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Editorial

The Effect of the Endplate Cartilage in the Degeneration of Intervertebral Disc

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Editorial

Intervertebral Disc (IVD) degeneration is a major cause of low back pain, a common disease which affects about 80% of the population worldwide and to be considered as the first step of spinal degenerative disease [1,2]. The endplate cartilage plays an important role in the maintenance of the nutritional supply and mechanical properties [3]. Researchers have shown that endplate nutritional disorders directly lead to IVD degeneration, and obvious degeneration of endplate cartilage can be seen before the IVD degeneration [4]. It can provide new theoretical basis for the early prevention and treatment of IVD degeneration to study endplate cartilage degeneration which are treated as the initiating factor of IVD degeneration.

In our previous study, we constructed pcDNA3.1-VEGFl65 plamid and injected into the degenerated IVD based on the phenomenon that the Vascular Endothelial Cell Growth Factor (VRGF) and its Receptor (VEGFR) expression were significantly decreased in the degenerative endplate cartilage, and found it could remitted the IVD degeneration in terms of the immuohistochemical staining results. What's more, it can be observed that the nucleus pulposus and annulus fibrosus structure and function were also returned to almost normal by scanning electron microscope test. This method could provide a new way of thinking for the prevention and treatment of IVD degeneration [5].

As we know, the endplate cartilage is not only the gateway of nutrient transport between the IVD and vertebral marrow but also a mechanical interface between vertebral and resilient discs [6]. End plate chondrocytes are subjected to significant mechanical loads, such as cyclic tensile strain during physiological movement of the spine. In the recent years, we focused on the effect of the tension stress on the endplate cartilage degeneration. We applied Intermittent Cyclic Mechanical Tension (ICMT) to endplate chondrocytes by using an FX-4000T Flexer cell Tension Plus unit. Our results showed that there was no change in viability after the application of ICMT, but it could induce the down regulation of ankh gene expression of endplate chondrocytes. While the ankh gene expression could significantly be improved when treated with cell growth factor TGF-β1 which suggested that we can use the corresponding cytokines to treat IVD degeneration [7].

To the best of our knowledge, autophagy can be observed in diverse models and tissues facing mechanical injury. Palpable calcification of endplate chondrocytes were detected after ICMT treatment in our previous study. We investigated the relationship between autophagy and ICMT mediated calcification. It directly showed the ICMT induced calcification was partially resisted by increased autophagy activity, implicating that autophagy may prevent end plate chondrocyte calcification [8].

Furthermore, we investigated the molecular mechanism of ICMT-induced endplate cartilage degeneration and explored the regulatory role of the E-cadherin/β-catenin complex in the process.

Inhibition of Wnt/β-catenin signaling suppressed the decrease in ICMT-induced cartilaginous gene expression. E-cadherin expression was inhibited by ICMT stimulation, resulting in a decrease in the interaction between E-cadherin and β-catenin proteins. Overexpression of E-cadherin rescued the cartilaginous gene expression by enhancing the interaction between E-cadherin and β-catenin proteins. These findings will be useful as the basis for future studies exploring the Wnt/β-catenin signaling pathway and E-cadherin/β-catenin complex as potential therapeutic targets to prevent IVD degeneration [9].

In addition to the above work, we also established different kinds of models to explore the IVD degeneration process. They were the natural degeneration model of rat endplate chondrocyte, the natural degeneration model of human cervical endplate chondrocyte and *in vitro* organ culture model of rabbit IVD under cyclic mechanic pressure [10-12]. All these models may provide a basic condition for studying the mechanism of IVD degeneration.

Tissue engineering has been gained more and more attention and regarded as a promising approach for IVD regeneration. Except for the above studies, we have made some explorations in the field of Annulus Fibrosus (AF) tissue engineering at the same time. We had isolated and identified Annulus Fibrosus-Derived Stem Cells (AFSCs) of rabbits with self-renewing capability and multi-differentiation potential [13]. In addition, we constructed the aligned electro spinning fiber scaffolds to simulate the actural outer annulus fibrosus structure [14]. As for the selection of substrate for AF tissue engineering, we fabricated genipin cross-linking of Decellularized Annulus Fibrosus Matrix (DAFM)/chitosan hydrogels and made the biocompatibility experiment study of rabbit AFSCs with the hydrogels. The results showed the hydrogels had good biocompatibility with AFSCs *in vitro* [15].

In conclusion, we have found a number of regulatory factors in the process of IVD degeneration and made some explorations of AF tissue engineering. In the future, we will combine endplate cartilage, nucleus pulposus and annulus fibrosus into an engineered IVD and regard the endplate cartilage cells metabolism as the breakthrough point in order to improve the engineered IVD graft survival rate

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in vivo by improving nutritional pathway. In summary, we try to develop engineered IVD which could mimic the actual IVD in terms of micro structure, biochemical composition and mechanical property and provide theoretical and practical basis for the treatment of IVD degeneration.

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