

Stimulation of gene expression and loss of anular architecture caused by experimental disc degeneration--an in vivo animal study

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STUDY DESIGN: An external compression model was used to evaluate gene and protein expression in intervertebral discs with moderate disc degeneration. **OBJECTIVE:** To determine messenger ribonucleic acid and protein expression levels of relevant disc components. **SUMMARY OF BACKGROUND DATA:** An animal model of mechanically induced disc degeneration was developed and characterized histologically. However, little is known at the molecular level in moderate disc degeneration. **METHODS:** There were 8 New Zealand white rabbits subjected to monosegmental posterior compression to induce moderate disc degeneration. Twelve animals served as controls or sham controls. Discs were analyzed using immunohistochemistry for collagen type 1 (COL1), COL2, aggrecan, and bone morphogenetic protein-2/4 (BMP-2/4). For gene analysis, conventional and quantitative polymerase chain reactions were used for COL1A2, COL2A1, aggrecan, BMP-2, biglycan, decorin, osteonectin, fibromodulin, fibronectin, matrix metalloproteinase-13 (MMP-13), and tissue inhibitor of MMP-1. Gene expression for nontreated, sham-treated, and compressed discs was quantified in relation to the housekeeping gene glyceraldehyde-3-phosphate dehydrogenase. **RESULTS:** Immunohistochemistry of compressed discs showed a loss of anular architecture, and a significant reduction of BMP-2/4 and COL2 positive cells. Gene expression analysis showed a significant up-regulation of COL1A2, osteonectin, decorin, fibronectin, tissue inhibitor of MMP-1, BMP-2, and MMP-13 in compressed discs. **CONCLUSIONS:** Experimental moderate disc degeneration is characterized by a loss of BMP-2/4 and COL2 positive cells, although gene expression of disc constituents, catabolic enzymes, and growth factors is stimulated to reestablish disc integrity.

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