Anti-Apoptotic Effects of IGF-1 and PDGF on Human Intervertebral Disc Cells In Vitro
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Previous studies have shown that there is a small cell population in the human aging intervertebral disc. Earlier work from our laboratory suggested that apoptosis (programmed cell death) may be a major contributing factor to the decline in cell number. A wide variety of inhibitors of apoptosis have now been identified; the present report presents our findings on the actions of IGF-1 and PDGF in retarding or preventing apoptosis. The objective of this study was to determine whether two selected cytokines, insulin-like growth factor-1 (IGF-1) and platelet derived growth factor (PDGF), were effective in decreasing apoptosis in human cells from the annulus grown in culture for ten days. Human cells from the annulus were grown in tissue culture in an experimental design to study the anti-apoptotic effect of two selected cytokines. Positive and negative controls for apoptosis were included in the experimental tests. Experimental studies were approved by the authors’ human subjects institutional review board. Results from this work demonstrated a significant reduction in the percentage of apoptotic disc cells following exposure to 50 and 500 ng/ml IGF-1 or exposure 100 ng/ml PDGF. These findings expand our understanding of the cell biology of the disc cell and show that selected cytokines can retard or prevent programmed cell death in vitro. This type of research may have future therapeutic potential in the treatment of disc degeneration.

This technical report presented methods for in situ detection of apoptotic cells in vitro. Technical tips on procedures and useful laboratory techniques were presented. These methods were developed during our studies of programmed cell death in disc cells.

Gap junctions mediate cell-cell communication by allowing passage of small molecules (<1 kD in size) from one cell to another. Findings in this research provide evidence for gap junction formation and connexin (Cx) 43 and 45 gene expression in human intervertebral disc cells in vivo and in vitro. These findings in cells from the annulus are important in conjunction with the well recognized loss of disc cells during aging and disc degeneration. As a result of this loss of cells, cell-cell communication, which we propose is an important, but as yet poorly understood mechanism which links and coordinates cellular function throughout the entire population of disc cells, is also disrupted. These studies provide additional information on the fundamental cell biology of the disc cell and provide an additional framework for understanding aging, degeneration and potential repair of the human disc.