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# CAN LOCATIONS OF CARTILAGE DAMAGE INITIATION BE EXPLAINED BY SHEAR-STRAIN INDUCED TEARS OF INTERFIBRILLAR BONDS?

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## Introduction

It is generally believed that mechanical overloading is one of the major causes for the onset of cartilage degeneration. The first goal of this study was to determine the influence of cartilage thickness on the prevalence and the location of collagen damage after mechanical overloading. The second goal of this study was to determine the cause and nature of this collagen damage. It was hypothesized that initial cartilage damage involves loosening of interfibrillar bonds between the collagen fibrils, while the fibrils themselves remain intact until severe degenerative changes are induced [1,2]. In this study both the maximal shear strains along the collagen fibrils and the maximal fibril strains were tested as possible candidates for causing collagen damage during mechanical overloading.

## Material and methods

Ten 8.5 mm osteochondral plugs were loaded 5 times with a spherical indenter ( $r=2$  mm) with 25 N in 19.5 seconds. Coll II degradation was evaluated histologically using antibodies against denatured coll II (Col2-3/4M) [3]. The shear strains along the collagen fibrils and the fibril strains were computed with a fibril-reinforced poroviscoelastic swelling FEA-model, which includes a description of the arcade-like collagen network and the swelling behavior of cartilage [4]. Cartilage thickness was measured for each sample. FEA simulations were performed for cartilage of variable thickness. The distribution of the computed strains was compared to the experimentally determined locations of collagen damage.

## Results

In the experimental data, the thinnest samples showed the most damage, located at the surface. Intermediate samples were damaged below the surface and the thickest samples were undamaged (Fig. 1, 2).

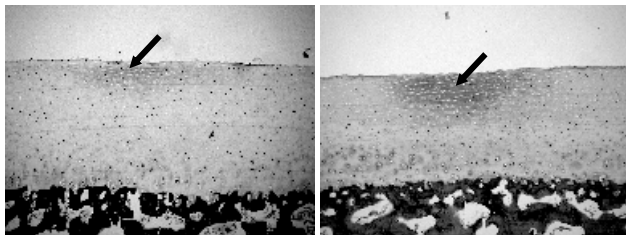


Fig 1: Col2-3/4M staining of cartilage with a thickness of 1.054 mm (left) and 0.871 mm (right). The dark areas represent mechanically induced collagen damage.

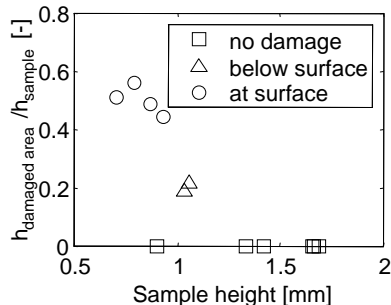


Fig 2: Relative height of the damaged areas as a function of cartilage thickness.

From the FE-analysis it was found that the fibril tensile strains were maximal at the articular surface, at the center of contact with the indenter. The peak strains in the interfibrillar bonds were larger (Fig. 3, 4) and were located just below the surface, under the center of contact (Fig 3). Both parameters depended on sample height (Fig. 4).

The combination of experimentally found damage, sample thickness and computed strains reveals that the cross-links between the collagen fibrils fail at approximately 30.9% strain and that the fibrils themselves start to rupture at a strain of approximately 27.5% (Fig. 3). Samples thicker than 1.3 mm remain undamaged, samples with a thickness  $0.9 < h < 1.3$  mm are damaged just below the articular surface, and samples thinner than 0.9 mm develop damage at and below the articular surface as a result of the applied mechanical overloading (Fig. 4).

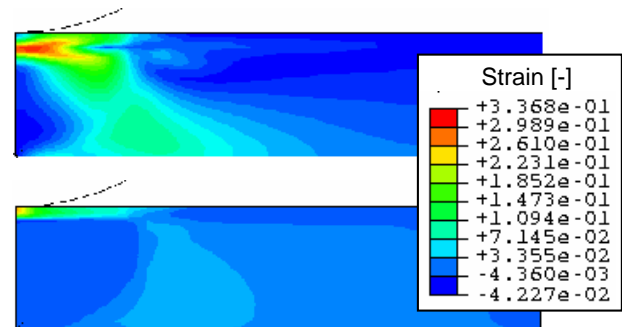


Fig 3: Maximal fibrillar (top) and interfibrillar strains (bottom).

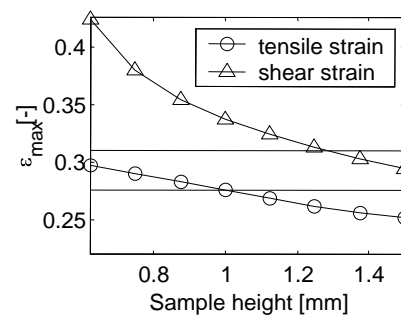


Fig 4: Relative value of the maximal fibril and interfibrillar strain as a function of cartilage thickness.

## Conclusions

Probability and location of collagen damage after mechanical overloading are highly dependent on cartilage thickness. The locations of damage predicted with the FEA model correspond well with experimental data. These findings strongly support the hypothesis that the earliest change in the collagen matrix integrity is loosening of the bonds between the large fibrils, and not rupture of the fibrils themselves.

## Reference

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