The mechanical consequence of removing the superficial zone of articular cartilage

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INTRODUCTION

Articular cartilage (AC) functions as a load-bearing, low friction, and wear-resistant material in diarthrodial joints. The distribution of AC matrix composition is highly depth-dependent. The fluid fraction in AC is 80% and decreases from surface to the depth of the tissue [1]. Collagen constitutes 70% of the tissue dry weight, and is highest in the superficial and deep zones and lowest in the middle zone [2]. Proteoglycans (PG’s) constitute 20–30% of the tissue dry weight. PG’s are lowest in the superficial zone, and highest in the middle zone. Although the PG content is lower in the deep zone than in the middle zone, the fixed charge density (FCD) is highest in the deep zone [3]. Apart from AC composition, its structure is also depth-dependent. In the superficial zone collagen fibers are densely packed, and are arranged parallel to the articular surface. In the middle zone collagen fibers are more randomly arranged. In the deep zone, the collagen fibers have their largest diameters and are arranged perpendicular to the subchondral bone (Fig. 1) [4].

Therefore when the superficial layer of cartilage is degraded, for instance during early osteoarthritis, the depth dependent structure and composition of the tissue is altered and consequently the response of the tissue to mechanical loads will change. It is commonly thought that without the superficial layer, cartilage becomes weaker, and therefore is strained more than intact cartilage, when subjected to external loading. Surprisingly, it was recently found that in cartilage without superficial tangential zone (STZ), the axial compressive strain in the directly-loaded region decreased by 2-3% rather than increased, and this effect was consistent over a range of osmotic pressures [5].

In this paper we aim to find an explanation for the decreasing axial compressive strain in a STZ-removed cartilage sample under compression compared to an intact cartilage sample, by using our previously validated fibril-reinforced poroviscoelastic swelling (FPVES) model of articular cartilage which is also able to account for the depth dependent structure and composition of the articular cartilage [6]. We hypothesize that this phenomenon is due to the depth dependent composition of articular cartilage.

MATERIALS AND METHODS

Computational Model

In the fibril-reinforced poroviscoelastic swelling theory articular cartilage is assumed as biphasic, consisting of a solid and a fluid phase. The solid phase consists of a non-fibrillar part, which contains charged PG’s and therefore exhibits an osmotic swelling pressure, and a fibrillar part representing the collagen network [6].

Approach

In order to test our hypothesis, we first calibrate our FPVES model of cartilage [6] using experimental data from Bevill et al. as our reference [5]. In the reference experiments are two sets of cartilage samples: Intact (1.74 mm thick) and STZ-removed (1.07 mm thick) (Fig. 1).

Figure 1. Intact (top), STZ-removed (bottom) cartilage specimen and model (experimental photos from [5])
Similar to the reference experiments, in silico the tissue was equilibrated at 0.15 M saline concentration for three hours, and after that a nominal compressive stress of 4.5 MPa was applied with a channel indenter for three hours. The indenter consists of two rectangular platens (8 mm x 3 mm) separated by a 1 mm channel relief zone (Fig. 2). First the model parameters were fitted such that the simulations matched the experimental results of the intact samples. Second, using the calibrated parameter value set, various simulations on the intact and STZ-removed cartilage samples were computed.

In fact, there are two main differences between healthy intact and STZ-removed samples, which together determine the stress-strain behavior of the sample. One is the tissue height, as the STZ-removed samples only have 62% of the intact group height. The other is the change in depth dependent structure and content of the matrix. In order to understand whether sample thickness or the change in depth-dependent matrix organization determines the difference in the experimental results, simulations are performed with both 1.74 and 1.07 mm tissue heights, and at each height a ‘normal’ depth dependent composition and a composition as if the surface was removed. As output, the axial compressive strains on the directly loaded surface of the experiments and computations are compared.

RESULTS

In simulations, the strain in STZ-removed tissue (1.07 mm thick) is 4% less than the strain in intact tissue (1.74 mm thick), similar to the reference experiments (Table 1, top rows). Interestingly, when tissue height was the same, the difference in strain between the two conditions increased (compare Table 1, top rows with bottom rows). If the change in strains would have been caused by a difference in sample height between intact and STZ-removed conditions, the strains should have ameliorated. Hence, we conclude that change in the thickness of the tissue is not responsible for the change in tissue strain after removal of the STZ.

To further test our hypothesis that in fact the depth dependent tissue composition is responsible, three additional simulations were performed. First, osmotic swelling pressure was assumed constant over the depth of the tissue, second, the stiffness of the non-fibrillar solid part was assumed constant, and third, both of them were constant with depth (Table 2).

Table 2. The strains on the directly loaded surface of the cartilage in simulations

<table>
<thead>
<tr>
<th>Tissue /Thickness (mm)</th>
<th>Constant osmotic pressure</th>
<th>Constant stiffness</th>
<th>Constant osmotic pressure and stiffness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact / 1.74</td>
<td>50%</td>
<td>50%</td>
<td>50%</td>
</tr>
<tr>
<td>STZ-removed / 1.07</td>
<td>47%</td>
<td>50%</td>
<td>52%</td>
</tr>
</tbody>
</table>

Table 1 and 2 together show that part of the depth dependent effect is explained by the osmotic swelling pressure, while the effect of depth dependent stiffness is more important. The explanation is that the solid fraction is increasing with tissue depth, so removing 38% of the tissue height from the upper layer will increase the average solid fraction and therefore enhance the average stiffness of the whole tissue.

In case both non-fibrillar stiffness and osmotic swelling pressure are constant over the depth of the tissue, the STZ-removed condition strains more than the intact one (Table 2). So the more the matrix composition depth dependency is removed in simulations, the more strain occurs in STZ-removed condition. As this is contrary to experimental observations, these computational results inversely support our hypothesis.

CONCLUSION

This paper explains the observation that larger strains are found in intact cartilage compared to STZ-removed tissue, based on the depth dependent composition of cartilage. Even though the arcade-like structure of the collagen itself contributes to a stiffer tissue, removing the STZ changes the depth dependent composition such that the bulk compressive stiffness of the tissue increases.

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REFERENCES