition, the flow of the interstitial fluid phase through the porous and permeable solid matrix gives rise to flow-dependent viscoelasticity [1,5].

The complex mechanical behavior of articular cartilage has been simplified into the multiphase mechanics of two interacting constituents: an incompressible, elastic solid phase and an incompressible fluid phase. Mow et al. [1] proposed this biphasic poroelastic (BPE) model of articular cartilage, in which the viscoelasticity of the tissue was assumed to be solely due to the fluid flow-dependent frictional drag interactions created by a flow of interstitial fluid through a porous solid matrix. The linear BPE model assumes linear elasticity of the solid matrix, constant permeability, and infinitesimal deformation of the tissue, and results in three material parameters necessary to describe the mechanical response of the tissue: two elastic Lame constants associated with the porous solid matrix and permeability. The linear

BPE model has been used to investigate the viscoelasticity exhibited by articular cartilage under various mechanical loading conditions [6]. The biphasic theory was extended by Mak [7] to the biphasic poroviscoelastic theory (BPVE), by including the intrinsic viscoelasticity of the solid matrix in the formulation. Therefore, this model is capable of describing both the flow-dependent and flow-independent mechanisms of viscoelasticity in articular cartilage. Using assumptions of linear viscoelasticity, constant permeability, and infinitesimal deformation of the tissue, the linear BPVE model has been reported to accurately account for the complete creep response of articular cartilage under confined and unconfined compression [8,9].

Some studies proposed that the intrinsic viscoelasticity of the solid matrix was a dominant mechanism needed to describe its response to unconfined compression [10,11]. However, other theoretical and experimental investigations suggested that flow-dependent viscoelasticity was the dominant dissipative mechanism in cartilage [9,12,13,14]. Because of this discrepancy, the objective of this study was to evaluate the contributions of the fluid flow-dependent and fluid flow-independent viscoelastic mechanisms to the mechanical response of articular cartilage during stress relaxation in confined compression. Our approach was based on the theoretical model and experimental results of Soltz and Ateshian [2]. As that model uses BPE theory, which envisages a flow-dependent mechanism only, we enlarged it to Fung's quasi-linear viscoelastic (QLVE) theory [15].

2. Methods

2.1. Constitutive modeling of articular cartilage

In the confined compression experiment, the cartilage sample is compressed inside a cylindrical chamber with an impermeable side wall and, therefore, the matrix displacement and the fluid flow are strictly one-dimensional along the axes *x* [2]. The total stress tensor σ_x inside the tissue is the sum of the interstitial fluid pressure *p* and the elastic stress σ_x^s resulting from the deformation of the solid matrix

(1) $$\sigma_x = -p + \sigma_x^s \ ,$$

(2) $$\sigma_x^s = (\lambda + 2\mu) e_x = H_A \, e_x \ ,$$

where λ and μ are the Lame constants, H_A is aggregation modulus and $e_x = du/dx$.

The momentum (equilibrium) equation for the mixture, under quasi-static conditions and in the absence of body forces, is given by

(3) $$\frac{\partial \sigma_x}{\partial x} = -\frac{\partial p}{\partial x} + \frac{\partial \sigma_x^s}{\partial x} \ .$$

The mass conservation equation for the mixture is given by

(4) $$\frac{\partial}{\partial x}\left(v^s + \omega\right) = 0 \ ,$$

where $\omega = \varphi^f \left(v^f - v^s\right)$ is the flux of fluid relative to the solid, φ^f is the fluid volume fraction (tissue porosity), and v^s, v^f are the solid and fluid phase velocities, respectively.

Following the original BPE theory [1], the relation between fluid flux and pressure gradient is taken to be linear, according to Darcy's law:

(5) $$\omega = -k \frac{\partial p}{\partial x} \ ,$$

where *k* is the hydraulic permeability.

Combining the equations (1) to (5), and having in mind that $v^s = \frac{\partial u}{\partial t} + u\frac{\partial u}{\partial x}$ we obtain the following differential equation for the axial displacement u:

$$\frac{1}{kH_A}\frac{\partial u}{\partial t} = \frac{\partial^2 u}{\partial x^2} \quad . \tag{6}$$

2.2. Fung's quasi-linear model of viscoelasticity

In the linear biphasic theory for infinitesimal strain [1], σ_x^s is taken to be the generalized Hooke's law. The BPE theory was extended by Mak [7] to the biphasic poroviscoelastic theory (BPVE), by including the intrinsic viscoelasticity of the solid matrix as a viscoelastic law based on the Boltzmann superposition principle. In the present analysis, we adopt the QLVE formulation proposed by Fung [15]

$$\sigma^{ve}(t) = \sigma^e(e(t)) + \int_{t_0}^{t} \sigma^e(\tau)\frac{\partial G}{\partial \tau}d\tau \,, \tag{7}$$

where σ^e is the elastic response, and $G(t)$ is the reduced relaxation function. Following the Boltzmann superposition principle, it was customary to analyze the relaxation function into the sum of exponential functions and identify each exponent with the rate constant of a relaxation mechanism. Instead of that, Fung [15] supposed a continuous relaxation spectrum of time from $10^{-2}\,[s]$ to $10^{+2}\,[s]$ and defined the relaxation function as

$$G(t) = \left\{1 + C\left[E_1\left(\frac{t}{\tau_2}\right) - E_1\left(\frac{t}{\tau_1}\right)\right]\right\} \Big/ \left[1 + C\ln\left(\frac{\tau_2}{\tau_1}\right)\right], \tag{8}$$

where $E_1\left(\frac{t}{\tau}\right)$ is the exponential integral function, and C, τ_1, and τ_2 are constants. The parameter C is a constant proportional to the amplitude of the continuous relaxation spectrum. The time constants define the width of the spectrum, and govern the fast and slow relaxation phenomena, respectively. The intrinsic viscoelastic effect is diminished so that for $C = 0$ the relaxation function $G(t) = 1$. From equation (7) we see that the stress $\sigma^{ve}(t)$ for the solid matrix will then reduce to that of a linearly elastic solid. Alternatively, if the width of the spectrum reduces to zero, i.e. $\tau_1 = \tau_2$, this linear elastic relation is also recovered. Hence, for the limiting cases $C = 0$ or $\tau_1 = \tau_2$, the BPVE theory will reduce to the linear biphasic theory [1].

For $t \to \infty$, $E_1(t/\tau_1) \to 0$ and $E_1(t/\tau_2) \to 0$, and

$$G(\infty) = 1/[1 + C\ln(\tau_2/\tau_1)] \,. \tag{9}$$

The exponential integral function E_1 is tabulated and approximated in [16]. Here we used the following approximation for the reduced relaxation function [15, p. 286]

(10) $$G(t) = \frac{1 + C\gamma - C\ln\left(\frac{t}{\tau_1}\right)}{1 + C\ln\left(\frac{\tau_2}{\tau_1}\right)} \ ,$$

where γ is the Euler-Mascherony constant.

3. Results

In the confined compression relaxation experiment, circular samples are compressed by a suddenly applied constant (isometric) extension. Solid matrix deformation and interstitial fluid flow in the radial direction are prevented by lateral confining walls and, thus, are neglected in the analysis. Under these idealized conditions, the displacement and fluid movement occur only in the axial direction. The governing equations (1) to (5) express them mathematically, which results in a partial differential equation (6). The initial condition for this equation is given by

(11) $$u = (x, t = 0) = 0 \ ,$$

and the boundary conditions are

(12) $$\begin{aligned} &u(x = 0, t) = 0, \\ &u(x = h, t) = -u_a \ , \\ &u_a = \begin{Bmatrix} V_0 t & t \le t_0 \\ V_0 t & t > t_0 \end{Bmatrix}. \end{aligned}$$

In this investigation, we used the experimental results of Soltz and Ateshian [2] to specify the boundary conditions. Shortly, a bovine specimen, thickness $h = 1\,[mm]$ and diameter $4.78\,[mm]$, was compressed in a latex tube at constant velocity $V_0 = 0.25\left[\mu m/s\right]$ from $t = 0$ to $t = t_0 = 300\,[s]$. At $t > t_0$, the displacement was maintained constant and the stress relaxation was recorded. The hydraulic permeability, k, was $k = 5.94 * 10^{-15}\left[m^4/N.s\right]$ and the aggregation modulus was $H_A = 0.64\,[MPa]$.

Once the solution of Eq. (6) has been obtained, the fluid pressure is given by

(13) $$P(x,t) = H_A\left\{\frac{\partial u}{\partial x}(x = 0) - \frac{\partial u}{\partial x}(x = h)\right\}$$

and the theoretical solid matrix stress is

(14) $$\sigma^t(x,t) = H_A\, e(x,t) \qquad \text{where} \qquad e = u/h \ .$$

Comparison between the experimental and the theoretical results was assessed through the following estimation measure

(15) $$Est = \frac{1}{n}\sum_{i=1}^{n}\left(\frac{\sigma^t(i)}{\sigma^e(i)} - 1\right) \ .$$

Fig. 1 represents the theoretical stress through the depth of the articular cartilage, namely $x = h, x = 0.8h$ and $x = 0.6h$. It is evident that large magnitudes of stress occur near the articular surface ($x = h$) just after the stop of loading, while at depths greater than 20% the stress variation remains negligible.

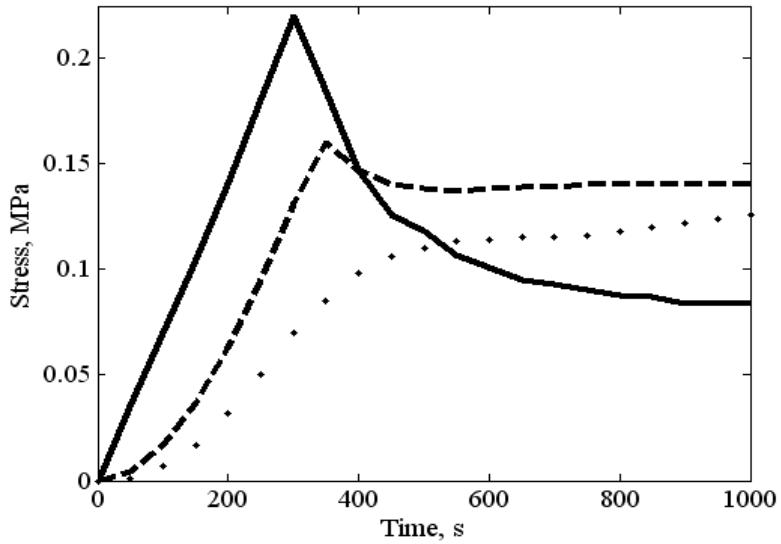

Fig. 1. Theoretical stress at $x = h$ (continuous line), $x = 0.8h$ (dashed line), and $x = 0.6h$ (dotted line).

Fig. 2 shows the interstitial pressure (continuous line) and solid matrix stress as functions of time during compression of the articular cartilage. Two peculiarities must be outlined, first, both follow an increasing trend during the sudden loading and decreasing trend during stress relaxation, and, second, the interstitial pressure is much higher than the solid matrix stress. The same trend of stress during the stress relaxation experiment was reported in [2], while for creep experiments, stresses are increasing during the sudden loading of the sample $(t < t_0)$ and after $(t > t_0)$, the solid matrix stress continuously increases but the fluid pressure falls [9].

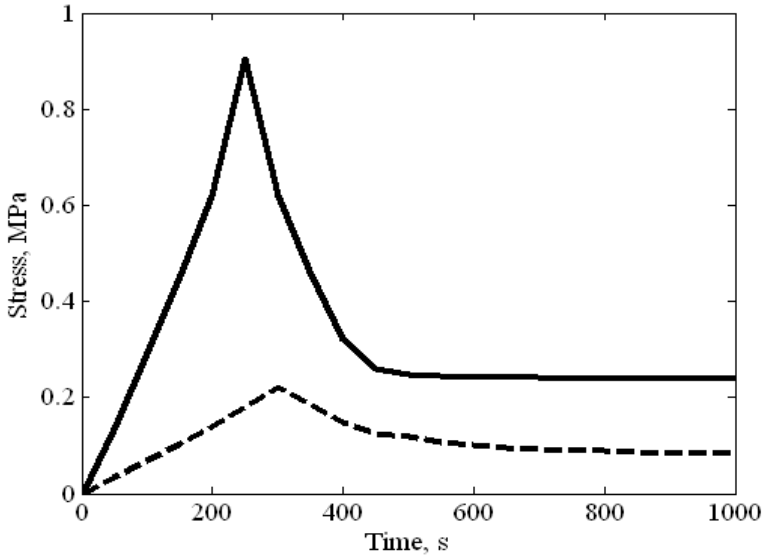

Fig. 2. Theoretical interstitial pressure (solid line) and solid matrix pressure (dashed line) for stress relaxation of articular cartilage.

A comparison between the experimental confined compression stress relaxation response from Soltz and Ateshian [2] (solid line) and the corresponding theoretical curve (dashed line) according to Eq. (13) is shown in Fig. 3. It is evident that a great discrepancy exists between them and, consequently, the estimation measure equals 41.4 %. Therefore, this result merely shows that the BPE theory, or the flow-dependent mechanism, is not able solely to reproduce the experimental stress relaxation.

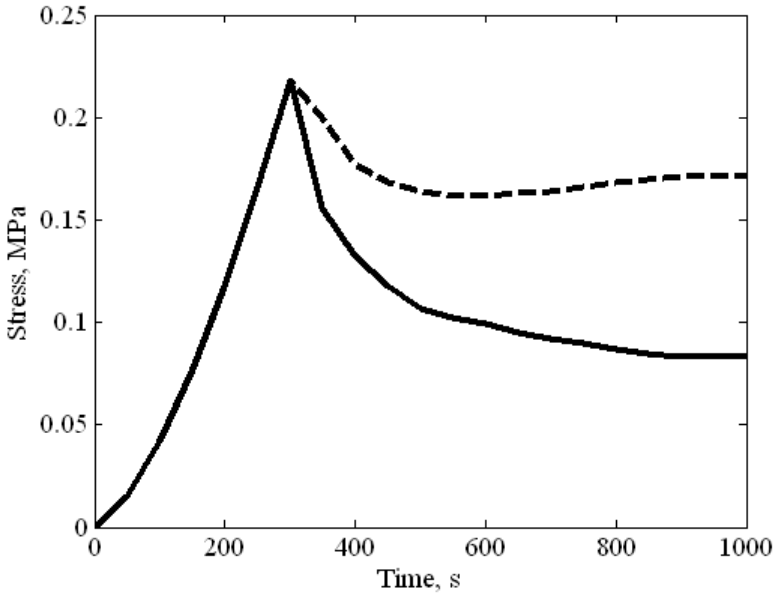

Fig. 3. Experimental confined compression stress relaxation response from Soltz and Ateshian [2] (solid line) and corresponding theoretical curve (dashed line) according to Eq. (13).

Having in mind the physical meaning of the viscoelastic parameters in formula (10), we determined their values based on experimental stress relaxation data in [2] as follows. The parameter C was proportional to the amplitude of the continuous relaxation spectrum, that is $C \approx G(t_0) - G(\infty) = 0.62$. The fast relaxation parameter $\tau_1 = 1.78\,[s]$ was calculated through the boundary condition $G(t_0) = 1$, and the slow relaxation parameter $\tau_2 = 1849\,[s]$ – using Eq. (9). Finally, Eq. (10) reduces to

(16) $$G(t) = 1 - 0.097\ln(t)$$

and remembering that $\sigma^e(t > t_0)$ is a constant in the isometric experiment, Eq. (7) reduces to

(17) $$\sigma^{ve}(t) = \sigma^e(t_0)[1 - 0.097\ln(t)] \ .$$

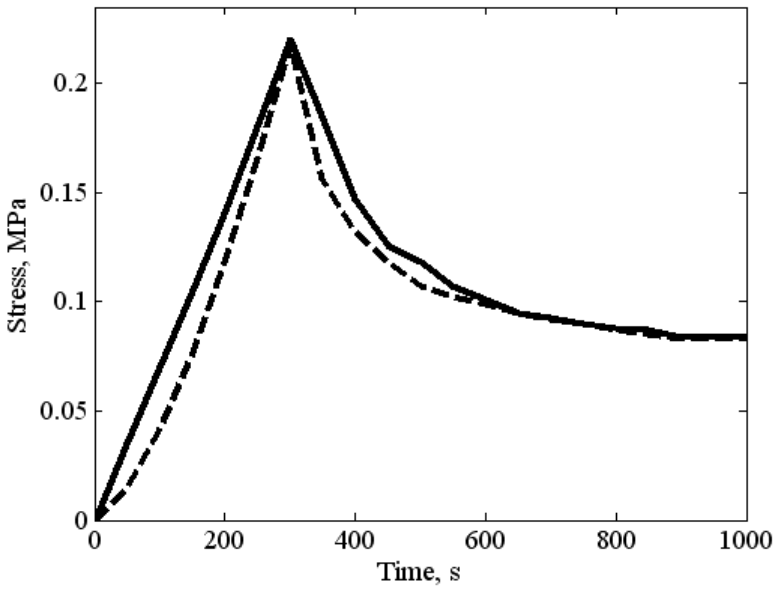

Fig. 4. Experimental confined compression stress relaxation response from Soltz and Ateshian [2] (solid line) and corresponding theoretical curve (dashed line) for flow-independent stress relaxation according to Eq. (17).

Flow-independent stress relaxation according to Eq. (17) is shown in Fig. 4. Good agreement (5.7 %) between the theoretical and experimental results demonstrates that the viscoelastic model adopted in this study successfully predicted the stress relaxation of articular cartilage.

4. Discussion and conclusion

The objective of this study was to evaluate the contributions of the fluid flow-dependent and fluid flow-independent viscoelastic mechanisms to the mechanical response of articular cartilage during stress relaxation in confined compression. Our approach was based on the BPE theory [1] including the QLVE theory [15] and the experimental results of Soltz and Ateshian [2]. The results shown in Fig. 3 demonstrate that the BPE model failed in predicting stress relaxation, that is, the flow-dependent viscoelastic mechanism is not able solely (coincidence 41.4 % of the theoretical and experimental data) to cover the stress relaxation mechanism. On the other hand, the BPVE model was very successful at predicting stress relaxation with a 5.7 % difference between the theoretical and experimental data.

The problem of the role of fluid flow-independent and fluid flow-independent viscoelastic mechanisms is debatable in the literature. Our results support the hypothesis that the intrinsic viscoelasticity of the solid matrix is a dominant mechanism needed to describe its response to unconfined compression [10,11] while other theoretical and experimental investigations suggested that flow-dependent viscoelasticity is the dominant dissipative mechanism in cartilage [9,12,13,14]. Furthermore, others emphasized that fluid-flow dependent viscoelasticity dominates the compressive response of cartilage, whereas intrinsic solid matrix viscoelasticity dominates the tensile response [13] or concluded that the short-term viscoelastic behavior of articular cartilage, when subjected to a fast ramp strain rate, is primarily governed by a fluid flow-independent viscoelastic mechanism, whereas the long-term viscoelastic behavior is governed by a fluid flow-dependent viscoelastic mechanism [11].

Finally, it seems reasonable to support the suggested idea of a combination of the fluid flow-dependent and fluid flow-independent viscoelastic mechanisms in accounting for the true mechanical behavior of articular cartilage under compression [10,17] with the following additional arguments. First, the majority of authors accept that the permeability is a constant during compression while it was empirically demonstrated that there is an exponential decrease of the permeability function with the deformation of the solid matrix [1]. Second, Fung's QLVE assumes only time dependence of the reduced relation function but in the more general case of nonlinear viscoelasticity, the material constants may vary with strain [18].

Acknowledgments

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