SUPPLEMENTARY INFORMATION

TROCHLEAR CARTILAGE DEFORMATION

Shear Strain (E_{xz}) – During patello-femoral articulation, E_{xz} varied with tissue depth ($p<0.001$) for TRO cartilage in articulation with both an intact and defect-containing surface, and markedly decreased in a depth-dependent manner ($p<0.001$) when TRO samples were slid against a focal defect (Figure 5A,B). With increasing depth from the articular surface, E_{xz} decreased from ~0.1 and ~0.03-0.04 near the surface to low magnitudes (≤ 0.01) near the tidemark for TRO cartilage in articulation with an intact and defect-containing surface, respectively (Figure 5C). Cartilage E_{xz} of TRO samples articulating against a defect was ~2-3x lower than that for samples articulated against intact cartilage near the surface (Figure 6A), at 20% tissue depth (Figure 6B), and overall (Figure 6C) for all lateral regions. Differences were statistically significant near the surface ($p<0.05$), while differences at 20% depth ($p=0.17$) and overall ($p=0.11$) displayed strong trends. Local (0.4-0.6) and overall E_{xz} (0.2) did not vary laterally for TRO cartilage in articulation with intact and a defect-containing PAT cartilage. Thus, TRO cartilage in direct apposition to the focal defect shears less as it slides over the proximal defect edge of PAT cartilage than when slid against an intact surface.

Axial Strain (E_{zz}) – Similar to TRO E_{xz}, $-E_{zz}$ decreased significantly with depth from the articular surface ($p<0.001$) for both intact (Figure 5D) and defect cases (Figure 5E), and was markedly lower ($p<0.001$) for TRO cartilage in articulation with a focal defect than with an intact surface following lateral articulation. With increasing depth from the articular surface, $-E_{zz}$ decreased markedly from ~0.36 and ~0.1-0.15 near the surface to ~0.05 and ~0.01 near the tidemark for TRO cartilage in articulation with an intact and defect-containing surface, respectively (Figure 5F). Surface (Figure 6D) and overall (Figure 6F) $-E_{zz}$ of cartilage

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articulating against a defect were ~2-4x lower ($p<0.01$) than that of cartilage in articulation against an intact surface. At 20% tissue depth, differences in cartilage $-E_{zz}$ due to a defect were not significant ($p=0.3$) (Figure 6E). Effects of lateral location were not apparent ($p=0.3$) on $-E_{zz}$ at 20% depth, while surface and overall $-E_{zz}$ tended ($p=0.1$) to decrease from EDGE to FAR lateral regions. Thus, TRO cartilage in direct apposition to the focal defect compresses less as it slides over the proximal defect edge of PAT cartilage than when compressed and slid against an intact surface.

Lateral Strain (E_{xx}) – Similar to PAT E_{xx}, the depth-variation as well as the magnitudes of E_{xx} were markedly different for TRO cartilage in articulation against an intact surface than that against a focal defect following lateral motion. When in articulation with intact PAT cartilage, TRO cartilage E_{xx} remained negligible (≤ 0.01) with tissue depth ($p=0.6$) for all lateral regions (Figure 5G), while for samples in articulation against a defect, E_{xx} varied markedly with tissue depth ($p<0.05$) and lateral region ($p<0.01$) (Figure 5H). At the FAR lateral region, E_{xx} decreased monotonically with increasing tissue depth, while at MID and EDGE lateral regions, peaked at ~20-30% tissue depth (Figure 5I). Near the surface, differences in E_{xx} between intact and defect cases were not statistically significant ($p=0.3$), but with increasing lateral distance, E_{xx} increased from -0.01 to 0.06 ($p<0.05$) for trochlear cartilage in articulation with a defect (Figure 6G). For samples sliding against a defect, TRO E_{xx} tended to be higher than that for samples in articulation against intact cartilage at 20% depth ($p=0.1$) (Figure 6H) and overall ($p=0.08$) at all lateral regions (Figure 6I). For defect cases, E_{xx} peaked ($p<0.05$) in the MID lateral region at 20% depth and decreased ($p<0.01$) overall from FAR to EDGE lateral regions. For intact samples, E_{xx} at 20% depth and overall remained negligible (<0.01) for all lateral regions. Thus,

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TRO cartilage initially adjacent to the defect edge compresses laterally, and regions farther away laterally are expanded laterally, as it slides over the proximal defect edge of PAT cartilage.

DISCUSSION

The biomechanical environment and boundary conditions of knee joint loading were considered in the experimental design of the present study. During joint loading, patello-femoral cartilage likely undergoes high compressive strain (5-10%)[26] and low sliding velocities (estimated from refs[27] and[28]) during contralateral toe-off and heel rise phases of gait, which were mimicked in this study by 12% compression and a 0.1mm/s sliding velocity. To replicate the physiologic articulation and lubrication of cartilage surfaces, osteochondral samples were isolated from the trochlear and patella of the same knee and tested in apposition with normal bovine synovial fluid. Although, samples were tested under a matching overall compressive strain instead of an identical compressive load which would occur physiologically, significant elevations in strains due to the presence of a focal defect were noted despite the conservative axial loading protocol. Furthermore, the aggregate modulus of human[29] and bovine[18] cartilage are both depth-varying; and thus, relative depth-varying changes in deformation in bovine cartilage due to a focal defect would likely be representative of that for human cartilage.

Under compression and prior to lateral motion, the tissue deformation of cartilage near, and in apposition to, a focal defect are consistent with previous studies.[12] For the current samples, PAT cartilage near the focal defect collapsed inward into the defect, and the apposing TRO cartilage partially filled the empty defect to a tissue depth of ~10% when compressed, being consistent with previous qualitative descriptions.[11,12] As a result, similar trends in intra-tissue strains were consistent with previous reports,[12] with E_{xz} and $-E_{zz}$ of cartilage near the defect

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being elevated, and strain of TRO cartilage in apposition to the focal defect increasing laterally (E_{xx}) and in shear (E_{xz}), and decreasing axially ($-E_{zz}$) (Figure 7) (data not reported).

The changes in cartilage tissue deformation were elucidated in the present study for a relatively small focal defect, which could also provide insight into cartilage strains near the edges of osteochondral grafts following mosaicplasty. During patello-femoral joint articulation, the unloaded contact area between intact surfaces[30] is reduced by ~30% for surfaces with a relatively small (~1 cm^2) focal defect. This was mimicked in the present study with sample contact area being reduced from 30mm^2 to ~21mm^2 following the creation of a 3mm wide focal defect. Relatively small focal defects were addressed to elucidate how changes in mechanical deformation may contribute to the degeneration of surrounding cartilage. Furthermore, large void regions exist between implanted osteochondral grafts immediately following mosaicplasty, and when loaded, tissue strains near the such regions are likely similar to those near a focal defect edge. Although moderate to good outcomes have been reported for mosaicplasty,[31] such abnormal strains in cartilage near the graft edges may be involved in causing the early osteoarthritic changes noted in some of the patients (~17%) following this procedure.[32]

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