Lipid metabolism

Lipid metabolism refers to the biochemical processes involved in the synthesis, degradation, and modification of lipids, which are a diverse group of hydrophobic molecules including fats, oils, phospholipids, and steroids. These processes are essential for various physiological functions in organisms, including energy storage, membrane structure, signaling, and hormone production. Here's an overview of some key aspects of lipid metabolism:

Lipid Digestion and Absorption: Lipids in the diet are broken down into fatty acids and glycerol through the action of enzymes like lipases in the digestive system. These breakdown products are then absorbed by the small intestine and transported into the bloodstream.

Fatty Acid Synthesis: In a process called lipogenesis, excess dietary carbohydrates and proteins can be converted into fatty acids and stored as triglycerides in adipose tissue for future energy needs. This process primarily occurs in the liver.

Fatty Acid Oxidation: Fatty acids are oxidized in the mitochondria through beta-oxidation to produce energy in the form of ATP. This process involves a series of enzymatic reactions that sequentially remove two-carbon units from the fatty acid chain.

Ketogenesis: During periods of low carbohydrate availability, such as fasting or prolonged exercise, fatty acids are converted into ketone bodies (e.g., acetoacetate, beta-hydroxybutyrate) in the liver. These ketone bodies can serve as alternative energy sources for tissues like the brain.

Cholesterol Metabolism: Cholesterol is an important component of cell membranes and a precursor for steroid hormones, bile acids, and vitamin D. It can be synthesized de novo in the liver or obtained from the diet. Excess cholesterol is transported back to the liver for elimination or storage.

Lipoprotein Metabolism: Lipids are transported in the bloodstream as lipoprotein particles, including chylomicrons, very-low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). These lipoproteins play crucial roles in lipid transport, delivery, and clearance from circulation.

Regulation of Lipid Metabolism: Lipid metabolism is tightly regulated by various hormones (e.g., insulin, glucagon, leptin) and transcription factors (e.g., SREBP, PPARs) in response to physiological conditions such as nutrient availability, energy balance, and metabolic demands.

Disruptions in lipid metabolism can lead to various metabolic disorders, including obesity, dyslipidemia, insulin resistance, and cardiovascular diseases. Understanding the intricate mechanisms of lipid metabolism is crucial for developing strategies to manage and prevent these health conditions.

APOE4 is widely recognized as a genetic risk factor for Alzheimer's disease (AD), implicated in 60-80% of all AD cases. Recent research suggests that microglia carrying the APOE4 genotype display abnormal lipid metabolism and accumulate lipid droplets, which may exacerbate the pathology of AD. Microglia play a critical role in immune surveillance within the central nervous system and are responsible for removing harmful particles and preserving neuronal function. The APOE4 genotype causes abnormal lipid metabolism in microglia, resulting in excessive accumulation of lipid droplets.

This accumulation not only impairs the phagocytic and clearance capabilities of microglia but also disrupts their interactions with neurons, resulting in disorganization and neurodegenerative alterations at the neuronal network level. In addition, the presence of APOE4 modifies the metabolic landscape of microglia, particularly affecting purinergic signaling and lipid metabolism, thereby exacerbating the pathological processes of AD. In conclusion, the accumulation of lipid droplets and abnormal lipid metabolism may be critical mechanisms in the progression of AD in microglia carrying the APOE4 genotype [1)]

Metabolic reprogramming refers to the ability of cancer cells to alter their metabolism in order to support the increased energy request due to continuous growth, rapid proliferation, and other characteristics typical of neoplastic cells.

Metabolic reprogramming is a key feature of gliomas and is thought to reflect the adaptation to the increased nutritional requirements of tumor cell proliferation, growth, and survival. Mutations in the IDH gene can shape metabolic reprogramming and may generate some vulnerabilities in glioma cells, such as abnormal lipid metabolism and sensitivity to endoplasmic reticulum stress (ERS). They analyzed the prominent metabolic features of malignant gliomas and the key pathways regulating glioma metabolism [2)].

In a study, Choo et al. investigated the relationship among the three important phenotypes, namely temozolomide resistance, cell shape and lipid metabolism, in GBM cells. They first observed the distinct difference in cell shapes between TMZ-sensitive (U87) and resistant (U87R) GBM cells. They then conducted NMR-based lipid metabolomics, which revealed a significant increase in cholesterol and fatty acid synthesis as well as lower lipid unsaturation in U87R cells. Consistent with the lipid changes, U87R cells exhibited significantly lower membrane fluidity. The transcriptomic analysis demonstrated that lipid synthesis pathways through SREBP were upregulated in U87R cells, which was confirmed at the protein level. Fatostatin, an SREBP inhibitor, and other lipid pathway inhibitors (C75, TOFA) exhibited similar or more potent inhibition on U87R cells compared to sensitive U87 cells. The lower lipid unsaturation ratio, membrane fluidity and higher fatostatin sensitivity were all recapitulated in patient-derived TMZ-resistant primary cells. The observed ternary relationship among cell shape, lipid composition, and TMZ-resistance may be applicable to other drug-resistance cases. SREBP and fatostatin are suggested as a promising target-therapeutic agent pair for drug-resistant glioblastoma [3)]

Pending Classification

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