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Metabolism of Osteoarthritic Bone Cartilage

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Opinion Article

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DESCRIPTION

Osteoarthritis (OA) is a term used to describe a group of mechanically-induced joint disorders that are caused by both genetic and acquired factors. The current pathophysiological paradigm views OA as a disease that affects the entire joint. The functional unit formed by the articular cartilage and the subchondral bone appears to be of particular interest in these models. Cartilage and bone are constantly challenged biomechanically as they receive and dissipate the stress associated with movement and loading. Recent evidence suggests that cartilage and bone can communicate across the calcified tissue barrier; vessels reach out from bone into the cartilage zone, patches of unclarified cartilage come into contact with bone, and micro cracks and fissures aid in molecule transfer.

The most common type of articular disease is osteoarthritis (OA). An imbalance between the synthesis and degradation of articular cartilage and subchondral bone, as well as capsular fibrosis, osteophyte formation, and varying degrees of synovial membrane inflammation, characterise this idiopathic joint disease. Hyaluronic acid in the syovial fluid provides natural joint lubrication. In normal, young, and healthy joints, hyaluronan is abundant. Hyaluronan is smaller in size, has a lower molecular weight, and has a lower concentration in degenerative O.A. Intraarticular viscosupplementation can help to restore joint lubrication and shock absorption in people with osteoarthritis. Alternatively, intraarticular injections of steroids and long-acting local anaesthetics can relieve pain and secondary inflammation in OA local inflammation is reduced quickly and effectively.

The physiological turnover of synovial joint tissues, including articular cartilage, is aided by metabolism. Chondrocytes and cells in joint tissues other than cartilage undergo metabolic changes in osteoarthritis (OA), shifting from a resting regulatory state to a highly metabolically active state. Inflammatory mediators, metabolic intermediates, and immune cells all play a role in the pathophysiology of OA, influencing cellular responses. Future treatments for OA could target key metabolic pathways and mediators.

OA is a degenerative joint disease that is a leading cause of adult disability. OA has no cure and no effective treatment to slow or stop its progression. Current pharmacologic treatments, such as analgesics and nonsteroidal anti-inflammatory drugs, may reduce pain and provide some relief, but they have no effect on the disease's progression. The use of these drugs on a regular basis can have serious side effects.

The function of cartilage and synovial joints is dependent on metabolism. Mammalian cells switch from a resting regulatory state to a highly metabolically activated state to maintain energy homeostasis under adverse microenvironmental conditions. This phenomenon also results in an increase in metabolic intermediates for the biosynthesis of inflammatory and degradative proteins, which activates key transcription factors and inflammatory signalling pathways involved in catabolic processes, as well as the continued perpetuation of pathogenic drivers.

The matrix of the cartilage is dehydrated. Water accounts for 80% of the total weight. Water plays a critical role in joint lubrication and wear resistance. Proteoglycan and type II collagen make up the majority of the dry weight. Osteoarthritis is a very active catabolic process, not the result of a decrease in metabolic activity. Damaged cartilage has a higher rate of matrix synthesis and cell replication than normal cartilage Proteoglycan content decreases in direct proportion to the severity of the disease. Despite increased synthetic activity, the chondrocyte's capacity is eventually exceeded by the rate of matrix degradation, resulting in cartilage erosion.