



# 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.

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## 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 emphasize 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  $\varepsilon$  is the strain associated with a damaged state under the applied stress  $\sigma$ , 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

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

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 ( $\sim 930$  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  $3 \times 10^{-5}$  and  $0.7 \text{ s}^{-1}$  to a peak stress between 3.5 and 14 MPa. Observe that for articular cartilage samples compressed at  $\dot{\varepsilon}_0 = 3 \times 10^{-5} \text{ s}^{-1}$ , the peak compressive stresses 3.5 MPa and 14 MPa corresponded to peak compressive strains  $\varepsilon_0 = 83 \pm 5\%$  (mean  $\pm$  standard deviation) and  $\varepsilon_0 = 95 \pm 7\%$ , 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]):

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

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  $\varepsilon_0$ , 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  $\tau_g$  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  $\kappa$  is the hydraulic permeability in the direction of the flow. So, taking into account the biphasic rheology, we generalize Eq. (2) as

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

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:

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

Here,  $t_c$  is the contact duration,  $\varepsilon$  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  $\varepsilon h$  for a thin cylindrical incompressible elastic sample bonded to a rigid substrate as follows:

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

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  $v_0 = 1.25 \text{ ms}^{-1}$  corresponding to a free drop of the impactor from a height 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  $\dot{\varepsilon}_g = 10^{-3} \text{ s}^{-1}$ .

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

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

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  $\varepsilon(t_c) = 0$ .

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