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Homeostasis of the Cartilage and Osteoarthritis

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Introduction

In terms of arthritis, osteoarthritis (OA) is the most prevalent kind. Osteophyte production, subchondral bone sclerosis, articular cartilage degradation over time and synovial membrane inflammation are its hallmarks. In the articular cartilage, OA is linked to an aging-related deterioration of the homeostatic balance between processes for degradation and repair. By switching the expression of genes and/or proteins from having anabolic to catabolic effects, this dysregulation causes senescence, differentiation, proliferation and death in joint cells. The development of OA depends heavily on cartilage-degrading enzymes such matrix metalloproteinase (MMP)-13, disintegrin and metalloproteinases with thrombospondin motifs (ADAMTS)-4 and ADA [1,2].

Description

The extracellular matrix, which is mostly made up of type II collagen and proteoglycans like aggrecan and chondrocytes make up cartilage. The extracellular matrix (ECM), which is full of type II collagen, proteoglycans and related macromolecules, is one way that chondrocytes in articular cartilage manage cartilage homeostasis. New avenues for osteoarthritis research using mass spectrometry-based proteomic techniques have emerged with the development of newer high throughput technologies. Global proteomics investigations utilizing mass spectrometry have been widely carried out in recent years to clarify the pathophysiology of articular cartilage. However, proteome investigations employing cartilage tissue, cartilage explants, OA synovial fluid and synovial cells have also been carried out. The majority of researches have concentrated on proteins found directly in the secretome of cartilage cell cultures.

Due to its hypo vascularity, mature articular cartilage has a limited capacity for regeneration and repair after injury. When chondrocytes undergo catabolic and aberrant differentiation as a result of ageing or excessive cartilage usage, cartilage ECM is lost, which results in OA. By reacting at the molecular level to numerous physiological stimuli, including mechanical stress, articular cartilage maintains homeostasis. Osteoarthritis begins to form and worsen when this equilibrium is upset. A significant risk factor for OA is ageing and aged chondrocytes display a variety of senescent characteristics. Due to dysfunctional autophagy, aged chondrocytes in particular have decreased tolerance to oxidative stress and altered cellular homeostasis. Another risk factor for OA is synovitis, a frequent degenerative alteration in osteoarthritic joints that aggravates cartilage loss.

Damaged joint tissue releases proinflammatory substances including interleukin-6 and tumour necrosis factor, which cause inflammation and synovial growth, both of which are known to contribute to synovitis. However, the knee, hand, hip and spine are the most often impacted joints by OA. Joint discomfort and impairment may result from the gradual and permanent degradation of the cartilage matrix, which lowers quality of life. While joint replacement surgery is frequently necessary in the late stages of the illness, exercise therapy and symptomatic therapy, such as pharmacologic therapy, are the only known therapies for early-stage.

All of the vertebrate species depend on cartilage and Sox9 is crucial for cartilage growth. Acan is a significant ECM protein that is found in the growth plate and articular cartilage and it has been shown to express itself in all articular cartilages throughout development and beyond Haseeb et al. created a line mice that are cartilage-specific Sox9 conditional knockout mice that, when given a tamoxifen injection, delete Sox9 in order to study the function of Sox9 postnatally. Proteoglycans and hypertrophic zones were lost in 3-month-old Sox9-deleted mutant mice [3,4].

This mutant miRNA gene also produced a lot of mutant miR-140-5p expression without any issues with miRNA processing. The mutation significantly reduced the targets of WT miR-140-5p and repressed those of mutant miR-140-5p in chondrocytes, indicating that the mutation has both gain- and loss-of-function consequences. Furthermore, Ybx1, a conserved RNA-binding protein and mutant miR-140-5p fight for an overlapping binding site [5].

Conclusion

In addition to providing a summary of earlier studies, this review focuses on Sox9, the cartilage's master transcription factor and ncRNAs, primarily miRNAs. Numerous mechanisms that contribute to the preservation of cartilage homeostasis and OA have been identified to far, but our knowledge of how these factors interact and are connected to one another is still lacking.

Conflict of Interest

None.

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