Opinion

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Alteration of Lipid Metabolism

Wei Sun *

Department of Respiratory and Critical Care Medicine, University of Electronic Science and Technology, Chengdu, China

***Corresponding author:** Wei Sun, Department of Respiratory and Critical Care Medicine, University of Electronic Science and Technology, Chengdu, China, E-mail: doclswd@163.com

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Description

Cancer frequently disrupts lipid metabolism, which alters metabolism intermediates and contributes to their out-of-control growth and metastasis. Cancer therapy resistance is also caused by a shift in lipid metabolism that increases the amount of polyunsaturated fatty acids in membrane phospholipids. Cancer cells that have high levels of PL-PUFAs are more susceptible to lipid peroxidation, putting them at risk for Ferro ptosis, a novel form of iron-dependent oxidatively regulated cell death. The adaptive lipidome remodeling, LPO patterns and LPO scavenging capability of heterogeneous cancer cells all play a role in the commitment of cancer to ferroptotic cell death. Ferro ptosis is getting more attention in cancer research because it treats cancer, changes membrane lipid homeostasis, and doesn't work with other treatments. As a result, a deeper comprehension of the molecular mechanisms underlying changes in lipid metabolism may open up new avenues for overcoming cancer resistance. Lipid composition and metabolic processes associated with ferro ptosis induction in cancer are the focus of this review, which aims to comprehend altered lipid metabolism in cancer.

Repeat and Sustain Damage Stimulation

At five years after diagnosis, Idiopathic Pulmonary Fibrosis (IPF) has a higher mortality rate than most types of cancer due to irreversible destruction of alveolar structures and excessive extracellular matrix deposition. The alveolar epithelial cells, which are essential for maintaining alveolar structure and function, play a crucial role in the onset and progression of IPF. Alveolar epithelial type I cells (AT1) and alveolar epithelial type II cells (AT2) are the two types of AECs. Physiologically, AT2 cells can proliferate and differentiate into AT1 cells. The alveolar epithelium's normal structure and function is maintained by AT2 cells' dynamic balance of proliferation, differentiation, and apoptosis. AT2 cells undergo injury and repair under conditions of repeated and sustained damage stimulation. This results in the secretion of numerous pro-fibrotic cytokines and further induces the proliferation and differentiation of lung fibroblasts into highly active myofibroblasts capable of synthesizing the Extracellular Matrix (ECM). Alveolar structures eventually become deformed and destroyed as a result of excessive ECM deposition. As a result, AT2 cells and fibroblasts play a crucial role in controlling the progression of IPF. Under hypoxic conditions, lipid metabolism is a distinct metabolic mode of the lung that primarily makes use of fatty acid oxidation for energy. Alveolar surfactant, which is crucial to maintaining normal alveolar surface tension, is synthesized from lipids like triglycerides, phospholipids, sphingolipids, and other fatty acids, which are important components of the human body. IPF's disordered lipid metabolism not only makes it harder for AT2 cells to repair damage, but it also makes it easier for fibroblasts to become myofibroblasts. As a result, identifying key molecules that are involved in the processes of lipid metabolism could be helpful in the treatment of pulmonary fibrosis in the future. As important regulators of metabolic processes, sirtuins play a role in gluconeogenesis, lipid metabolism, and mitochondrial activity, assisting in the maintenance of cellular energy supply homeostasis. Sirtuins are multifunctional proteins that are involved in the deacetylation of both histone and non-histone lysine residues. They are members of the nicotinamide adenine dinucleotide (NAD+) dependent histone deacetylases. By deacetylating the peroxisome proliferator-activated receptor (PPAR) and the peroxisome proliferator-activated receptor-gamma coactivator-1alpha (PGC-1), sirtuins control the entire process of lipid metabolism. The significance of sirtuins in IPF has received increasing attention in recent years. The protective mechanism of sirtuins in IPF may be related to the regulation of inflammatory response, fibrosis, cellular senescence, and energy metabolism, which are anticipated to become therapeutic targets for IPF as newly discovered beneficial factors.

Regulation of Numerous Lipogenic Genes

Copper (Cu) is an essential nutrient for most organisms and is naturally found in sediments and water. Cu is also a key ingredient in an aquaculture feed additive and the primary component of the respiratory pigment hemocyanin found in crustaceans. Cu concentration in the aquatic environment rises as a result of the rapid growth of the industrial and agricultural sectors' utilization of the metal. For instance, the concentration of Cu in China's Yangtze River and Hun River exceeded the Chinese National Water Quality Standard for Fisheries by 21–153 g/L. According to Wei and Yang (2016), aquatic organisms commonly come into contact with Cu through respiration and ingestion. Cu can cause an organism's normal physiological processes to be disrupted when its concentration is slightly

higher than normal. When Cu is present in excess, aquatic organisms are known to suffer from decreased growth rate, malformations, endocrine disorders, immunosuppression, lipid metabolism problems, and even mortality. Triglycerides (TG), phospholipids, and cholesterol are the main components of lipids, which are essential substances in all animals. According to Schmitz and Ecker, lipid metabolism and inflammatory responses are regulated by polyunsaturated fatty acids (PUFAs), which make up the majority of phospholipids. Lipid homeostasis is coordinated by a large number of essential enzymes and transcription factors and involves a number of key processes, including fat absorption, fatty acid synthesis, and lipid catabolism. The multi-enzyme fatty acid synthase and acetyl-CoA carboxylase catalyze the synthesis of palmitic acid during the synthesis and metabolism of fatty acids, which are based on acetyl-CoA as a synthetic raw material. Palmitic acid (PA) is transformed into monounsaturated fatty acids by the enzyme stearoyl-CoA desaturase (SCD). According to Jensen-Urstad and Semenkovich fatty acids and glycerol are the precursors of triglycerides, which are produced during the process of fat synthesis. Carnitine palmitoyltransferase 1 (CPT1) converts long-chain acyl-CoA species to long-chain acyl-carnitines for fatty acid beta oxidation (Zimmermann et al., 2003). Hormone sensitive triglyceride lipase (HSL) is the primary enzyme that primarily hydrolyzes TAG, diacylglycerol and monoglycerol. By regulating the transcription of enzyme-encoding genes, transcription factors mediate lipid homeostasis during lipid metabolism. Sterol regulatory element binding protein 2 primarily controls the transcription of cholesterol enzymes, while sterol regulatory element binding protein 1 (SREBP1) is a transcription factor that is crucial to the regulation of numerous lipogenic genes. SREBP1 controls the genes for fatty acid synthesis in Atlantic salmon, but the function of SREBP may vary from organism to organism. The Chinese mitten crab Eriocheir sinensis, like the majority of aquatic animals, only contained one SREBP homolog (designated SREBP, unpublished). Salmon fads2d6a and elovl5a are directly affected by the SREBP gene in fish. In most cases, SREBP-1 binds to the liver X receptor to cause the expression of Fads1 and Fads2. It is necessary to clarify the role that the SREBP gene plays in the transcriptional regulation of lipid metabolism due to the evolutionary loss of the liver X receptor in some aquatic animals.