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ANATOMY OF NUCLEUS PULPOSUS

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INTRODUCTION

The nucleus pulposus is the soft, gelatinous central portion of the intervertebral disk. The nucleus pulposus affords disk mobility and its ability to absorb mechanical energy under compressive loading. Intervertebral disk moves within the disk with changes in posture [1].

In the normal disk, the nucleus pulposus moves anteriorly with lumbar extension and frequently communicates with the epidural space and surrounding structures. In a study of methylene blue injection into the nucleus, 14% of 105 disks showed leaks, with 93% of the leaks located in the posterolateral region lateral to the posterior longitudinal ligament (PLL). Injected dye showed contact with the adjacent root in 27% of the leaks [2].

Elastin fibers of the nucleus pulposus are oriented radially and axially, an orientation that is ideal for the restoration of a deformed disk because of bending. The ability of the nucleus pulposus to resist compression is related to the ability of its proteoglycan composition to retain water. This ability decreases with disk age and the decline of its aggrecan content [1].

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Strucure of the Nucleus Pulposus

The nucleus pulposus consists of

- Extracellular matrix
- Chondrocyte-like nucleus pulposus cells.
- Proteoglycans (comprise 50% of the dry weight of the intervertebral disk).
- Type II collagen (comprising 25% of its dry weight)
- Collagen types III, V, VI, IX, and XI
- Chondrocyte-like nucleus pulposus cells with a round cytoplasm occupy the matrix of the nucleus pulposus in the adult.
- Large, vacuolated “notochordal cells” are a second cell population within the nucleus pulposus that are more abundant in youth and sparse in adult intervertebral disks [3].

The nucleus pulposus is an avascular tissue, relying upon diffusion to provide oxygen and nutrients and carry away waste products of metabolism, and it also lacks innervation. It is similar to articular hyaline cartilage in both of these features.

Pathogensis of Intervertebral Disc (IVD) Degeneration

Intervertebral disc (IVD) degeneration (IDD) is a widely known contributor to low back pain (LBP) which is one of the most prevalent musculoskeletal disorders

worldwide and results in a massive socioeconomic burden [4–7]. Degenerative discs show structural failure that is characterized by disc height collapse, annulus fibrosus (AF) fissures, loss of proteoglycans (PGs) and water in nucleus pulposus (NP), and cartilage endplate (CEP) calcification. IDD is a multifactorial disorder. Its etiological factors include aging, smoking, infection, abnormal mechanical stress, diabetes, trauma, and genetic predisposition [8–14]. The pathogenesis of IDD involves a complex signaling network and various effector molecules [15, 16]. However, the understanding of IDD pathogenesis is limited. Elucidating the molecular mechanism of IDD in detail will contribute to developing new measures for the prevention and treatment of IDD.

Recent studies have reported that the establishment and progression of IDD are tightly associated with reactive oxygen species (ROS) and oxidative stress [17–19]. Although the roles of ROS and oxidative stress in various diseases have been widely investigated, including cardiovascular diseases, diabetes, and osteoarthritis [20–22], little attention has been paid to the effect of oxidative stress on the structure and function of IVDs until now.

Accelerated cellular senescence causes fundamental changes in the ability of disc cells to maintain the intervertebral disc (IVD) matrix, thus leading to IVD degeneration. Cells isolated from non-degenerate and degenerate human tissue were assessed for mean telomere length, senescence-associated β-galactosidase (SA-β-gal), and replicative potential. Expression of P16INK4A(increased in cellular senescence) was also investigated in IVD tissue by means of immunohistochemistry. RNA from tissue and cultured cells was used for real-time polymerase chain reaction analysis for matrix metalloproteinase-13, ADAMTS 5 (a disintegrin and metalloprotease with thrombospondin motifs 5), and P16INK4A. Mean telomere length decreased with age in cells from non-degenerate tissue and also decreased with progressive stages of degeneration. In non-degenerate discs, there was an age-related increase in the cellular expression of P16INK4A. Cells from degenerate discs (even from young patients) exhibited increased expression of P16INK4A, increased SA-β-gal staining, and a decrease in replicative potential. Importantly, there was a positive correlation between P16INK4A and matrix-degrading enzyme gene expression. Our findings indicate that disc cell senescence occurs in vivo and is accelerated in IVD degeneration. Furthermore, the senescent phenotype is associated with increased catabolism, implicating cellular senescence in the pathogenesis of IVD degeneration [23].

Cellular processes that lead to a reduction in fully functional cells and altered cellular activity include apoptosis and cellular senescence. Although apoptosis has been reported in age-related IVD degeneration, with higher rates of apoptosis present in older individuals [24], no studies, to date, have comprehensively investigated cellular senescence in ageing or degenerate IVDs. The accumulation of senescent cells in vivo with age, together with their changed pattern of gene expression [25], implicates cellular senescence in ageing and age-related pathologies. Indeed, Roberts and colleagues [26] and Gruber and colleagues [27] have shown increased staining for senescence-associated β-galactosidase (SA-β-gal) in cells from herniated discs and degenerate discs, respectively. Based on this one biomarker of senescence, they postulate that cellular senescence may be

involved in the pathogenesis of disc degeneration. Similarly, the involvement of cellular senescence has been linked to osteoarthritis, and investigators have shown that chondrocytes in articular cartilage from older individuals and osteoarthritic cartilage display a senescent phenotype (as assessed by several markers) that correlates with changes in matrix homeostasis, leading to matrix destruction [28, 29]. However, to date, no such studies correlating senescence and altered cell function have been conducted in cells from degenerate IVD tissue.

Here, we hypothesise that cellular senescence (assessed by mean telomere length [MTL], SA-β-gal staining, p16INK4Aexpression, and cell growth kinetics) occurs at an accelerated rate in IVD degeneration and that, importantly, the senescent phenotype is related to altered disc cell function associated with the characteristic features of IVD degeneration.

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