THE IMPORTANCE OF THE COLLAGEN NETWORK ARCHITECTURE IN ARTICULAR CARTILAGE FOR THE MECHANICAL LOADING OF INDIVIDUAL COLLAGEN FIBRILS

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INTRODUCTION

The mechanical function of articular cartilage is determined by its high water content and the particular architecture of the collagen network. This network consists of cross-linked fibrils that extend perpendicular from the subchondral bone and curve gradually to a course parallel to the articular surface in the superficial zone [1]. It is thought that loss of the integrity of this collagen network is important in cartilage pathologies. For instance, cartilage swelling, which is proportional to the amount of collagen damage [2], is an early sign of articular cartilage degeneration in osteoarthritis [3]. Hence, it is hypothesised that for a mechanical analysis of functional changes during pathologies, the specific architecture of the collagen network must essentially be included. To test this hypothesis, a numerical model which incorporates this architecture is needed. However, such numerical models are not available at present.

The two aims of this paper are therefore 1) to present a poroelastic finite element model for articular cartilage, which is the first one to include a description of the arcade-like collagen fibril network, and 2) to show the relevance of using such a model with respect to fibril stresses in selective parts of the collagen network.

METHOD

The solid part of the biphasic cartilage model consists of a non-fibrillar and a fibrillar part. The non-fibrillar matrix resembles all cartilage contents except for the collagen fibrils. The fibrillar part, resembling the collagen matrix, is subdivided into a primary and a secondary fibril network. Primary fibrils run perpendicular to the subchondral bone in the deep zone, split up into smaller fibrils in the radial zone, and continue parallel to the articular surface in the superficial zone (Figure 1). This results in an arcade-like structure [1], with superficial fibrils running in all directions. The secondary fibrils form a homogeneous 3D network throughout the cartilage.

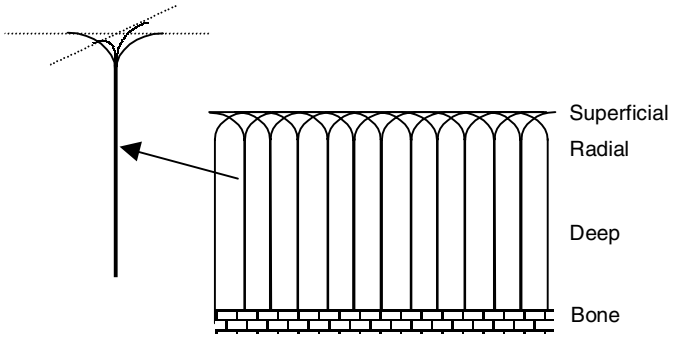

Figure 1. Right: schematic representation of the arcade model [1]. Left: Orientation of four primary collagen fibrils as implemented in the finite element model.

The non-fibrillar solid matrix is linear elastic, with homogeneous and strain-dependent permeability (k):

$$k = k_0\left(\frac{1+e}{1+e_0}\right)^M$$

where e represents the void ratio, defined as the fluid fraction divided by the solid volume fraction. M is a positive constant.

In the fibrillar part, both primary and secondary fibrils only resist tension and exhibit strain-dependent stiffness [4], according to

$$E_{f,p} = \rho_y C(E_0 + E_\varepsilon \varepsilon_f)$$

and

$$E_{f,s} = E_0 + E_\varepsilon \varepsilon_f$$

where E_0 and E_ε are positive material constants, ε_f is the fibril strain, ρ_y represents the local fibril density and C is the fraction of primary to secondary fibrils.

The initial orientation of each fibril is given by $\vec{v}_0$. After deformation, the new fibril vector ($\vec{v}_{new}$) is computed by

$$\vec{v}_{new} = \underline{F} \cdot \vec{v}_0$$

where $\underline{F}$ is the deformation gradient tensor. The logarithmic fibril strain can then be computed as

$$\varepsilon_f = \log \frac{\|\vec{v}_{new}\|}{\|\vec{v}_0\|}$$

and the fibril stress equals

$$\sigma_f = \int \partial \sigma_f = \int E_f \partial \varepsilon .$$

Substitution of the equations which determine the strain-dependent stiffness for the primary and secondary fibrils, results in the following equations for the stress in the primary and secondary fibrils:

$$\sigma_{f,p} = \rho_y C \left(E_0 + \frac{1}{2} E_\varepsilon \varepsilon_f \right) \varepsilon_f$$

$$\sigma_{f,s} = \left(E_0 + \frac{1}{2} E_\varepsilon \varepsilon_f \right) \varepsilon_f$$

The total stiffness matrix of the collagen network is then obtained by summation of the fibril stiffness matrices of each fibril after rotation to the local element coordinate system. Finally, the total solid stiffness matrix is obtained by adding the anisotropic fibrillar stiffness matrix to the isotropic non-fibrillar one.

RESULTS

Material parameters are partly derived from the literature [5] and partly by fitting the model to unconfined compression and indentation data from the literature [6] (Figure 2).

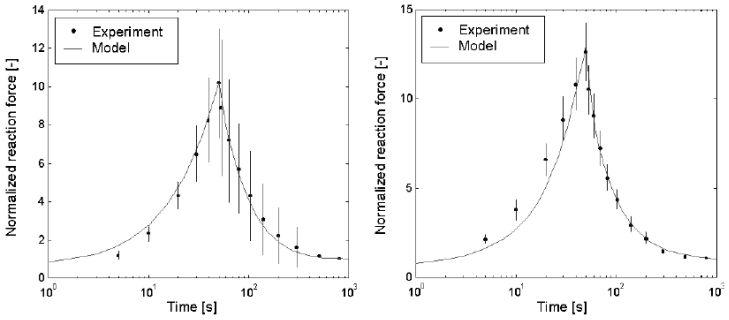

Figure 2: Axial reaction force, normalized to equilibrium, in unconfined compression tests (left) and indentation tests (right) along with the fibril-reinforced model curve fit.

The importance of taking the collagen network architecture into account for the mechanical loading of the collagen fibrils is illustrated by considering the stresses in the collagen fibrils during an indentation test (Figure 3). It is found that primary fibrils at the same location in the superficial zone experience different mechanical loading, depending on their origin. In an indentation test, fibrils which curve away from the indentor are strained, whereas fibrils bending towards the center of the indentor are not strained. Hence, the former fibrils are more stressed, and consequently more susceptible to damage.

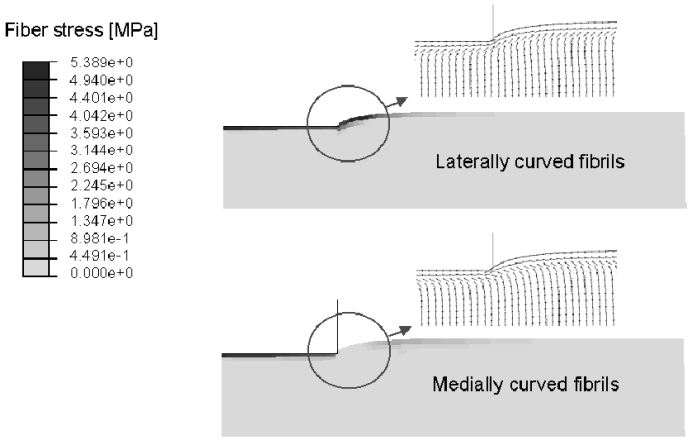

Figure 3: Contour plots of the maximal fibril stresses for fibrils bending away from the indentor (top) and towards the indentor (bottom).

DISCUSSION

With a newly developed poroelastic fibril-reinforced finite element model of articular cartilage that incorporates the arcade-like architecture of the collagen network, stresses in collagen fibrils in externally loaded articular cartilage are computed. It is found that fibrils at the same location in the cartilage experience different mechanical loads, depending on their orientation. This finding has important clinical implications, as it indicates that particular parts of the collagen network are more susceptible to damage than others, even though they are located at the same position in the cartilage. Such effects have not been published previously, and can only be studied numerically if the collagen architecture is taken into account as has been done in the present model. Hence, this model provides new insights into the functioning of the specific architecture of the articular cartilage collagen network. This contributes to understanding cartilage adaptation and cartilage pathologies such as osteoarthritis.

REFERENCES

1. Benninghoff, A., 1925, "Form und Bau der Gelenkknorpel in ihren Beziehungen zur Funktion", Z Zellforsch, Vol. 2, pp. 783-862.
2. Bank R.A., Soundry, M., Maroudas, A., Mizrahi, J., TeKoppele, J.M., 2000, "The increased swelling and instantaneous deformation of osteoarthritic cartilage is highly correlated with collagen degradation", Arthritis & Rheumatism, Vol. 43, pp 2202-2210.
3. Maroudas, A., 1976, "Balance between swelling pressure and collagen tension in normal and degenerated cartilage", Nature, Vol. 260, pp. 808-809.
4. Li, L.P., Buschmann, M.D., Shirazi-Adl, A., 2000, "A fibril reinforced nonhomogeneous poroelastic model for articular cartilage: inhomogeneous response in unconfined compression", Journal of Biomechanics, Vol. 33, pp. 1533-1541.
5. Korhonen, R.K., Laasanen, M.S., Toyras, J., Rieppo, J., Hirvonen, J., Helminen, H.J., Jurvelin, J.S., 2002, "Comparison of the equilibrium response of articular cartilage in unconfined compression, confined compression and indentation", Journal of Biomechanics, Vol. 35, pp. 903-909.
6. DiSilvestro, M.R., Suh, J.K., 2001, "A cross-validation of the biphasic poroviscoelastic model of articular cartilage in unconfined compression, indentation, and confined compression", Journal of Biomechanics, Vol. 34, pp. 519-525.