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Cell Signaling Pathways Related to Pain Receptors in the Degenerated Disk

Many of the causes of low back pain are still unknown; sufficient evidence indicates that both degenerative and mechanical change within the intervertebral disk (IVD) is a relevant factor. This article reviews intracellular signaling pathways related to pain receptors in the degenerated IVD. Several reports have demonstrated the number of nerve fibers in the IVD was increased in degenerated disks. In recent years, some groups have reported that an increase in nerve fibers is associated with the presence of inflammatory mediators and/or neurotrophins in the IVD. Cell signaling events, which are regulated by inflammatory mediators and neurotrophins, must be identified to clarify the mechanism underlying low back pain. Major intracellular signaling pathways (nuclear factor kappa β, mitogen-activated protein kinases, and Wnts) potentially play vital roles in mediating the molecular events responsible for the initiation and progression of IVD degeneration. These signaling pathways may represent therapeutic targets for the treatment of IVD degeneration and its associated back pain.

退化椎間盤內與疼痛受體相關的細胞信號通路

下腰痛的很多原因都還是個未知數; 足夠的證據顯示，在椎間盤（IVD）的退變和機械性變動是相關的因素。本文綜述了在退化的椎間盤與疼痛受體有關的細胞內信號通路，。一些報告表明，在退化的椎間盤內，神經纖維的數量在 IVD 增加了。近年來，一些研究指出，神經纖維增加與 IVD 內炎症介質和/或神經營養素的存在有關。受炎症介質和神經營養素所控制的細胞信號事宜必須確定以闡明引致下腰痛的運行機制。主要的細胞內信號通路（核因子 kβ ，絲裂原活化蛋白激酶和 Wnts ）在調解與負責椎間盤退變性起始和發展的分子事宜有著至關重要的作用。這些信號通路可能是代表治療 IVD 退變性和相關的背痛的治療目標。