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Editorial

Osteocyte Dysfunction and Osteoarthritis in Joint Homeostasis

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EDITORIAL

The most common type of arthritic disease, Osteoarthritis (OA), affects the load-bearing joints, such as the knee and hip. It is also recognised as a major source of joint discomfort and dysfunction in older persons, contributing to a lower quality of life. Articular cartilage, subchondral bone, and synovium are just a few of the tissues that can be affected by OA. Sclerosis, cyst, and osteophyte formation on plain x-ray, as well as Bone Marrow Lesions (BMLs) on magnetic resonance imaging, are all radiological characteristics of osteoarthritic subchondral bone that have been proved to highlight the anomalies of bone mineralization. Increased bone turnover with an increase in osteoblastic over osteoclastic activities is thought to cause sclerosis and the production of osteophytes. Sclerostin, periostin, and Dentin Matrix Protein 1 (DMP-1) signalling abnormalities are thought to be linked to BMLs and sclerosis. Meanwhile, cysts surrounding by less mineralized bone and osteoid development uncoupled from mineralization could indicate that Wnt/catenin signalling and the OPG/RANKL/RANK pathway differentially regulate osteoblasts and osteoclasts geographically.

To maintain the equilibrium of adult bone metabolism, bone cells strictly govern and accurately coordinate resorption and creation during the bone remodelling process. Primary osteoblastic dysfunction is thought to be the cause of morphological and functional changes in osteoarthritic bones. Osteoarthritic osteoblasts had attenuated responses to cytokine stimulation, as well as an abnormal mineral-to-collagen ratio. Meanwhile, OA is thought to be caused by osteocyte malfunction and aberrant osteoblast-osteocyte differentiation. Mechano-transduction is carried out by osteoocytes, which make up 90-95 percent of bone cells. Osteocytes have been reported to die in response to mechanical stimuli like as microcracks. After osteocytes die, bone remodelling begins with the activation of osteoclastogenesis and bone resorption. Sclerostin production by osteoocytes also acts as a critical regulator of osteoblast differentiation. Sclerostin inhibits osteoblastic activity and stops the cycle of bone remodelling by inhibiting Wnt signalling. There is mounting evidence that osteocytes play a key role in maintaining bone homeostasis and integrity by acting as a central regulator of bone remodelling.

The importance of subchondral bone in the evolution of OA is well known. Anomaly bone shape at the tissue level, irregular osteocyte activities at the cellular level, and changed protein expressions at the molecular level describe subchondral bone disturbances. In osteoarthritic subchondral bones, the Wnt/-catenin pathway, which is important for osteogenesis and bone remodelling, is abundant. Want inhibitor sclerostin is expressed in calcified cartilage and subchondral bone. Its absence is frequently linked to the development of OA, most likely due to the activation of Wnt signalling. Sclerotic bones with reduced mineralization characterise subchondral Bone Marrow Lesions (BMLs), which are thought to predict the course of OA. The inorganic concentration of bone plugs from knee OA specimens was likewise much reduced, according to our findings. Osteoid development around trabeculae or in the marrow space of OA bone was also detected, which was consistent with BMLs. DMP-1 is most likely linked to abnormal mineralization. As it is required for mineral nucleation, DMP-1 is responsible for the mineralization of collagen content.

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