INTRODUCTION: Focal defects in articular cartilage are common in symptomatic knees, and prone to enlarge over time if untreated. Articular defects in the knee are common in the medial femoral condyle and also in the patella. Under axial compressive load, the presence of focal defects leads to markedly elevated levels of macroscopic deformation and strains within cartilage tissue. However, during knee movement, focal defects are subjected to both axial compression and sliding motion, and the effects of such lateral motion on intra-tissue deformation remain to be elucidated. The local mechanical environment, reflected in tissue strain, markedly regulates chondrocyte metabolism; thus, defects may lead to progressive cartilage degeneration through elevated strains during physiological loading. Therefore, the objective of this study was to determine the effect of a focal cartilage defect on cartilage strains during the physiological stimuli of axial compression and sliding motion, as would occur during joint articulation.

METHODS: Samples. Macroscopically normal cartilage was harvested from the patella and trochlea of knees from 4 adult bovine animals (1-2yrs) as osteochondral blocks (each, ~3x8x7mm, WLH). Donor matched patella-trochlea samples (n=4) were stained with PBS+propidium iodide (20µg/ml, PPI)+proteinase inhibitors (PI) for 2h at 4°C, and then submerged in normal bovine synovial fluid+PPI+PI for ~12h prior to mechanical testing. Experimental Design. Samples were microscopically tested in shear as intact and then retested with a 3mm wide full thickness defect in the center of the patellar cartilage. Blocks were rinsed, allowed to reswell, and incubated for ~12-16h at 4°C inbetween tests. Microscopic Shear Testing. Each apposed sample pair (Fig. 1, schematic) was tested in a bi-axial loading chamber and viewed with an epi-fluorescence microscope. Cartilage was compressed by 1–AX=15%. After stress-relaxation for 1h, lateral displacements were applied, one for preconditioning, and then one for tests (each ∆x of +1 then –1 mm at 100 µm/s). Images (view field ~3x2mm²) contained the full thickness of the patellae cartilage and a partial view of the trochlear surface (Fig. 1A-F). Data Analysis. Cells (~500/image) were tracked using custom software to determine maximum displacement and Lagrangian strains in an area of ~1x1.5mm² in the patellar cartilage when the tissue was sliding away from the defect. For defect samples, strains were averaged and interpolated depth-wise, in regions (~0.2x1.5mm²) at (EDGE), ~0.4 mm (MID) and ~0.8 mm (FAR) away from the defect edge (Fig. 1D). Similarly for intact tissue, strains in corresponding regions were determined (Fig. 1A). Data are presented as mean±SEM. The effect of a focal defect and location on shear (E_xz) and axial (E_xx), and lateral (E_yy) strains were assessed by two-way repeated-measures ANOVA. Differences between defect and intact samples at various locations were assessed by Bonferroni post-hoc testing.

RESULTS: A focal defect resulted in marked changes in intra-tissue strains (Fig. 1). The effects of a defect on shear, axial, and lateral strains each varied markedly depending on depth from the articular surface (p<0.01, Fig. 2). To highlight the effects, at several depths relative to the articular surface (surface, 0% or 5% and superficial-deep, 20% or 25%), strains were graphed as a function of distance (EDGE, MID, FAR) from the defect edge (Fig. 3).

- Shear Strain. E_xz (Fig. 1AD, 2A, 3A) at the surface tended to be decreased by a defect FAR from the defect edge, from 0.12 to 0.02 (p=0.08). In contrast, E_xz at 25% depth was increased markedly FAR from the defect edge, from 0.02 to 0.08 (p<0.05).

- Axial Strain. E_xx (Fig. 1BE, 2B, 3B) at 5% depth was decreased markedly with a defect (p<0.05), being significantly lower (~0.08–0.10, i.e., compressed) at the EDGE up to the FAR regions than in intact tissue (+0.02, i.e., stretched). E_xx at 20% depth was not affected by a defect (p=0.5).

- Lateral Strain. E_yy (Fig. 1C, 2C, 3C) at 20% depth was markedly increased from ~0 in the intact tissue to 0.02 at EDGE (p<0.05) and 0.08 at the MID (p<0.01) region away from the defect edge. E_yy at the surface remained near 0 for both intact and defect groups (p=0.5).

DISCUSSION: These results provide the first maps of altered strain distributions in cartilage due to the combination of focal defects and cartilage-on-cartilage lateral articulation. These results extend findings of compression (without sliding) of cartilage with focal defects to identify regions of cartilage adjacent to the defect that undergo dramatic changes in tissue strain due to sliding, such as marked increases in shear and lateral strain at 20% and 25% depths, respectively. In these regions, such altered cartilage mechanics during knee movement may be a trigger of chondrocyte responses, such as cell death and matrix damage, and cause focal defects to expand and progress.


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Figure 1. Micrographs of patellar cartilage that is intact (A-C) or containing a focal defect (D-F) with superimposed shear (A,D), axial (B,E), and lateral (C,F) strain maps after applied lateral motion.

Figure 2. Effect of a focal defect on local shear (A), axial (B), and lateral (C) strain vs. depth when micro-shear-tested. Intact strains were not significantly different (p=0.7) between EDGE, MID, and FAR regions and thus averaged to produce a single strain vs. depth profile.

Figure 3. Effect of a focal defect on (A) shear (E_xz), (B) axial (E_xx), and (C) lateral (E_yy) strain magnitudes at particular tissue depths and lateral locations (EDGE, MID, FAR).