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**:** Discogenic low back pain; Intervertebral disc degeneration; In ammation; Oxidative stress; Matrix degradation; Neuronal remodelling; Molecular mechanisms; Musculoskeletal disorders

Low Back Pain (LBP) is one of the leading causes of disability worldwide, with Discogenic Low Back Pain (DLBP) accounting for a signi cant proportion of cases. DLBP originates from degeneration of the Intervertebral Disc (IVD), the brocartilage structure situated between vertebrae that act as a shock absorber. e condition is o en characterized by persistent pain and discomfort, and it has become a major contributor to healthcare costs due to its chronic nature and complex pathophysiology. Recent advances in molecular biology and genetics have provided deeper insights into the cellular and biochemical changes underlying disc degeneration and pain. While mechanical stress and aging have been long recognized as key factors contributing to disc degeneration, the molecular mechanisms that drive DLBP remain an area of active research. is review aims to summarize the current understanding of these mechanisms, focusing on the role of in ammation, oxidative stress, extracellular matrix (ECM) degradation, and neuronal changes within the IVD [1,2].

e intervertebral disc is composed of three primary regions: the annulus brosus (AF), nucleus pulposus (NP), and the cartilaginous endplate. e NP acts as a gel-like core, providing compressive resistance, while the AF surrounds the NP in concentric lamellae, o ering tensile strength. Healthy discs have a highly hydrated structure and a rich extracellular matrix (ECM) composed of proteoglycans, collagen bers, and other molecules that allow for load-bearing and cushioning functions [3].

e degenerative process of the IVD is initiated by a variety of factors, including aging, mechanical stress, and genetic predisposition. Over time, the disc loses water content, leading to a reduction in its elasticity and a decline in its ability to resist mechanical forces. e molecular events that drive this degeneration are complex, involving changes in the ECM composition, cellular apoptosis, and in ammation.

**:** e ECM is central to disc function, and its degradation is one of the earliest signs of disc degeneration. Key enzymes involved in ECM degradation include matrix metalloproteinases (MMPs) and aggrecanases, which break down collagen and proteoglycans, respectively. Excessive activity of these enzymes leads to the loss of disc integrity and contributes to disc instability [4,5].

**:** In ammatory processes play a pivotal role in DLBP. Activated disc cells, particularly in the NP and AF, release pro-in ammatory cytokines such as interleukins (IL-1β, IL-6) and tumor necrosis factor-alpha (TNF-α). ese cytokines promote matrix degradation and further contribute to pain by sensitizing nociceptive neurons. Additionally, immune cells in ltrate the degenerated disc, amplifying the in ammatory response and creating a vicious cycle that exacerbates degeneration.

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related pathways that may predispose individuals to disc degeneration. Additionally, epigenetic changes, such as DNA methylation and histone modi cations, can alter gene expression in response to environmental