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A phenomenological model of damage in articular cartilage under impact loading

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ABSTRACT

Damage progression in high-strain rate and impact tests on articular cartilage is considered. A new type of kinetic damage evolution law is proposed and used to draw implications about the accumulated damage and the coefficient of restitution. Based on the developed damage model, a new fracture criterion is introduced.

1. Introduction

As a living tissue, articular cartilage can be characterized in terms of viability, e.g., using the Mankin score [18]. It is long known that some mechanical properties of articular cartilage reflect the degree of viability [22,31]. For instance, early osteoarthritic (OA) degeneration of cartilage manifests itself in the decrease of indentation stiffness [7,15]. On the other hand, in impact experiments, an overloading of articular cartilage sample is accompanied by the damage accumulation and can result in cell deaths. Thus, the accumulation of damage in cartilage tissue and the decrease of its viability can be viewed as two sides of the same state evolution process.

It should be noted that there is a difference between cell viability (i.e., how many cells are alive) and mechanical 'viability' (or ability to support load), which can be regarded as the structural integrity of tissue. It is also to emohasize that these concepts are not the same although they may be related and only the latter is assessed qualitatively in the Mankin score, which is a histopathological classification of the severity of osteoarthritic lesions of cartilage [29].

We consider the damage accumulation in blunt impact and high-rate compression experiments, which can be modeled as a uniform one-dimensional deformation process. For the sake of simplicity, we neglect the effect of energy dissipation associated with the interstitial fluid flow, as it was shown by [11], in the short-time deformation process, articular cartilage may be regarded as an elastic material. At the same time, in view of the recent experimental evidence [9], we discuss the extension of the impact model with damage to account for the viscous dissipation effect.

We employ a phenomenological modeling approach [17,26] and introduce a scalar damage variable, D, which is assumed to be dimensionless. Following [19], one can use the hypothesis of strain equivalence and consider first the elastic constitutive law $\sigma = E(1-D)\varepsilon$, where ε is the strain associated with a damaged state under the applied stress σ, and E is Young's modulus of the virgin (undamaged) material. The question of damage modeling in biological tissues was considered in a number of recent studies [20,24,27]. In particular, [10] used the Kachanov–Rabotnov kinetic equation

$$\dot{D} = \frac{C\sigma^m}{(1-D)^k}, \quad D(0) = 0, \tag{1}$$

where C, m, and k are constants, and the dot denotes the differentiation with respect to time t. However, it can be shown that the above equation does not reflect some important features of the damage accumulation observed in experiments on articular cartilage. In particular, [13] documented greater matrix damage in cartilage samples subjected to a high rate of loading (~930 MPa/s), compared to samples exposed to a low rate of loading (40 MPa/s).

In the present paper, based on the experimental facts, we propose a new type of damage kinetic equation and apply it to draw

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implications, which are then checked against other experimentally observed phenomena.

2. Kinetic equation for the damage evolution

First, we consider the ramp compression experiment [25] characterized by a constant strain rate, $\dot{\varepsilon}_0$, between 3×10^{-5} and 0.7 s^{-1} to a peak stress between 3.5 and 14 MPa. Observe that for articular cartilage samples compressed at $\dot{\varepsilon}_0 = 3\times10^{-5}$ s^{-1}, the peak compressive stresses 3.5 MPa and 14 MPa corresponded to peak compressive strains $\varepsilon_0 = 83\pm5\,\%$ (mean $\pm$ standard deviation) and $\varepsilon_0 = 95\pm7\,\%$, respectively. It should be noted the deformation of underlying bone was neglected in the sample strain evaluation. In what follows, we make use of the following result [25]: mechanically induced damage to the cartilage extracellular matrix depended upon applied strain rate and peak stress, with cracking probability being an increasing function of both parameters.

Since for large deformations like those mentioned above, the linear elastic constitutive law does not apply, we reformulate the damage kinetic equation (1) in terms of strain and generalize it as follows (see also [10,33]):

$$\dot{D} = \frac{C\varepsilon^m|\dot{\varepsilon}|^n}{(1-D)^k}. \tag{2}$$

Now, integrating Eq. (2) for the strain evaluation $\varepsilon = \dot{\varepsilon}_0 t$, $t\in(0, t_0)$, where $t_0 = \varepsilon_0/\dot{\varepsilon}_0$ is the time necessary to achieve a peak strain ε_0, it can be easily shown that the accumulated damage $D_0 = D(t_0)$, provided that $D(0) = 0$, is governed by the product $\varepsilon_0^{m+1}\dot{\varepsilon}_0^{n-1}$, and for $n>1$, it increases with increasing the strain rate.

Observe that in impact tests, where the strain evolution is similar to the half-sine law variation $\varepsilon = \varepsilon_0\sin\omega t$, $t\in(0, \pi/\omega)$, the right-hand side of Eq. (2) vanishes at the state of peak compression, where the stress is close to its peak value, that seems to be in contradiction with the experimental observation [25] that at high strain rates, the matrix damage appeared to be primarily a function of peak stress. However, it can be checked that according to Eq. (2), the accumulated damage $D_0 = D(\pi/\omega)$ is again governed by the product $\varepsilon_0^{m+1}\dot{\varepsilon}_0^{n-1}$ provided $\omega = \dot{\varepsilon}_0/\varepsilon_0$.

Second, as it was shown by [23] in their ramp compression tests, the so-called "gel diffusion" rate of deformation $\dot{\varepsilon}_g = 1/\tau_g$ appears to represent a threshold for the transition between low and high strain rate "modes" of matrix and cell injury. Recall that the gel diffusion time constant τ_g is introduced for articular cartilage sample treated as a biphasic poroelastic material [6] and is given by $\tau_g = a^2/(H_A\kappa)$, where a is a characteristic distance through which interstitial fluid flows, H_A is a characteristic elastic modulus, and κ is the hydraulic permeability in the direction of the flow. So, taking into account the biphasic rheology, we generalize Eq. (2) as

$$\dot{D} = \frac{C}{(1-D)^k}\int_0^t e^{-\dot{\varepsilon}_g(t-\tau)}\frac{d}{d\tau}(\varepsilon(\tau)^m|\dot{\varepsilon}(\tau)|^n)\,d\tau, \tag{3}$$

where the compressive strain is assumed to be positive. Note that, generally speaking, the form of Eq. (3) was motivated by the form of viscoelastic constitutive relations, such that for high-strain compression, when $\dot{\varepsilon}_g \ll \dot{\varepsilon}_0$, Eq. (3) reduces to Eq. (2). It should be noted that the determination of the gel diffusion time crucially depends on the method of sample fixation (e.g., whether it is stripped of the bone, or placed into a confining chamber to prevent the radial flow of the interstitial fluid).

3. Damage evolution under impact loading

We consider a cartilage sample of thickness h and radius a impacted with a rigid impactor of mass M, so that the equation of the impactor motion can be written as follows:

$$\begin{aligned} &Mh\ddot{\varepsilon} = -F, \quad t\in(0, t_c),\\ &\varepsilon(0) = 0, \quad \dot{\varepsilon}(0) = \dot{\varepsilon}_0. \end{aligned} \tag{4}$$

Here, t_c is the contact duration, ε is the strain in the cartilage sample, F is the contact force, and $\dot{\varepsilon}_0 = v_0/h$ is the initial strain rate with v_0 being the initial impactor speed. Assuming that under impact loading, articular cartilage deforms like an incompressible material [14], and using the asymptotic solutions [1,2,4,5,8], we represent an approximate relation between the contact force F and the impactor displacement εh for a thin cylindrical incompressible elastic sample bonded to a rigid substrate as follows:

$$F = \frac{3\pi a^4}{8h^2}G(1-D)\varepsilon, \tag{5}$$

where G is the shear modulus of the virgin material (for an incompressible material $G = E/3$).

The impact problem with damage is formulated by Eqs. (3), (5), (4). In impact testing, the contact duration is usually very small and one may assume that $\dot{\varepsilon}_g/\omega \ll 1$, where

$$\omega = \sqrt{\frac{3\pi a^4 G}{8h^3 M}}$$

is a characteristic angular frequency, thus simplifying Eq. (3) to the form of Eq. (2). For example, t_c is about 0.5 ms for the impactor mass $M = 100$ g and the initial impactor speed $v_0 = 1.25$ ms^{-1} corresponding to a free drop of the impactor from a hight of 80 mm above the cartilage surface [9]. Correspondingly, it can be shown that in the range of small damage impacts, the accumulated damage $D_0 = D(t_c)$ is governed by the product $\omega^{-n-1}\dot{\varepsilon}_0^{m+n}$, which is proportional to $v_0^{m+n}M^{(m+n)/2}$. The latter quantity is reduced to the impact energy $Mv_0^2/2$, if $m = 1$ and $n = 1$.

In their experiments on matrix damage and chondrocyte viability, [16] observed an almost linear relation between the measured chondrocyte viability and the impact energy (defined as the initial kinetic energy of the impactor). Thus, assuming a proportionality between the viability and the quantity $1-D$, representing the residual resource of the damaged material, we should have an almost proportionality of D to the impact energy in the range of experimental data. On the other hand, approximately the same result is predicted by our model (3)–(4) provided $m = n = 1$. It is interesting to note that in the case $n = 1$, the parameter C, which enters Eqs. (2) and (3) is dimensionless.

Fig. 1 presents the experimental data of [16] and the theoretical predictions based on Eq. (3) in the case $k = m = n = 1$ with the only fitting parameter C (which is taken to be 1.82) for the following set of model parameters $h = 1.8$ mm, $a = 2.5$ mm, $M = 500$ g, $G = 385$ MPa, and $\dot{\varepsilon}_g = 10^{-3}$ s^{-1}.

Now, let us return to Eq. (4) and integrate it with Eq. (5) taken into account, thus arriving at the first integral

$$\frac{1}{2}(\dot{\varepsilon}^2 - \dot{\varepsilon}_0^2) + \omega^2\int_0^{\varepsilon}(1-D)\varepsilon\,d\varepsilon = 0. \tag{6}$$

Recall [3,28] that the contact duration, t_c, is determined as the time event $t = t_c$, when the contact reaction, $-F(t)$, vanishes. In view of Eq. (5), this condition reduces to the equation $\varepsilon(t_c) = 0$.

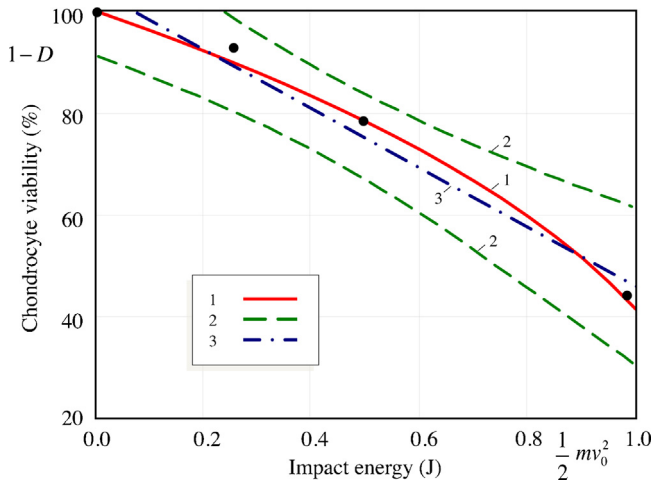

Fig. 1. Chondrocyte viability following impact as a function of impact energy: Experimental data (•), lines (2) of 95% confidence limits with the corresponding linear regression approximation (3) [16] and the damage model fit result (1) for $C = 1.82$.

Therefore, for the coefficient of restitution, e_*, Eq. (6) yields the following representation:

$$e_*^2 = 1 - \frac{2\omega^2}{\dot{\varepsilon}_0^2}\int_0^{\varepsilon_0}(D_- - D_+)\varepsilon\, d\varepsilon. \tag{7}$$

Here, ε_0 is the maximum deformation, D_+ and D_- are the damage evolutions in the loading and unloading phases, respectively.

Formula (7) allows to interpret, at least partially, the decrease of the coefficient of restitution with the increase of impact energy observed in experiments [30]. Again, in the range of small damage impacts, it can be shown that the quantity $1 - e_*^2$ is governed by the product $v_0^{m+n}M^{(m+n)/2}$, which reduces to the impact energy for $m = n = 1$. We note that the phenomenological model $m = n = k = 1$, $C = 1.82$ predicts $e_* = 0.83$ for the impact energy 1 J, while the drop in the coefficient of restitution observed in experiments is twice as much [12]. To account the viscous damping effect, the damaging viscoelastic model [10] can be applied.

4. Discussion and conclusion

First of all, we point out one peculiarity of the damage model (3). In the case of non-monotonic compression (for instance when $\varepsilon = \varepsilon_0 \sin\omega t$), the damage intensity, that is the right-hand side of Eq. (3), may take negative values during time intervals where the strain rate $|\dot{\varepsilon}|$ is small. This effect of healing is especially pronounced in the low strain mode of matrix damage, where the strain rate $\dot{\varepsilon}_0 = \omega\varepsilon_0$ goes down to the gel diffusion rate of deformation $\dot{\varepsilon}_g$. While for inorganic materials, it is usually assumed that $\dot{D} \geq 0$ during the deformation, the damage accumulation and healing process represent two competitive phenomena accompanying the deformation process in living tissues.

Second, in the range of small damage deformation, it was found that some reasonable predictions can be drawn from the simple case $m = n = 1$. That is why it is suggested to consider the following version of Eq. (3) as a first approximation to simplify the choice of the model parameters:

$$\dot{D} = \frac{C}{(1-D)^k}\int_0^t e^{-\dot{\varepsilon}_g(t-\tau)}\frac{d}{d\tau}(|\varepsilon(\tau)\dot{\varepsilon}(\tau)|^n)\, d\tau. \tag{8}$$

On the other hand, following [33], we rewrite Eq. (8) in terms of the specific dissipated power $\psi = \sigma\dot{\varepsilon}$ as follows:

$$\dot{D} = \frac{c}{(1-D)^k}\int_0^t e^{-\dot{\varepsilon}_g(t-\tau)}\frac{d}{d\tau}(|\psi(\tau)|^n)\, d\tau. \tag{9}$$

Here, c is a new dimensional constant, which is called the damage intensity factor. The last form of kinetic equation allows us to generalize the damage model for the three-dimensional case by substituting the expression $\psi = \sigma_{ij}\dot{\varepsilon}_{ij}$, where σ_{ij} are components of the stress tensor, $\dot{\varepsilon}_{ij}$ are the derivatives of the incremental (infinitesimal) strains ε_{ij} with respect to the time-like parameter t. It is to emphasize that the application of the damage model (9) is hindered by the necessity to specify the material constitutive law, which is a rather non-trivial question in the case of large deformations frequently encountered in experiments [23,32].

We further observe that the kinetic Eqs. (8) and (9) originate from Eq. (2) with $m = n$, which, in the case of half-sine loading $\varepsilon = \varepsilon_0 \sin\omega t$ predicts that the maxima of the damage intensity occur at the two time moments $t_1 = \pi/(4\omega)$ and $t_2 = 3\pi/(4\omega)$, which is consistent with the experimental evidence that the sample cracking can occur at the middle of the unloading phase [30].

Finally, returning to Eq. (3) and integrating it with respect to time, we obtain that an articular cartilage sample under compression will be fractured (completely damaged, $D(t_*) = 1$) at the time moment $t = t_*$, when the obtained double integral reaches some critical value, c_*. Therefore, after integration by parts the fracture criterion can be written in the form

$$\int_0^{t_*} e^{-\dot{\varepsilon}_g(t_*-\tau)}\varepsilon(\tau)^m|\dot{\varepsilon}(\tau)|^n d\tau \leq c_*, \tag{10}$$

which can be also modified by replacing $\varepsilon(\tau)^m$ with $\sigma(\tau)^m$, and represented in terms of the specific dissipated power ψ in the case $m = n$. Note that the fracture condition (10) belongs to the class of history-sensitive accumulation rules [21]. The quantity c_* represents a material property.

The present study results in a hierarchy of phenomenological models of damage evolution (2), (3), (8), and (9). Including the fracture criterion (10) they constitute the main result of the present study.

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References

[1] V.M. Aleksandrov, Asymptotic solution of the axisymmetric contact problem for an elastic layer of incompressible material, J. Appl. Math. Mech. 67 (2003) 589–593.
[2] I.I. Argatov, Development of an asymptotic modeling methodology for tibiofemoral contact in multibody dynamic simulations of the human knee joint, Multibody Syst. Dyn. 28 (2012) 3–20.
[3] I.I. Argatov, Mathematical modeling of linear viscoelastic impact: application to drop impact testing of articular cartilage, Tribol. Int. 63 (2013) 213–225.
[4] I. Argatov, G. Mishuris, Elliptical contact of thin biphasic cartilage layers: exact solution for monotonic loading, J. Biomech. 44 (2011) 759–761.
[5] G.A. Ateshian, W.M. Lai, W.B. Zhu, V.C. Mow, An asymptotic solution for the contact of two biphasic cartilage layers, J. Biomech. 27 (1994) 1347–1360.
[6] C.G. Armstrong, W.M. Lai, V.C. Mow, An analysis of the unconfined compression of articular cartilage, J. Biomech. Eng. 106 (1984) 165–173.
[7] W.C. Bae, M.M. Temple, D. Amiel, R.D. Coutts, G.G. Niederauer, R.L. Sah, Indentation testing of human cartilage sensitivity to articular surface degeneration, Arthritis Rheum. 48 (2003) 3382–3394.
[8] J.R. Barber, Contact problems for the thin elastic layer, Int. J. Mech. Sci. 32 (1990) 129–132.

[9] L.V. Burgin, L. Edelsten, R.M. Aspden, The mechanical and material properties of elderly human articular cartilage subject to impact and slow loading, Med. Eng. Phys. 36 (2014) 226–232.
[10] D.T. Davy, K.J. Jepsen, D.J. Krzypow, M.T. Fondrk, Nonlinear stress-strain behavior due to damage accumulation in cortical bone, in: IUTAM Symp. Synthesis in Bio Solid Mechanics. Solid Mechanics and its Applications, 2002, pp. 361–372.
[11] A.W. Eberhardt, L.M. Keer, J.L. Lewis, V. Vithoontien, An analytical model of joint contact, J. Biomech. Eng. 112 (1990) 407–413.
[12] L. Edelsten, J.E. Jeffrey, L.V. Burgin, R.M. Aspden, Viscoelastic deformation of articular cartilage during impact loading, Soft Matter 6 (2010) 5206–5212.
[13] B.J. Ewers, D. Dvoracek-Driksna, M.W. Orth, R.C. Haut, The extent of matrix damage and chondrocyte death in mechanically traumatized articular cartilage explants depends on rate of loading, J. Orthop. Res. 19 (2001) 779–784.
[14] J.J. Garcia, N.J. Altiero, R.C. Haut, An approach for the stress analysis of transversely isotropic biphasic cartilage under impact load, J. Biomech. Eng. 120 (1998) 608–613.
[15] S.M. Hosseini, W. Wilson, K. Ito, C.C. van Donkelaar, A numerical model to study mechanically induced initiation and progression of damage in articular cartilage, Osteoarthr. Cartil. 22 (2014) 95–103.
[16] J.E. Jeffrey, D.W. Gregory, R.M. Aspden, Matrix damage and chondrocyte viability following a single impact load on articular cartilage, Arch. Biochem. Biophys. 322 (1995) 87–96.
[17] L.M. Kachanov, On the creep rupture time, Izvestiya Akad. Nauk SSSR Otd. Tekhn. Nauk 8 (1958) 26–31.
[18] S. Knecht, B. Vanwanseele, E. Stüssi, A review on the mechanical quality of articular cartilage – implications for the diagnosis of osteoarthritis, Clin. Biomech. 21 (2006) 999–1012.
[19] J. Lemaitre, A Course on Damage Mechanics, Springer-Verlag, Berlin, 1992.
[20] M. Mengoni, J.P. Ponthot, A generic anisotropic continuum damage model integration scheme adaptable to both ductile damage and biological damage-like situations, Int. J. Plast. 66 (2015) 46–70.
[21] S.E. Mikhailov, I.V. Namestnikova, History-sensitive accumulation rules for lifetime prediction under variable creep loading, Arch. Appl. Mech. 81 (2011) 1679–1696.
[22] D. Milentijevic, P.A. Torzilli, Influence of stress rate on water loss, matrix deformation and chondrocyte viability in impacted articular cartilage, J. Biomech. 38 (2005) 493–502.
[23] V. Morel, T.M. Quinn, Cartilage injury by ramp compression near the gel diffusion rate, J. Orthop. Res. 22 (2004) 145–151.
[24] E. Pe na, Damage functions of the internal variables for soft biological fibred tissues, Mech. Res. Comm. 38 (2011) 610–615.
[25] T.M. Quinn, R.G. Allen, B.J. Schalet, P. Perumbuli, E.B. Hunziker, Matrix and cell injury due to sub-impact loading of adult bovine articular cartilage explants: effects of strain rate and peak stress, J. Orthop. Res. 19 (2001) 242–249.
[26] Y.N. Rabotnov, Creep Problems in Structural Members, North-Holland, Amsterdam, 1969.
[27] F. Richard, M. Villars, S. Thibaud, Viscoelastic modeling and quantitative experimental characterization of normal and osteoarthritic human articular cartilage using indentation, J. Mech. Behav. Biomed. Mater. 24 (2013) 41–52.
[28] N.S. Selyutina, I.I. Argatov, G.S. Mishuris, On application of Fung's quasi-linear viscoelastic model to modeling of impact experiment for articular cartilage, Mech. Res. Commun. 67 (2015) 24–30.
[29] J.A. Van der Sluijs, R.G.T. Geesink, A.J. van der Linden, S.K. Bulstra, R. Kuyer, J. Drukker, The reliability of the mankin score for osteoarthritis, J. Orthop. Res. 10 (1992) 58–61.
[30] F. Varga, M. Drzik, M. Handl, J. Chlpik, P. Kos, E. Filova, M. Rampichova, A. Necas, T. Trc, E. Amler, Biomechanical characterization of cartilages by a novel approach of blunt impact testing, Physiol. Res. 56 (Suppl. 1) (2007) S61–S68.
[31] N.P. Waters, A.M. Stoker, W.L. Carson, F.M. Pfeiffer, J.L. Cook, Biomarkers affected by impact velocity and maximum strain of cartilage during injury, J. Biomech. 47 (2014) 3185–3195.
[32] C.H. Yeow, Y.H. Ng, P.V.S. Lee, J.C.H. Goh, Tibial cartilage damage and deformation at peak displacement compression during simulated landing impact, 2010.
[33] M. Życzkowski, Creep damage evolution equations expressed in terms of dissipated power, Int. J. Mech. Sci. 42 (2000) 755–769.