Can the locations of cartilage damage initiation be explained by shear-strain induced tears of interfibrillar bonds?

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**Introduction**

It was hypothesized that crack formation during impact loading of cartilage is preceded by loosening of bonds between the large collagen fibrils in the transitional and deep zones (Fig 1.), while the fibrils themselves remain intact until severe degenerative changes are induced [1,2].

![Figure 1: Histological section of tibial cartilage. Moderate degradation of collagen type II has been made visible using toluidine blue[3].](image)

We tested the plausibility of this hypothesis by comparing shear strains at fibril bonds to tensile strains in the fibrils themselves as possible candidates for failure during impact loading. We used a recently developed FEA-model, which includes a description of the arcade-like collagen structure characteristic for articular cartilage [4].

**Material and methods**

The articular cartilage was assumed biphasic. The solid phase consists of a linear elastic and isotropic non-fibrillar part and a viscoelastic fibrillar part of large primary fibrils and smaller secondary fibrils [4]. Bundles of primary fibrils extend perpendicular from the subchondral bone, splitting up close to the articular surface into fibrils curving to a horizontal course, flush with the articular surface. The network of secondary fibrils was represented as a random, homogeneous 3D network.

![Figure 2: Left: Orientation of four primary collagen fibrils as implemented in the FEA model. Right: cartoon of the arcade model of Benninghoff [5].](image)

The cartilage samples were modeled as axisymmetric, with thickness 0.5 mm and radius 4 mm. A spherical indentor with a radius of 2 mm was placed on the cartilage surface and loaded with 15 N in 1 ms. With this model the maximal shear strains along the primary fibrils, as well as the maximal tensile strains in the primary fibrils themselves, were computed.

**Results**

It was found that the maximal tensile strains in the primary fibrils were highest at the cartilage surface, at the center of contact (Fig 1A.). The maximal tensile strain was 8.26%. The shear strains along the primary fibrils were much larger at 35.32% (Fig 1B.). These peak shear strains were located just below the surface, under the center of contact. A second site of high shear strains was observed at the cartilage-bone interface, with peaks at 15.22%; fibrillar tensile strains were not excessive in this region.

![Figure 3: Maximal tensile strains in the primary fibrils after impact (A) and the maximal shear strains along the primary fibrils after impact (B), of a spherical indentor (radius 2 mm) of a force of 15 N in 1 ms.](image)

**Conclusions**

If we compare the peak fibrillar tensile strain of 8.26% we found with the failure strain of rat-tail tendons (10-18%), our results indicate that tensile fibril failure is not likely in this stage. Conversely, the high shear strains we found indicate that inter-fibrillar bond failure is likely. Hence, initial damage is likely to occur at the interfibrillar bonds between the primary fibrils and probably not in the primary fibrils themselves. As the maximal shear strains occurred just below the surface, our study also predicts that the initial damage in articular cartilage after impact loading will occur just below the articular surface. This site corresponds with the locations of cartilage softening as found by McCormack and Mansour [6].

**References:**

[5] BENNINGHOFF,ZEHLFORSCH, 2:783-862 (1925)