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RHEOLOGICAL PROCESSES IN THE SURFACE LAYER OF ARTICULAR CARTILAGE UNDER LOADING

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Summary: *Articular cartilage in loaded joints consists of several layers that have different orientations of collagen fibers. In about 10-20% of the thickness, under the surface, there is the so-called 'superficial tangential zone', where the collagen fibers are oriented in one direction that is parallel with the surface and with the direction of joint movement. The strength and stiffness of these collagen fibers is greater than of other fibers. This zone represents the main member that ensures the strength of cartilage, and breaking of the collagen fibers in this zone is assumed to be the cause of cracks that are normal to the surface of cartilage. Such cracks were observed in joints with primary osteoarthritis. A mesomechanical rheological analysis of the deformation processes in the structure of this tangential zone discloses how strongly the stressing of the collagen fibers depends on the rate of loading of the joint, and how dangerous are very quick impact loadings.*

1. INTRODUCTION

Research of rheological, or generally mechanical processes in articular cartilage represents an import line because of scientific, as well as practical reasons. The practical – medical significance lies in the demand for making clear the aetiology of deterioration of cartilage and the ensuing osteoarthritis. The very complicated properties and structure of cartilage represent a challenge to scientific efforts for formulation and verification of mathematical models that would adequately describe cartilage from the mechanical point of view. The phenomenological models are aimed at lubrication and deformation under loading. The problem of lubrication is a special line, here we are going to concentrate on deformation under loading and the ensuing creation of cracks.

For modeling the deformation processes, the first natural scheme was description of cartilage as a biphasic poroelastic medium (Mow et al., 1980). This scheme was able to describe adequately the behavior of cartilage specimens under confined compression only. The reason was that in such conditions the reinforcing collagen fibers do not give any effect. However, confined compression does not correspond to the situation in vivo. For the description of the behavior of cartilage specimens in unconfined conditions, another – transversely isotropic – scheme was proposed by Cohen et al. (1992). Nevertheless, it was felt that for the description of the real behavior of cartilage in vivo it is necessary to take into account the reinforcing function of collagen fibers. In some papers (Farquhar , Dawson & Torzilli, 1990, Ault & Hoffman, 1992, Schwartz, Leo & Lewis, 1994) the authors related the microstructure of a composite solid to the macroscopic time-independent behavior of drained cartilage. This represented a step towards the understanding of the influence of the internal structure, but their model was rather complicated even for the time-independent process, and lacked the very important possibility of describing the time-dependent rheological processes that are essential for the

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understanding of the function of cartilage. Soulhat, Buschmann & Shiradzi-Adl (1999) and Fortin et al. (2000) presented a model that takes into account both the reinforcement by collagen fibers and the time-dependent rheological behavior. For modeling the response of the investigated specimens of cartilage in the form of circular discs, they assumed reinforcement of the disc by an axially symmetric network of collagen fibrils. This network consisted of three systems: straight fibrils normal to the surface of the specimen, straight fibrils in the radial directions, and circular fibrils. Although this network can hardly be recognized as realistic, they received a fairly good agreement of the described deformation processes with experimental results if the model parameters were assumed dependent on the degree of deformation and a curve-fitting procedure was used for their determination.

In all the above-mentioned models, cartilage is described as macroscopically homogeneous. In our approach we would like to get closer to the real configuration of the network of collagen fibers, which leads to the viewpoint that cartilage is not macroscopically homogeneous.

We are going to focus our attention especially on the creation of cracks at the surface of cartilage. Such cracks - apart from splitting at the interface between normal and calcified cartilage (tide mark) - have been observed in joints with primary osteoarthritis (Meachim & Bentley 1978). Whereas the splitting at the tide mark was possible to explain by a 'coated elastic sphere model' (Eberhard, Lewis & Keer 1991), the reasons for the appearance of the cracks normal to the surface are not so easy to find. Hlaváček (1994) discussed this question from the point of view of interstitial fluid motion and its possible influencing upon propagation of an already existing crack. He came to the conclusion that this effect could lead to the matrix failure at the crack edge. Nevertheless, it seems that the pivotal point of the problem consists in the initial creation of the cracks. The situation is different from that of technical materials, where small cracks exist a priori due to solidification, working, machining and corrosion and the heart of the problem is under what conditions they propagate. None of the factors enumerated above exists in articular cartilage and the heart of the problem is how the cracks originate.

The essential feature that we take into account in our way of modeling is distinguishing between different layers of cartilage, whose properties are different. According to Shrive & Frank (1994) and Kempson et al. (1973) the following layers can be distinguished:

(a) In the *deep zone* the collagen fibers are perpendicular to the subchondral bone to ensure a high strength of the attachment. The thickness of this layer is about 30% of the whole thickness of cartilage.

(b) Above this deep zone, there is *transition zone*, where the direction of the collagen fibers changes. The thickness of this transition zone is about 40-60%.

(c) Above this transition zone, there is a *superficial tangential zone* with collagen fibers lying parallel to the surface of cartilage and parallel to the local predominant direction of dynamic loading, to the predominant joint movement that is specific for the spot. It is well known that this direction can experimentally be determined by the so-called ‘split lines’ that can be created by pricking the cartilage surface with a sharp tool. The thickness of this layer with strong parallel collagen fibers is about 10-20% of the whole thickness of cartilage.

(c) Above this superficial tangential zone there is a surface layer – *lamina splendens*, where collagen fibrils form randomly oriented flat bundles. This ‘skin’ of the cartilage is only about 2 μm thick and it ensures an overall strength of the surface, and low friction.

Our approach is based on the above-mentioned findings, and starts from the following assumptions:

Assumption 1:

We assume that the *superficial tangential zone* with strong parallel collagen fibers is the most important factor resisting creation of cracks at the surface of cartilage. Therefore, we will concentrate our attention to this zone. Let us corroborate this assumption by some experimental findings:

- The significance of the *superficial tangential zone* follows from the mere fact of existence of the ‘split lines’.
- The predominant role of the *superficial tangential zone* from the point of view of strength and stiffness was shown by Broom (1984). He calls it ‘strain-locking role’.

- The strength and stiffness of collagen fibers is greater in directions parallel to the split lines than perpendicular to it (Kempson et al. 1973, Shrive & Frank 1994).
- The stiffness of cartilage as a whole (if taking into account the whole thickness of cartilage) is higher in the direction of the split lines (Kamalanathan & Broom 1993**).**

Assumption 2:

It is assumed that the superficial fissures arise due to the breaking of collage fibers in the *superficial tangential zone*, and that their predominant direction is therefore normal to the direction of fibers in this zone. This point of view is corroborated by the following findings:

- According to Broom (1986), extension *without rupture* of cartilage specimens in the direction normal to the direction of collagen fibers can be 6times up to 10times larger than in the direction of fibers. Collagen fibers extend less than 5% before rupture (Broom, 1984). The fissures can therefore be hardly parallel with the fibers in the *superficial tangential zone,* as extension in the direction normal to the fibers can be very large without creation of any rupture.
- According to Haut, Ide & DeCamp (1995) no impact fissures appeared to extend deeper than the upper one-third of the cartilage after an impact. This means that the fissures arose by braking through the relatively stiff *superficial tangential zone.* The deeper zone is more compliant and is not torn by the relatively small deformations.
- According to Donahue et al. (1983) blunt impacts resulted in damage of collagen fibrils in cartilage and cleavage of tissue proteoglycans.

Assumption 3:

The creation of the cracks is assumed to start with fracture of collagen fibers in the *superficial tangential zone*, not with debonding of the fibers with the matrix.

- This is corroborated by the experiments described by Broom (1984). These experiments were specifically aimed at the question of debonding and breaking of fibers.

Assumption 4:

Impact loading is assumed to be the primary cause of the creation of cracks in the *superficial tangential zone.* This is corroborated by the following findings:

- Experiments performed by Arokoski et al. (1994) showed that fatigue, resulting from a very long distance running training, lasting one year, caused in the canine joints no fissures, it caused only softening of cartilage. Hence, fatigue does not seem to be the cause of the creation of the cracks.
- The experimental results presented by Torzilli et al. (1999) 'clearly indicate that a joint impact stress at or above the critical threshold of 15-20 MPa will not only cause cell death, but also permanent damage to the collagen fiber matrix'.
- The question whether – under an impact load – breaking of the subchondral bone or the creation of cracks in the cartilage are primary, was discussed by Haut, Ide & DeCamp (1995). It was shown that the results of experiments in vitro of some authors can be interpreted such that primary damage was detected in the subchondral bone, whereas other authors detected primary damage in the cartilage (e.g. Donahue et al. 1983). The differences corresponded to different values of the contact pressures. Moderate contact pressures led to primary damage of cartilage, high values of contact pressures led to damage of subchondral bone. If we have in mind the development of damage in vivo, the first stage is no damage, some higher values of contact pressures lead to fissures in cartilage, and very high values to damage of subchondral bone. With our assumptions we will investigate the second stage.

2. MESOMECHANICAL ANALYSIS

Based on the above-presented assumptions, our model describes the superficial tangential zone of articular cartilage as a medium reinforced with unidirectional continuous fibers. The mathematical background of the analysis starts from the author's general model of fiber-reinforced materials, presented in (Kafka, 1987, 2001). The superficial tangential zone is described as a three-phase medium. One phase corresponds to the collagen fibers (phase f), the second phase to the matrix in

which the fibers are embedded, and the third phase to the thin constituent of synovial fluid that infiltrates the matrix. Apart from instantaneous small elastic compressibility, the filtration of the viscous liquid out of cartilage under the action of pressure causes time-dependent compressibility of the second and the third phases together (phase 'non-fibers' – labeled by index n). However, there are two singular cases in which this time-dependent compressibility of the n-phase gives no effect:
(i) Under an impact loading, where the filtration of the liquid out of cartilage does not take place, because there is no time for it.
(ii) Under long-lasting static loading, where the filtration of the liquid out of cartilage does not take place any more, because all the liquid is gone.

From these two singular cases the first one is more dangerous as the impact loading can lead to higher stresses.

From the macroscopic point of view, our mathematical model is transversely isotropic, its axis of symmetry $\boldsymbol{x_1}$ is parallel with the fibers, direction $\boldsymbol{x_2}$ is normal to the surface and parallel with the direction of compressive loading, direction $\boldsymbol{x_3}$ is parallel with the surface. The volume fractions and Young's moduli are v_f, v_n, E_f, E_n respectively.

The complete model of the structure is formulated on two scales:

The first scale is the structure composed of the fibers (f) and the rest of cartilage, i.e. the phase non-fibers (n).

The other scale describes the filtration in the structure of the n-phase, composed of the matrix (m) and the infiltrated viscous thin constituent of the synovial fluid (v).

3. FIRST MESOSCALE MODEL

On the first scale, the fundamental set of equations, on which our mesomechanical analysis is based, is according to Kafka (1987):

$$v_f \sigma_{ijf} + v_n \sigma_{ijn} = \bar{\sigma}_{ij} \tag{1}$$

$$v_f \varepsilon_{ijf} + v_n \varepsilon_{ijn} = \bar{\varepsilon}_{ij} \tag{2}$$

$$\dot{e}'_{ijf} = \dot{e}_{ijf} - \dot{\bar{e}}_{ij}, \qquad \dot{\varepsilon}'_f = \dot{\varepsilon}_f - \dot{\bar{\varepsilon}} \tag{3}$$

$$\dot{e}'_{ijn} = \dot{e}_{ijn} - \dot{\bar{e}}_{ij}, \qquad \dot{\varepsilon}'_n = \dot{\varepsilon}_n - \dot{\bar{\varepsilon}} \tag{4}$$

$$\dot{e}_{ijf} = \mu_f \dot{s}_{ijf}, \qquad \dot{\varepsilon}_f = \rho_f \dot{\sigma}_f \tag{5}$$

$$\dot{e}'_{ijf} = \mu_f \dot{s}'_{ijf}, \qquad \dot{\varepsilon}'_f = \rho_f \dot{\sigma}'_f \tag{6}$$

$$\dot{e}_{ijn} = \mu_n \dot{s}_{ijn}, \qquad \dot{\varepsilon}_n = \rho_n \dot{\sigma}_n + \sigma_n / h_n \tag{7}$$

$$\dot{e}'_{ijn} = \mu_n \dot{s}'_{ijn}, \qquad \dot{\varepsilon}'_n = \rho_n \dot{\sigma}'_n + \sigma'_n / h_n \tag{8}$$

$$\varepsilon_{11f} = \varepsilon_{11n} = \bar{\varepsilon}_{11} \tag{9}$$

$$\sigma_{ijn} - \sigma_{ijf} + \frac{\sigma'_{ijn}}{\eta_n^{\oplus}} = 0 \quad \text{for} \quad ij = 22, 33 \tag{10}$$

where $\sigma_{ij}, \varepsilon_{ij}$ mean stress- and strain-tensors, $v_f, v_n\ (v_f + v_n = 1)$ are volume fractions, $e_{ijf} / s_{ijf} [\delta_{ij}\varepsilon_f / \delta_{ij}\sigma_f]$ are the deviatoric [isotropic] parts of the average strain/stress tensors in the *f*-constituent and similarly with the *n*-constituent ('non-fibers'), and the macroscopic values are indicated by overbars. The symbols μ [=(1+ν)/E] and ρ [=(1-2ν)/E] mean deviatoric and isotropic elastic compliances, ν meaning Poisson´s ratio and E Young´s modulus. The symbols with primes – defined by Eqs. (3), (4), (6) and (8) – characterize the influence of fluctuations, of the heterogeneity of strain- and stress-fields. By the symbol h_n the time-dependent compressibility of the *n*-constituent is described in our model as a 'volumetric viscosity'. The value of h_n is positive, it is not a constant, the evolution of the value of h_n with changing deformation and loading follows from the analysis on the second scale. The symbol $\eta_n^{\oplus}$ is the 'structural parameter' that – in the model representation – describes the configuration of the imbedding of the fibers in the *n*-phase.

From the above set of basic equations, the macroscopic mesomechanical constitutive equation for a transversely isotropic material with unidirectional continuous fibers can be derived (for details see Kafka, 1987). In what follows, only a simplified version is presented, in which the values of Pisson's ratios are assumed identical, i.e. $\nu_f = \nu_n = \nu$, which enables elimination of one of the elastic constants $\rho_n = \dfrac{\mu_n}{\mu_f}\rho_f$:

$$\dot{\bar{\varepsilon}}_{11} = \frac{1}{v_f\,\mu_n + v_n\,\mu_f}\left[\mu_n \frac{\rho_f - \mu_f}{3}\dot{p} + v_n\,\mu_f \frac{\sigma_n}{h_n}\right] \tag{11}$$

$$\begin{aligned}\dot{\bar{\varepsilon}}_{22} = &\left\{\frac{2\mu_f + \rho_f}{3} + v_n \frac{\mu_n - \mu_f}{2\mu_f + \rho_f}\left[\frac{(\rho_f - \mu_f)^2}{3(v_f\,\mu_n + v_n\,\mu_f)} + (2\rho_f + \mu_f)M^{\oplus}\right]\right\}\dot{p} + \\ &+ \frac{v_n\,\mu_f}{h_n(2\mu_f + \rho_f)}\left[\left(\frac{\rho_f - \mu_f}{v_f\,\mu_n + v_n\,\mu_f} + 3\right)\sigma_n - 3(\mu_n - \mu_f)(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\right]\end{aligned} \tag{12}$$

$$\begin{aligned}\dot{\bar{\varepsilon}}_{33} = &\frac{\rho_f - \mu_f}{3}\left[1 + v_n \frac{\mu_n - \mu_f}{2\mu_f + \rho_f}\left(\frac{\rho_f - \mu_f}{v_f\,\mu_n + v_n\,\mu_f} + 3M^{\oplus}\right)\right]\dot{p} + \\ &+ \frac{v_n\,\mu_f}{h_n(2\mu_f + \rho_f)}\left[\left(\frac{\rho_f - \mu_f}{v_f\,\mu_n + v_n\,\mu_f} + 3\right)\sigma_n - 3(\mu_n - \mu_f)(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\right]\end{aligned} \tag{13}$$

where

$$M^{\oplus} = \frac{v_f\mu_f + \mu_n\eta_n^{\oplus}}{v_f(v_f\mu_n + v_n\mu_f) + \mu_n\eta_n^{\oplus}} \tag{14}$$

$$N^{\oplus} = \frac{v_f^2}{v_f(v_f\mu_n + v_n\mu_f) + \mu_n\eta_n^{\oplus}} \tag{15}$$

$$N'^{\oplus} = \frac{v_f}{v_f(v_f\mu_n + v_n\mu_f) + \mu_n\eta_n^{\oplus}} \tag{16}$$

$p = \bar{\sigma}_{22}$ is the compressive loading that is normal to the surface,

$\sigma_n = \frac{1}{3}(\sigma_{11n} + \sigma_{22n} + \sigma_{33n})$ is an internal variable represented by the isotropic part of the average stress tensor σ_{ijn} in the *n*-phase, σ'_n is another internal variable.

The respective evolution equations are given below:

$$\dot{\sigma}_{22n} = M^{\oplus}\dot{p} - \frac{\mu_f}{\rho_f}\left[(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\frac{1}{h_n}\right] \tag{17}$$

$$\dot{\sigma}_{33n} = -\frac{\mu_f}{\rho_f}\left[(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\frac{1}{h_n}\right] \tag{18}$$

$$\begin{aligned}\dot{\sigma}_{11n} = {} & \frac{\rho_f - \mu_f}{2\mu_f + \rho_f}\left[\frac{\mu_f}{v_f\,\mu_n + v_n\,\mu_f} - M^{\oplus}\right]\dot{p} + \\ & + \frac{\mu_f}{h_n(2\mu_f + \rho_f)}\left[-\frac{3v_f}{(v_f\,\mu_n + v_n\,\mu_f)}\sigma_n - \frac{2(\mu_f - \rho_f)}{\rho_f}(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\right]\end{aligned} \tag{19}$$

$$\begin{aligned}\dot{\sigma}_n = {} & \frac{\mu_f}{2\mu_f + \rho_f}\left\{\left[\frac{(\rho_f - \mu_f)}{3(v_f\,\mu_n + v_n\,\mu_f)} + M^{\oplus}\right]\dot{p} - \right. \\ & \left. -\frac{1}{h_n}\left[\frac{v_f}{v_f\,\mu_n + v_n\,\mu_f}\sigma_n + 2\frac{\mu_f}{\rho_f}(N^{\oplus}\sigma_n - N'^{\oplus}\sigma'_n)\right]\right\}\end{aligned} \tag{20}$$

$$\dot{\sigma}'_n = -\frac{\mu_f}{3\mu_n}\dot{p} + \frac{v_f\,\mu_n + v_n\,\mu_f}{\mu_n}\dot{\sigma}_n + \frac{v_f\,\sigma_n - \sigma'_n}{\rho_n h_n}. \tag{21}$$

The stress-components in the fibers (the *f*-phase) can easily be received from equation (1). This relatively complicated model can substantially be simplified, if it is assumed that the distribution of normal stresses in the directions normal to the fibers, i.e. in the x_2- and x_3- directions is approximated as homogeneous. In our model, such simplified version can easily be received by putting $\eta^{\oplus} = \infty$ (cf. Kafka 1987, 2001), which leads to $M^{\oplus} = 1,\ N^{\oplus} = N'^{\oplus} = 0.$ Then the only internal variable is σ_n. Let us explicitly write down the important formula for the tensile stress σ_{11f} in fibers, which results for this simplified model in the following form:

$$\dot{\sigma}_{11f} = -\frac{v_n}{v_f}\dot{\sigma}_{11n} = \frac{v_n}{(2\mu_f + \rho_f)(v_f\,\mu_n + v_n\,\mu_f)}\left[-(\mu_f - \rho_f)(\mu_n - \mu_f)\dot{p} + 3\frac{\mu_f}{h_n}\sigma_n\right] =$$
$$= \frac{v_n}{v_n E_n + v_f E_f}\left[\nu(E_n - E_f)\dot{p} + E_n E_f \frac{\sigma_n}{h_n}\right]. \qquad (22)$$

In the above formula, it evidently holds $E_f > E_n,\ \dot{p} < 0,$ and therefore, the immediate elastic response to compressive loading $\dot{p}$ is in the fibers positive, i.e. tensile. It is evident as well ($\sigma_n < 0,\ h_n > 0$) that the time-dependent inelastic compressibility of the n-phase causes decrease of this tensile stress in the fibers. For the fictitious case of $E_f = E_n$ (elastic homogeneity) the elastic response of the tensile stress in the fibers disappears. The existence of this elastic response is bound to elastic heterogeneity. For the case of incompressibility ($\nu = 0.5$) this elastic response reaches its maximum with regard to the possible variations of ν. For $\nu = 0$ this elastic response in the fibers disappears (maximum compressibility).

4. SECOND MESOSCALE MODEL

As it was mentioned above, in the preceding first scale model the positive 'coefficient of volumetric viscosity' h_n in the n-phase is not a constant. Description of evolution of the value of this coefficient h_n is the subject of this section.

In reality, the pores of the n-phase are infiltrated by a thin constituent of synovial fluid. Under a step-load, the n-phase displays an immediate elastic response that is followed by a time-dependent leaking out of the fluid, which causes decrease of volume of this phase. This decrease is limited, the rate of the decrease diminishes with time.

In our model representation, this process is described on the second mesoscale in such a way that the n-phase is represented as an elastic medium with pores that are filled with a liquid that has a fictitious property, called 'volumetric viscosity'. This is an analogue to the common deviatoric viscosity, only deviatoric stress and strain are replaced by isotropic parts of the respective tensors. Then, the model-medium has the same properties as the real n-phase: Under a step-load, the model-medium displays an immediate elastic response that is followed by a time-dependent decrease of volume. This decrease is limited, the rate of the decrease diminishes with time. This means that our model describes the decrease of the volume of the pores, but does not describe the way in which the liquid leaks out.

For the respective mathematical representation, a special case of the very general model – presented in (Kafka, 2001) – is used. For an elastic medium with pores that are filled with a viscous liquid, the following set of equations results from the mentioned general model:

$$\dot{\varepsilon}_n = \rho_n \dot{\sigma}_n + M_v^o \frac{\sigma_n - v_m \sigma_m}{h_v} \qquad (23)$$

$$\dot{\sigma}_m = M_m^o \dot{\sigma}_n + v_v \frac{\sigma_n - v_m \sigma_m}{\rho_m (v_v^2 + \eta_m^o) h_v} \qquad (24)$$

where

$$\rho_n = \frac{v_m \rho_m \eta_m^o}{v_v^2 + \eta_m^o} \qquad (25)$$

$$M_v^o = \frac{v_v + \eta_m^o}{v_v^2 + \eta_m^o} \tag{26}$$

$$M_m^o = \frac{\eta_m^o}{v_v^2 + \eta_m^o}\,. \tag{27}$$

In the above relations, subscript n relates to the 'non-fibers' n-phase, subscript v to the infiltrated synovial viscous fluid, and subscript m to the matrix in which the pores are imbedded. Symbols ε_n, σ_n mean average isotropic parts of the strain and stress tensors in the n-phase, σ_m is an analogous variable in the matrix, v_v and v_m are volume fractions of the pores and of the matrix in the n-phase ($v_v + v_m = 1$), h_v is a *constant* coefficient of the fictitious volumetric viscosity of the infiltrated synovial fluid, η_m^o is a structural parameter that characterizes the configuration of the matrix around the pores.

In this special case, the internal variable σ_m can be excluded, and constitutive equation simplified. This can be done in such a way that the term $(\sigma_n - v_m\sigma_m)$ is expressed from Eq.(24) and used in Eq.(23). In this way we receive a differential equation with constant coefficients that can be integrated, giving:

$$\sigma_m = M_m^o\sigma_n + v_v\,\frac{\varepsilon_n - \rho_n\sigma_n}{\rho_m(v_v^2 + \eta_m^o)}\,. \tag{28}$$

With the use of Eq.(28) in Eq.(23) we finally get the simplest form without internal variables of the volumetric constitutive equation for the n-phase:

$$\dot{\varepsilon}_n = \frac{1}{v_v^2 + \eta_m^o}\left\{v_m\,\rho_m\,\eta_m^o\,\dot{\sigma}_n + \frac{v_v}{h_v}\left[(1+\eta_m^o)\sigma_n - \frac{v_m}{\rho_m}\varepsilon_n\right]\right\} \tag{29}$$

or – in a differential form:

$$\mathrm{d}\varepsilon_n = \frac{1}{v_v^2 + \eta_m^o}\left\{v_m\,\rho_m\,\eta_m^o\,\mathrm{d}\sigma_n + \frac{v_v}{h_v}\left[(1+\eta_m^o)\sigma_n - \frac{v_m}{\rho_m}\varepsilon_n\right]\mathrm{d}t\right\}.$$

The immediate elastic response is given by the first addend in the curly braces. The time-dependent decrease of volume is given by the other addend. At the beginning of a step loading process $\varepsilon_n = 0$, and the rate of volume change is maximal. With proceeding time, the value of ε_n increases, which diminishes the value of the expression in the square brackets. If ε_n reached the value $\varepsilon_n = \rho_m\,\dfrac{1+\eta_m^o}{v_m}\,\sigma_n$, the time-dependent process would stop. However, it is evident that the approaching to such a state is asymptotic.

For every step $\mathrm{d}p$ or/and of time $\mathrm{d}t$ the corresponding value of the parameter h_n can be calculated form Eq.(29):

$$h_n = \frac{\sigma_n}{\dot{\varepsilon}_n}\,. \tag{30}$$

This completes the theoretical model representation. After determination of the model parameters, which are constants, it is possible to quantitatively describe the whole deformation process.

5. CONCLUSION

The theoretical description presented above was shown to agree with many experimental findings, and it explains and models the phenomenon of the surface cracks in articular cartilage, as they are observed in joints with primary osteoarthritis. The support of this work by the Grant Agency of the Czech Republic under grant No. 103/00/008 is gratefully acknowledged.

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