

# **Hyperlipidemia is the Compensatory Response to the Utilization Rate Decrease of Nutrients: Lipids are Not the Culprit**

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## **Main Text**

This paper would objectively explain the importance of lipids, the main reasons for hyperlipidemia, and the harm of lowering blood lipids.

### **1. Importance of lipids**

#### 1.1 Lipids are important components of cells

Lipids, like proteins and sugars, are important raw materials of cell membrane, cytoplasm and nucleus. Whether high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C) or very low-density lipoprotein cholesterol (VLDL-C), they are the products of the lipid's metabolism. They are nutrients needed by human and should not be divided into "good" or "bad". If lipids cannot meet the needs of cells, it would lead to cells dysfunction. Since the regeneration ability of skeletal muscle cells, smooth muscle cells,

cardiomyocytes, and nerve cells is very weak, these cells would be affected first. Once these cells are lost, it would lead to irreversible organ / tissue damage and would form various diseases, such as skeletal muscle diseases<sup>1</sup>, vascular diseases<sup>2-11</sup>, cardiomyopathy<sup>5</sup> and nervous system diseases.

### 1.2 Cholesterol is the main raw material of steroid hormones

The importance of steroid hormones to the body is self-evident<sup>12</sup>. Steroid hormone disorders can lead to various diseases<sup>12</sup>, which would not be described in this paper. The main raw material of steroid hormones is cholesterol.

### 1.3 Saturated fat constitutes adipose tissue

Adipose tissue is an important member of connective tissue family<sup>7,13-17</sup>. Our body is inseparable from adipose tissue<sup>1,13,16-18</sup>. The main component of adipocytes is saturated fat. Adipose tissue, which wraps around organ / tissue, plays an inseparable role in the development, migration or remodeling of organs / tissues<sup>1,13,16-18</sup>.

### 1.4 Lipids are important in the balance of immunity and the clearance of microorganisms

It has been explained that lipids are an important part of the cells, and immune cells are no exception. Lowering LDL-C can significantly increase the risk of infection<sup>19-21</sup>.

## **2. Inducement of hyperlipidemia**

Why do blood lipids rise? Must hyperlipidemia be harmful? Why have blood lipids been mistaken for the cause of atherosclerotic disease? These are real questions.

The main reasons for hyperlipidemia may include the following aspects:

### 2.1 Reduction of demand for lipids

Due to decreased secretion of thyroid hormone / growth hormone, etc., the body's demand for nutrients such as lipids decreases, resulting in hyperlipidemia.

### 2.2 The utilization rate decrease of lipids / sugar / amino acid leads to the compensatory synthesis of lipids

Under the risk factors of metabolic diseases (diabetes, etc.), aging, gene abnormality (LDL receptor gene abnormality, familial hypercholesterolemia, apolipoprotein gene abnormality, etc.), chronic accumulation of toxicants (environmental pollution, smoking, lipid-lowering drugs, or some chemotherapeutic drugs), etc., the utilization rate of nutrients is reduced (cell needs, but the normal level of blood lipids cannot meet the needs of cells). In order to meet the needs of cells, the body has to synthesize more cholesterol / LDL-C to meet the needs. Under normal physiological conditions, the cholesterol synthesized by our liver is more than three times that ingested through diet<sup>22,23</sup>. In these people with reduced utilization rate of nutrients, even if they eat a very low-fat / low cholesterol diet, their cholesterol / LDL-C is still very high, which is very common in clinic. This suggested that in these populations with reduced utilization rate of nutrients, the cholesterol synthesized by the liver is much more than that ingested by the diet (far more than three times).

### 2.3 Excessive intake of carbohydrates, lipids or proteins

Overeating too much carbohydrates, lipids, proteins, etc.

### **3. Harm of lowering blood lipids**

Clinically, LDL-C is often "demonized", and clinicians often mistake it for the inducement of atherosclerosis. Lots of objective studies have reported that LDL-C / saturated fat is not the cause of atherosclerosis<sup>22,23</sup>. Lots of objective human pathological studies have proved that: the human endothelium is intact in initial lesion of atherosclerosis<sup>24</sup>, "fatty streaks" and "atherosclerotic plaques" were two types of lesions occurring as unrelated pathological processes<sup>25</sup>, and human atherosclerotic plaques (except for the patients with familial hypercholesterolemia) are mainly composed of various fibrous tissues<sup>4,6,26</sup> which is completely different from the so-called "atherosclerotic plaque" in arteries of other animals on a high-fat diet.

My previous reports have demonstrated that various risk factors could increase the risk of dysfunction / loss of smooth muscle cells / cardiomyocytes<sup>10,11,27</sup>. Dysfunction / loss of smooth muscle cells / cardiomyocytes would lead to vascular / cardiac media dysfunction<sup>1-5,7-11,28</sup>. Vascular / cardiac media dysfunction can significantly increase the mechanical force borne by single endothelial cells / fibroblasts in blood vessels / heart<sup>1-5,7-11,28</sup>. Thus, these cells would transform into myofibroblasts, which leads to the fibrous connective tissue remodeling of blood vessel / heart<sup>5</sup>. Depending on the dysfunction of blood vessel / heart and the degree of fibrous connective tissue remodeling, it would lead to atherosclerosis, hemangioma, stiffening, hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), ventricular aneurysm, myocardial sclerosis, etc<sup>2,4,5,8</sup>. The media dysfunction theory can explain the

phenomena of human blood vessels<sup>1-18,27-31</sup>, which cannot be explained by those traditional hypotheses.

Blood lipids, blood glucose, protein, oxygen, etc. are essential for human survival, cell survival, or connective tissue remodeling, etc. Lipids are certainly involved in connective tissue remodeling. However, even if lipids are reduced to a very low level, fibrous connective tissue remodeling would still progress, which is the reason why atherosclerotic disease would still progress after the blood lipids is reduced to a very low level with lipid-lowering agents in clinic. But some reports still advocate “the lower the LDL-C, the better”, which is as absurd as the old "bloodletting anesthesia". If LDL-C is reduced to zero, people would die first and no disease would progress again. The statement that "the lower the LDL-C, the better" cannot be falsified, so it is not a scientific problem. All nutrients have a suitable concentration range, and so do blood lipids. Too high triglycerides increase the risk of pancreatitis. For people with low utilization rate of nutrients, an appropriate increase of blood lipids might be beneficial, so excessive reduction of blood lipids is harmful to the body.

3.1 Lowering blood lipids increases the risk of myocardial infarction<sup>32,33</sup>.

3.2 Lowering blood lipids increases the risk of infection, autoimmune diseases or tumors<sup>19-21</sup>.

3.3 Lowering blood lipids increases the risk of diabetes.

3.4 Lowering blood lipids increases the risk of damage of skeletal muscle cells, smooth muscle cells, cardiomyocytes and nerve cells.

3.5 Lowering blood lipids increases the risk of aneurysms, atherosclerotic

disease, heart failure, skeletal muscle diseases and neurological diseases.

Since lowering blood lipids increases the risk of dysfunction of muscle cells / nerve cells, thus it might increase the risk of these diseases.

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