Discussion on lipids and lipoproteins.

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Abstract

Triglycerides and cholesterol cannot be transferred alone because they are soluble in proteins and cannot be delivered in water. The development and function of lipoproteins are facilitated by the complex particles known as lipoproteins, which have a central core made up of triglycerides and cholesterol esters that is surrounded by free cholesterol, phospholipids, and apolipoproteins. Based on their size, lipid makeup, and apolipoproteins, plasma lipoproteins can be categorised into seven different groups. While HDL is anti-atherogenic, chylomicron remnants, VLDL, IDL, LDL, and LP are all pro-atherogenic. Starting in the colon, food lipids are incorporated into chylomicrons to form the exogenous lipoprotein route. Lipoprotein lipase breaks down the triglycerides carried by chylomicrons in the circulation, releasing free fatty acids that are then broken down by muscle and adipose tissue and forming chylomicron remains. Remaining chylomicrons are then absorbed by the liver. The production of VLDL in the liver is the first step in the endogenous lipoprotein pathway. Lipoprotein lipase breaks down the triglycerides transported by VLDL in muscle and adipose tissue, releasing free fatty acids and resulting in the formation of IDL. The liver is the main site of absorption for the LDL receptor, which is where the IDL are primarily absorbed after being further converted to LDL.

Keywords: Pro-atherogenic, Lipoprotein, Lipase, Fatty acid, Cholesterol, Phospholipids.

Introduction

Lipids like cholesterol and triglycerides must be carried with proteins in the circulation since they cannot be dissolved in water. To prevent toxicity, large amounts of fatty acids from meals must be delivered as triglycerides. These lipoproteins are essential for the small intestine to absorb and transport dietary lipids, for lipids to travel from the liver to peripheral tissues, and for lipids to travel from peripheral tissues to the liver and intestine. Transporting harmful external hydrophobic and amphipathic substances, including bacterial endotoxin, away from invasion and infection sites is a secondary role [1].

Structure of lipoproteins

A central hydrophobic core of non-polar lipids, predominantly cholesterol esters and triglycerides, makes up the complex particles known as lipoproteins. A hydrophilic membrane made of phospholipids, free cholesterol, and apolipoproteins encircles the hydrophobic core. The size, lipid content, and apolipoproteins of plasma lipoproteins are used to classify them into seven different groups [2].

Chylomicrons: The transfer of dietary triglycerides and cholesterol to peripheral tissues and the liver involves these large, triglyceride-rich particles produced by the colon. Apolipoproteins A-I, A-II, A-IV, A-V, B-48, C-II, C-III, and E are present in these particles. The primary structural protein is called Apo B-48, and one Apo B-48 molecule can be found in each chylomicron particle. Depending on how much fat is consumed, chylomicron size fluctuates. Due to the higher amount of triglyceride being transported after a high-fat meal, larger chylomicron particles are formed, but during a fast, chylomicron particles are small and carry lower amounts of triglyceride [3].

Low-Density of Lipoproteins (LDL): These particles, which are produced from VLDL and IDL particles, are significantly more concentrated in cholesterol. The majority of the cholesterol in the blood is carried by LDL. Each LDL particle contains one Apo B-100 molecule, which is the most common apolipoprotein. A range of particles with varied sizes and densities make up LDL. Infections, inflammatory conditions, obesity, type 2 diabetes, low HDL levels, and hypertriglyceridemia are all associated with an abundance of tiny, dense LDL particles. For a variety of reasons, these small dense LDL particles are thought to be more pro-atherogenic than big LDL particles. Small, dense LDL particles remain in the bloodstream for a longer period of time because they have a lower affinity for the LDL receptor [4].

Receptor for LDL: In addition to the liver, most additional organs include the LDL receptor. As a result of its recognition of Apo B-100 and Apo E, it mediates the endocytotic uptake of LDL, chylomicron remnants, and IDL. Following internalisation, the lipoprotein particle is broken down in lysosomes, releasing the cholesterol. The expression of LDL

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receptors and the activity of HMGCoA reductase, a crucial enzyme in the manufacture of cholesterol, are both decreased by the supply of cholesterol to the cell. Plasma LDL levels are greatly influenced by hepatic LDL receptors. The amount of cholesterol present in the cell controls the number of LDL receptors. SREBP, a transcription factor, is transported from the endoplasmic reticulum to the golgi when cellular cholesterol levels are reduced. There, proteases break and activate SREBP, which subsequently migrates to the nucleus and increases the production of LDL receptors (Figure 4). On the other hand, when cellular cholesterol levels are high, SREBP remains dormant in the endoplasmic reticulum and LDL receptor expression is low. PCSK9 controls the pace at which LDL receptors degrade, as will be addressed later [5].

Conclusion

By absorbing circulating lipoproteins and producing cholesterol on their own, peripheral cells build up cholesterol. The majority of cells lack a system for breaking down cholesterol. Cholesterol can be converted to glucocorticoids, oestrogen, testosterone, and other steroid hormones by cells that manufacture them. Cholesterol can be eliminated by intestinal cells, sebocytes, and keratinocytes secreting it into the intestinal lumen or onto the skin's surface. However, reverse cholesterol transport is necessary for the majority of cells to reduce their cholesterol burden. Clinically, it may be crucial to the prevention of atherosclerosis for macrophages to effectively efflux cholesterol into the reverse cholesterol transport system. There are two routes for the liver to absorb cholesterol after it has been transferred from cells to HDL. As was previously mentioned, HDL and hepatic SR-BI receptors can interact, leading to the selective uptake of cholesterol from HDL particles. As an alternative, CETP can transfer cholesterol from HDL particles to particles that carry Apo B, which then allows the liver to absorb lipoproteins that contain Apo B. There are many routes for cholesterol to be removed after it has been delivered to the liver. Bile acids produced from cholesterol can then be released in the bile. As an alternative, cholesterol might be released right into the bile.

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