Study Design: In vitro

Objective: To examine the effects of various magnitudes and durations of compression on nucleus pulposus inflammatory, catabolic, and anabolic gene expression.

Summary of Background Data: It is clear that mechanical forces are involved in initiating disc degeneration, but also have the potential to exert beneficial effects. However, the signaling pathways initiated by mechanical stress and thresholds for these responses have not been elucidated. We have developed a metabolically active compression system with the advantages of having the ability to test cells in vitro as well as within their native matrix and control exposure to environmental factors. We hypothesized that nucleus pulposus cells would respond to compressive stress with different thresholds for alterations in catabolic and anabolic gene expression.

Methods: A chamber capable of imparting 0-20 MPa of hydrostatic compression onto nucleus pulposus cells was fabricated. Healthy rabbit nucleus pulposus cells were cultured in alginate beads and exposed to static compression at 0.7, 2, and 4 MPa for 4 or 24 hours. Gene expression analysis (real time PCR) was performed to compare markers of inflammation (iNOS, COX-2), matrix catabolism (MMP-3), anti-catabolic/anabolic metabolism (TIMP-1, aggrecan) in control and compressed cells.

Results: Cell viability after exposure to compressive stress up to 4 MPa was 80%, equivalent to that of control. Compression resulted in magnitude and duration dependent changes in gene expression. Increasing magnitudes showed more beneficial gene expression changes, while increasing duration resulted in gene expression changes consistent with traumatic effects.

Conclusion: These data demonstrate favorable effects of physiologic compression in relation to matrix homeostasis, and detrimental effects of traumatic loading levels. These data provide an improved understanding of how compression affects cell metabolism, which has the potential to be exploited to initiate repair and prevent matrix breakdown.