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Focal Articular Defects Modify Cartilage Contact Mechanics

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1 Introduction

Focal defects in articular cartilage enlarge over time. Mechanobiological mechanisms are likely to be involved in such responses, with aberrant biomechanical signals triggering cartilage deterioration. Although increases in articular contact stresses are minimized by the recruitment of new contact area [1], marked increases in macroscopic tissue deformation have been observed qualitatively [2]. Thus, we hypothesized that the presence of a focal defect alters the mechanics of opposing cartilage surfaces, producing locally elevated tissue strains and abnormal sliding between surfaces. The objective of the study was to quantify, using a 2-D experimental model of cartilage articulation, the effects of full-thickness defects on cartilage contact mechanics.

2 Materials and Methods

Sample Preparation. Macroscopically normal osteochondral blocks (2.5 x 10 x 5 mm^3; L x W x H) with flat articular surfaces were obtained from the femoral condyles of cadaveric human donors or bovine knees and stored at -70 ºC until testing. Blocks were thawed and stained for 4 hr at 4°C with propidium iodide in PBS + proteinase inhibitors (PI), and equilibrated 12 hr at 4 °C in bovine synovial fluid + PI.

Microcompression Testing (Fig. 1-2). Samples, comprising pairs of blocks (from the same knee), were placed in a microscope-mounted test chamber with their cartilage-bone surfaces flush with the glass chamber bottom and their articular surfaces in opposition and aligned perpendicular to the direction of loading (z-axis). Intra-tissue deformation was imaged through the chamber bottom (x-z plane) using fluorescent video microscopy (4x, 10x). Samples were compressed in the z-axis to 20% total tissue strain (axial displacement/sum of cartilage thicknesses), at a strain rate of 1.5%/s (~90 um/s). Digital images were acquired at 5 frames/s during loading. Full-thickness defects (4 mm wide) were created in one block from each sample, and the samples were retested.

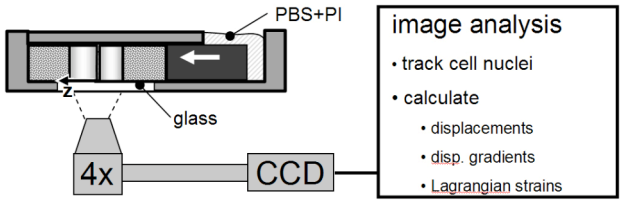

Figure 1 : Schematic of sample loading and image acquisition. Samples are microscopically imaged during loading, and sequential images are analyzed for sample displacement and strain.

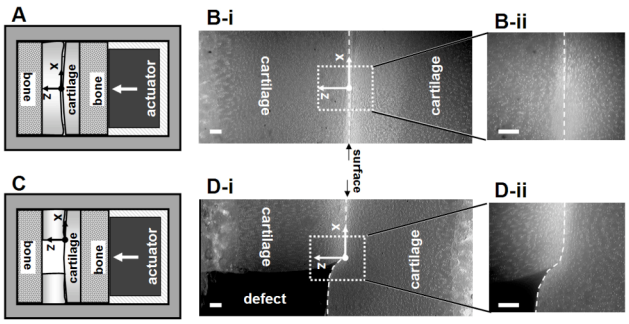

Figure 2 : Sample orientation (**A, C**) and representative images (**B, D**). Deformation of intact (**A, B**) and defect-containing (**C, D**) samples was imaged at 4x (**B-i, D-i**) and 10x (**B-ii, D-ii**) magnifications. Bar = 100 μm.

Digital Image Analysis. An evenly spaced sampling of cell nuclei, acting as fiducial markers, were automatically chosen in the unloaded image and tracked through the image sequence by an iterative image correlation process. Displacement

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gradients were calculated at uniformly spaced grid-points by finite difference approximations and used to calculate Lagrangian strains. From displacement fields, sliding between opposing surfaces was also quantified.

3 Results

Human and bovine cartilage displayed similar behaviors **(Fig. 3)**. Intact samples displayed a depth-varying decrease in compressive strain. The presence of a focal defect affected intra-tissue strain distributions in both the adjacent and opposing cartilage surfaces. The tissue at the defect rim displayed elevated compressive strains, as well as marked increases in tensile and shear strain. The opposing cartilage surface developed elevated tensile and shear strains in the region opposing the defect rim, while strains approached zero in the regions over the defect.

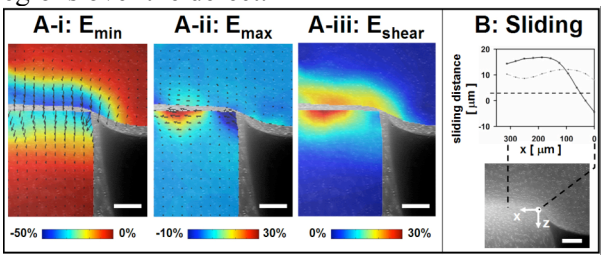

Figure 3 : Representative strain maps (**A**) and sliding distances (**B**). Strain maps of maximum compressive (**A-i**), tensile (**A-ii**), and shear (**A-iii**) strains arising near a defect in bovine cartilage compressed to 10% total tissue strain. The distance (**B**) that the opposing surface travels over each point on the adjacent cartilage surface (in the x-direction), in bovine samples compressed to 10% (gray dashed line) or 20% (black solid line) total strain. Bar = 100 μm.

4 Conclusion

The creation of a focal defect dramatically alters cartilage contact mechanics. Local increases in peak compressive, tensile, and shear strains are observed even in tissue compressed to the same total strain; peak strains are likely to be elevated even further, since higher overall strains are expected, to compensate for lost contact area. These conditions may lead to injurious levels of strain under physiological loading, possibly causing both cell death [3] and solid matrix damage [4]. Also, the induction of sliding between surfaces, due to the uneven deformation of the two sides, may accelerate cartilage wear. The effects of a defect may be accentuated by tangential loading, as would be present in the joint. The current study extends past research on cartilage deformation to delineate the details of altered contact mechanics due to a focal cartilage defect.

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References

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