Effects of Link Protein N-terminal Peptide and Antioxidative Fullerol on Intervertebral Disk Degeneration

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Intervertebral disc degeneration (IVD) is a major cause of chronic lower back pain and disability that results in considerable social and medical costs. The development of IVD is a complex process which is poorly understood, but is known to be associated with proteolytic degradation of proteoglycan aggregates and reactive oxygen species (ROS). Link N peptide (LN) is the N-terminal peptide of link protein, which stabilizes the proteoglycan aggregates. Fullerol (a polyhydroxylated derivate of fullerene) has been shown to decrease the level of ROS and potentially enhancing antioxidative enzyme gene expression. In the present study, we investigated the separate and combine effects of LN and fullerol on rabbit primary fibrosus cells treated with or without IL-1B, which induced matrix degradation, oxidative stress and inflammatory cytokines production. Our data revealed that LN and fullerol combined promotes disc matrix production, which is evidenced by increased expression of extracellular matrix macromolecules collagen II and aggrecan. Also, it showed the significance of the combined effects of LN and fullerol in terms of anti-inflammatory and anti-oxidative properties, evidenced by decreased expression of cytokine IL-6 and increased expression of catalase and SOD2, known markers seen in cellular inflammatory responses. These results suggest LN and fullerol have may have a beneficial effect on disc degeneration and shed light on their potential clinical use for therapeutic agents for the treatment of lower back pain.