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Commentary

Pathophysiology of Osteoarthritis (OA) and its Lipid Metabolism

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DESCRIPTION

Fats also known as triglycerides are produced in the body by adipocytes and hepatocytes from carbohydrate precursors or by food intake. The process of oxidizing fatty acids for either the production of energy or the creation of new lipids from smaller constituent molecules is known as lipid metabolism. Because glucose products like acetyl CoA can be converted into lipids, lipid metabolism is linked to carbohydrate metabolism. Lipid metabolism is the process by which the pancreas and small intestine secrete lipase, which hydrolyses the fatty acids in fat into free fatty acids and monoglycerides, the majority of the fat the body consumes is emulsified into small particles by bile.

Pancreatic lipases and enzymes that break down fats after they are emulsified by bile salts are where lipid metabolism begins and breaking down ingested triglycerides into smaller chain fatty acids and then monoglyceride molecules. A digestive hormone known as Cholecystokinin (CCK) is released by intestinal cells in the intestinal mucosa when food reaches the small intestine in the form of chyme. CCK causes the pancreas to release pancreatic lipase and the gallbladder to contract to release stored bile salts into the intestine. Additionally, CCK makes its way to the brain, where it may suppress hunger.

Degradation of the articular cartilage is one of the effects of Osteoarthritis (OA), which is characterized as a failure of the joint organ that affects all the tissues in the joint and around the joint. The affects including, a thickening of the bone below the chondra, formation of osteophytes, varying degrees of inflammation in the synovium, weakening of the ligaments, capsule hypertrophy of the joint and modifications in the bursa, periarticular muscles, nerves, and local fat pads. Degradation of cartilage is thought to be the primary feature among these. This is due to the fact that articular cartilages are anatomically in the first line of defense when it comes to responding to the local biomechanical environment, by absorbing and dispersing mechanical loads that are placed on the articular joint, creating a low-friction system that allows for mobility. Cartilage is maintained and modified by anabolic and catabolic mechanisms that are tightly controlled. The loss of cartilage homeostasis in OA is caused by dysregulation caused by the presence of various biofactors. This leads to the degradation of the collagen- and proteoglycan-rich Extracellular Matrix (ECM), articular surface fibrillation and erosion, cell death, matrix calcification, and vascular invasion.

Due to the mechanical overload placed on the joints, osteoarthritis has long been associated with obesity. However, that OA is a metabolic disease because it affects non-weight-bearing joints as well. In point of fact, an altered metabolism of lipids might be the root cause. First, it has been demonstrated that adipokines are important regulators of OA pathogenesis. Second, serum cholesterol has been found to be a risk factor for the development of OA in epidemiological methodologies. Thirdly, before histological changes occur, lipid deposition in the joint is seen in the early stages of OA. Fourth, proteomic studies have demonstrated a significant link between lipid metabolism and OA. Finally, the expression of genes related to lipid metabolism and cholesterol influx and efflux have been found to be deregulated in recent gene expression profiling. Osteoarthritis is frequently referred to as a disease of wear and tear. However, osteoarthritis affects the entire joint in addition to the breakdown of cartilage. It alters the structure of the bone as well as the deterioration of the connective tissues that keep the joint in place and link muscle to bone.

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