



The aging spine: the role of inflammatory mediators in intervertebral disc degeneration

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Human intervertebral disc undergoes multifactorial biochemical and morphologic degenerative changes during the process of aging. The frequency of degeneration, especially lumbar degeneration increases sharply with age and is regarded as a major cause of discogenic low back pain. Since degenerative discs are often asymptomatic, the pathobiology of discogenic back pain remains unclear. Degenerated discs spontaneously produce increased amounts of inflammatory mediators suggesting their role in the degenerative process of the intervertebral disc. However, the relationship between aging, degenerative processes, and actual illness is far from clear. Basic science research has demonstrated that the intervertebral disc is an avascular tissue element occupied by inadequately characterized cells in an extensive extracellular matrix. While the annulus fibrosus is predominantly collagenous, the matrix of the central nucleus pulposus is rich in proteoglycans. With aging, the substance of proteoglycans significantly decreases which is believed to be a critical factor in intervertebral disc degeneration. A variety of inflammatory mediators have been implicated in the degeneration of the intervertebral disc including nitric oxide (NO), interleukins, matrix metalloproteinases (MMP), prostaglandin E2 (PGE2), tumor necrosis factor alpha (TNF-alpha) and a group of cytokines. MMPs, PGE2, and a variety of cytokines have been already been shown to play a role in the degradation of articular cartilage. Nitric oxide is a novel mediator that is drawn into much attention recently for its role in disc abnormalities. Elevated nitric oxide production derived from NO synthase activity has been manifested in cerebrospinal fluid in patients with degenerative lumbar disease. However, the regulatory mechanism of NO and its relationship to the clinical manifestations are unclear. The biochemical events that occur with the 'aging spine' and in particular, the role of inflammatory mediators in intervertebral disc degeneration have not been studied assertively. Correspondingly, the association between degeneration of the intervertebral disc and the nociceptive mechanism of back pain is also not fully elucidated. However, there is high incidence of degenerated disc disorders manifested as back and neck pain and are among the most commonly encountered complaints in elderly population. It is hypothesized that the degenerative cascade ultimately leads to extensive structural defects and loss of normal motion segment function and configuration.

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