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Mini Review

Lethal Lung Cancer and Lipid Metabolism

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Abstract

One of the most important health problems today both in industrialised and developing nations, is lung cancer. Although there are numerous therapeutic options, the prognosis for survival is still very dismal. Although altered metabolism is one of the characteristics of human cancer, lipid metabolism problems are less well understood. A deeper understanding of them could be useful to develop new methods for accurate diagnosis, prognosis estimation, and the creation of therapeutic agents based on bioactive substances like cerulenin, SCD1, ACLY inhibitors, statins, polyphenolic compounds, etc (Tsankova NM 2006). The basis of lipid metabolism in lung cancer is reviewed in this research along with potential indicators. To advance our understanding in this area, more research is very necessary.

Keywords: Bioactive compounds, Biomarker, Lipid metabolism, Lung cancer

INTRODUCTION

Lipid metabolism entails the synthesis of the structural and functional lipids (such as cholesterol, prostaglandins, glycolipids, and sphingolipids) that are unique to each tissue as well as the breakdown of lipids to meet the body's metabolic needs (such as the production of energy). The metabolism of lipids is perpetually in a state of dynamic balance. This implies that while certain lipids are synthesised and stored, others are continuously oxidised to suit the body's metabolic needs. Therefore, the focus of this chapter is on the metabolic processes that the body uses to synthesise and/or breakdown the different types of lipids (Lubin FD et al., 2008). ATP-citrate lyase is necessary for transformation in vitro, cholesterol synthesis in prostate cancer is increased, and fatty acid oxidation is a significant source of energy for prostate cancer cells. Cancer cells also reactivate de novo lipid synthesis. As triglyceride hydrolysis is hindered in Atg5^{-/-} cells, autophagy, namely lipophagy, regulates lipid metabolism in hepatocytes and is crucial for the destruction of lipid droplets in adipose tissue (Jakobsson J 2008). Further research is needed to determine whether these pathways impact tumour lipid metabolism. They consist of a chain of hydrocarbons with varying lengths that ends in a carboxylic acid group (-COOH). One of the

most fundamental types of biological lipids is the fatty acid structure, which is shown in the table below. It is frequently employed as a constituent of lipids with more complex structural makeup, such phospholipids and triglycerides. Fatty acids are crucial energy sources because their metabolism produces significant amounts of ATP. Lipids are a source of both energy and carbon. Complex lipids must first be hydrolyzed in order to be utilised for energy production. The release of fatty acids from derivatives like phospholipids is accomplished by hydrolytic enzymes known as lipases (Denning DP et al., 2012).

The majority of biological compounds are lipids. In addition to being the main structural element of biological membranes in all organisms, this large class of chemicals plays a number of essential roles in microbes. Microbes can metabolise lipids to utilise as their main source of energy among them. Although it isn't stated explicitly, the "Organic Acid Metabolism" component in this module introduces the idea of lipid metabolism by outlining the steps involved in the metabolism of fatty acids by -oxidation. This atom will add to the metabolic process that allows lipids to be broken down and used. Lipids are constituted of fatty acids (Johnsen HL et al., 2016).

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in industrialised and developing nations, is lung cancer. Although there are numerous therapeutic options, the prognosis for survival is still very dismal. Although altered metabolism is one of the characteristics of human cancer, lipid metabolism problems are less well understood. A deeper understanding of them could be useful to develop new methods for accurate diagnosis, prognosis estimation, and the creation of therapeutic agents based on bioactive substances like cerulenin, SCD1, ACLY inhibitors, statins, polyphenolic compounds, etc. The basis of lipid metabolism in lung cancer is reviewed in this research along with potential indicators (Lauzon RJ et al., 2000). To advance our understanding in this area, more research is very necessary. There are two sources of fats that organisms can use to get energy: from eaten dietary fats and from stored fat. Lipid metabolism is sometimes thought of as the digestion and absorption process of dietary fat, however this is incorrect. Both types of fat are used by vertebrates (including humans) to generate energy for the operation of organs like the heart. Lipids are hydrophobic molecules, thus solubilizing them is necessary before lipid metabolism can start. Lipid metabolism frequently starts with hydrolysis, which is accomplished with the aid of several digestive system enzymes (Lauzon RJ et al., 1993). Plants also have lipid metabolism, however the processes are somewhat different from those in animals. The absorption of the fatty acids into the intestinal wall's epithelial cells comes next following the hydrolysis. Fatty acids are packed and transferred to other parts of the body in epithelial cells. Lipid digestion, lipid absorption, lipid transport, lipid storage, lipid catabolism, and lipid production are examples of metabolic processes. The beta oxidation process, which takes place in the mitochondria and peroxisome cell organelles, is responsible for lipid catabolism (Kim MY et al., 2015).

DISCUSSION

Metabolic reprogramming is one of the characteristics of cancer. To support cell proliferation, tumour cells change how they can metabolise proteins, lipids, and carbohydrates. Cancer cells demonstrate considerable metabolic changes in comparison to non-malignant cells. Cancer cells exhibit uncontrolled proliferation even in the presence of food scarcity, whereas normal cells adjust anabolic and catabolic pathways in response to variations in nutrient supply (Pálka I et al., 2015). Despite the fact that the majority of research on metabolic dysregulation in cancer focuses on carbohydrates, the significance of changes in lipid metabolism is beginning to be understood, and an increase in de novo lipogenesis is now thought to be a new hallmark in many aggressive cancers. Additionally, cancer cells exhibit elevated levels of de novo adipogenesis due to enhanced expression of several lipogenic enzymes, including FASN (fatty acid synthase) and ACLY (ATP citrate lyase). Furthermore, recent research has shown that constitutive activation of growth-promoting pathways makes cells dependent on unsaturated fatty acids (FA) to survive in an oxygen-depleted environment.

Pancreatic lipases, which break down lipids after they have been emulsified by bile salts, start the breakdown of ingested triglycerides into shorter chain fatty acids and then into monoglyceride molecules in the intestine. Cholecystokinin (CCK), a digestive hormone, is released by intestinal cells in the intestinal mucosa when food enters the small intestine as chyme. In order to release the stored bile salts into the intestine, the pancreas releases pancreatic lipase, and the gallbladder contracts in response (James JJ et al., 2003). Additionally, CCK travels to the brain, where it can reduce hunger. Triglycerides are broken down into free fatty acids by pancreatic lipases and bile salts working in tandem. The gut membrane can be crossed by these fatty acids. However, after passing through the membrane, they unite once more to create new triglyceride molecules. These triglycerides are packed with cholesterol molecules in phospholipid vesicles known as chylomicrons within the intestinal cells. The lymphatic and circulatory systems' watery environments are made possible by the movement of lipids and cholesterol by chylomicrons. Chylomicrons exit the enterocytes through exocytosis and travel to the intestinal villi's lacteals to enter the lymphatic system.

CONCLUSION

With potential biomarkers involved in lung carcinogenesis, prognosis, prevention, and treatment, the relationship between lipid metabolism and lung cancer is an intriguing one. Deepening our understanding of the underlying underpinnings of lung cancer may help us create new diagnostics and potent therapy approaches. The implications of lipid metabolism in the biology of lung cancer are the subject of the first review of the literature to be published. Hopefully, the lack of research on this subject

REFERENCES

1. Tsankova NM (2006). Sustained hippocampal chromatin regulation in a mouse model of depression and antidepressant action. *Nat Neurosci.* 9: 519-532.
2. Lubin FD, Roth TL, Sweatt (2008). Epigenetic regulation of bdnf gene transcription in the consolidation of fear memory. *J Neurosci.* 28: 10576-10586.
3. Jakobsson J (2008). KAP1-mediated epigenetic repression in the forebrain modulates behavioral vulnerability to stress. *Neuron. Behav neur.* 60: 818-831.
4. Denning DP, Hatch V, Horvitz HR (2012). Programmed elimination of cells by caspase-independent cell extrusion in *C. elegans*. *Nature.* 488: 226-230.
5. Johnsen HL, Horvitz HR (2016). Both the apoptotic suicide pathway and phagocytosis are required for a programmed cell death in *Caenorhabditis elegans*. *BMC Biol.* 14: 39-56.
6. Lauzon RJ, Rinkevich B, Patton CW, Weissman IL (2000). A morphological study of nonrandom senescence in a colonial urochordate. *Biol Bull.* 198: 367-378.
7. Lauzon RJ, Patton CW, Weissman IL (1993). A morphological and immunohistochemical study of programmed cell death in

- Botryllus schlosseri (Tunicata, Ascidiacea). Cell Tissue Res. 272: 115-127.
8. Kim MY, Kim HS, Choi N, Yang JH, Yoo YB, et al (2015). Screening mammography-detected ductal carcinoma in situ: mammographic features based on breast cancer subtypes. Clinical Imag. 39: 983–986.
 9. Pálka I, Ormándi K, Gaál S, Boda K, Kahán Z (2015). Casting-type calcifications on the mammogram suggest a higher probability of early relapse and death among high-risk breast cancer patients. Acta Onco. 46: 1178–1183.
 10. James JJ, Evans AJ, Pinder SE, Macmillan RD, Wilson ARM, et al. (2003). Is the presence of mammographic comedo calcification really a prognostic factor for small screen-detected invasive breast cancers. Clinical Radiology. 58: 54–62.