Experimental and Finite Element Analysis of Strain Fields in Chondrocytes and the Extracellular Matrix of Cartilage Under Physiological Loading

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INTRODUCTION: The goal of this study was to investigate the deformation behavior of the chondrocyte and its microenvironment under transient loading, in order to address the relationship between the applied dynamic deformation and cellular strain. I*n-situ* strain measurements were performed on cells in the middle (MZ) zone at early time points during ramp loading. Furthermore, the cellular deformation results were interpreted in the context of a finite element analysis of chondrocyte deformation.

METHODS: ∅2mm cartilage samples from bovine calf carpometacarpal joints were cut diametrically to create semi-cylindrical specimens. The deformation at the zonal and cellular levels was characterized on the cross-section, under compressive loading using a custom motorized loading device mounted on an inverted microscope. The specimen was then loaded with a ramp displacement to 2% platen-to-platen strain, applied at 1 μm/sec. Deformation and strain were assessed using digital image correlation in four regions of interest within a field of view consisting of a cell with its surrounding pericellular matrix (PCM, Fig. 1). A total of 10 cells in two tissue samples were analyzed.

To investigate the local deformation behavior of chondrocytes *in-situ*, a finite element analysis (FEA) was also performed, using a multiscale approach[1]. The chondrocyte, modeled as a homogeneous protoplasm surrounded by a semi-permeable membrane, was embedded in the center of a cylindrical cartilage disk, modeled with the biphasic theory[2]. The FEA model was loaded as in the experimental study.

RESULTS: Representative principal normal strain contours, measured experimentally under transient ramp loading, are presented in Fig. 1. In general, the minimum (compressive) principal normal strain was observed to be highest inside the cell and lowest at the axial poles. The minimum strain (e_1) averaged -0.47±0.11% in the ECM, but was significantly higher in the intracellular region (e_1=-3.24±0.43%, $p < 0.05$). The PCM was exposed to a compressive strain of e_1=–2.05±0.69%, significantly smaller than measured intra-cellularly ($p < 0.05$).

For 2% ECM strain, the FEA produced e_1=-9.93±0.5% intra-cellularly. Strain contours showed the highest strain inside the cell and significantly lower e_1 strains at the axial poles (Fig. 2).

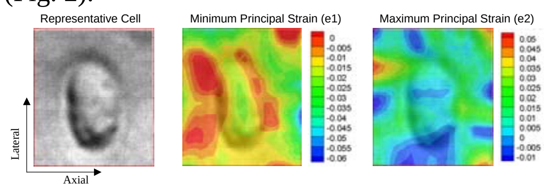

Fig. 1: Principal normal strains in a chondrocyte. The axial loading direction is horizontal.

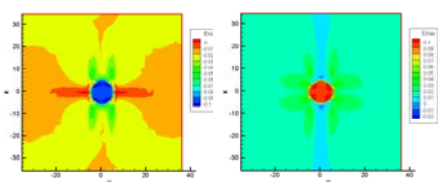

Fig. 2: Principal normal strains from FEA.

DISCUSSION & CONCLUSION: Experimental and FEA results demonstrate that intracellular strain magnitudes are considerably larger than the applied strain on the ECM, with a strain amplification factor of ~6. The patterns of strain distribution in the finite element results show a reasonable qualitative agreement with experimental results. This localized response under dynamic loading represents a novel finding relative to earlier studies of chondrocyte height and volume change under static equilibrium[3]. They suggest that mechanotransduction may be significantly mediated by this amplification mechanism.

REFERENCES: [1] F Guilak, VC Mow (2000) J Biomech 33, 1663-1673. [2]VC Mow et al (1980) J Biomech Eng 102, 73-84. [3]F Guilak, A Ratcliffe, VC Mow (1995) J Orthop Res 13, 410-421.

ACKNOWLEDGEMENTS: NIH AR46532.