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MECHANOSENSITIVE TRANSCRIPTION, METABOLISM, AND SIGNALING CASCADES IN JUVENILE AND OSTEOARTHRITIC CHONDROCYTES

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Introduction: Osteoarthritis (OA) is a common degenerative joint disorder characterized by cartilage breakdown, inflammation, and altered joint function. Investigating the cellular and molecular mechanisms underlying OA is essential for identifying the processes driving disease progression and for developing targeted therapeutic strategies. This study compares juvenile and OA chondrocytes in gene expression, metabolism, and kinase activity, and tests mechanical stimulation to better understand cartilage health and degeneration. Juvenile (jCH) and OA (pCH-OA) primary chondrocytes were mechanically stimulated using the Flexcell™ FX5K system. Gene expression, protein phosphorylation, and metabolism were analyzed pre- and post-stimulation. Principal component analysis and effect size analyses identified molecular and signaling differences. Gene expression revealed significant differences between jCH and pCH-OA, with COL1 and RUNX2 upregulated in jCH, and MMP3 and ACAN downregulated. PCA revealed distinct expression patterns and marker correlations. Cyclic tensile strain affected biomarkers such as RUNX2, IL8, TLR4, BMP2, and MMP1 in a cell type-specific manner. Metabolic profiling indicated lower ROS and NAD+/NADH, and higher glutamate, lactate, and formate, with changes primarily driven by mechanical stimulation rather than cell type. Protein analysis showed altered AKT, STAT3, and MAPK phosphorylation, reflecting different mechanotransduction in healthy versus OA chondrocytes. Juvenile and OA chondrocytes show distinct molecular, metabolic, and signaling profiles, with mechanical stimulation driving key biomarker and metabolic changes. These differences highlight altered mechanotransduction in OA, providing insights into cartilage degeneration and potential therapeutic targets.

Keywords: *Mechanosensitive transcription, metabolism, signaling cascades, in juvenile and osteoarthritic chondrocytes.*