

Effects of Melatonin upon Vascularity of Cartilage End Plate of Intervertebral Disc

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Editorial

Anatomically, intervertebral disc (IVD) consists of three parts: nucleus pulposus, annulus fibrosus and cartilage end plate (CEP) at the cranial and caudal vertebral interface of the disc [1,2]. In both animals and humans, the IVD consists largely of extracellular matrix with a low cell density. In many respects, that degeneration and age-related changes are characterised by biochemical and structural changes components of the IVD [1,3].

On the other hand, melatonin (MEL) is a secretory product synthesized by the pineal gland. It is formulated with N-acetyl-5-methoxytryptamine. MEL has a scavenger effect on hydroxyl and peroxy molecules and may have anti-ageing properties due to its antioxidant nature [4,5]. During the last decade, some authors suggested that pineal neurohormone MEL has various effects upon some bone markers and osteoblast differentiation in relation to aging [6-8]. Nevertheless, the mechanism of the effect of MEL on trabecular width, ligament thickness and degenerated IVD tissue are not clear to date [6]. Roth et al. [7] reported that MEL, applied in micromolar concentrations, was a mitogen for bone tissues. It has been suggested that MEL promotes osteoblast differentiation and matrix mineralisation via transmembrane receptors and regulates bone remodelling [7,8]. Turgut et al. [9] reported that MEL was applied as a treatment to the created degenerative effects of surgery on CEP and exogenous MEL significantly increased vascularization on trabecular reduction in width, suggesting its regenerative effects in IVD tissue degeneration.

Consequently, it is certain that further experiments and randomized controlled clinical studies should be done for standardization of the clinical use of MEL upon the vascularity of CEP of IVD. However, we

strongly hope that future search will confirm the useful effects of novel drug MEL treatment as a biological anti-ageing agent to treating IVD degeneration.

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