Metabolic Reprogramming of Macrophage in Atherosclerosis: Is it All about Cholesterol?

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Abstract

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Heart disease kills more people than any other worldwide. In an effort to understand the underlying mechanisms that cause heart disease, at least two distinct lines of thinking have emerged. First, obesity, diabetes, hypertension, high triglycerides, and low HDL cholesterol – the key constituents of the metabolic syndrome – have been recognized as major risk factors. Second, inflammation – the immune system's ancient defense program against infection and injury – has surfaced as a critical component accompanying most stages of disease. Although metabolism and inflammation are essential to survival, involving all tissues throughout life, we still know very little how these processes influence each other. After a contribution to the development of new therapeutics for cardiovascular diseases at Pfizer, the research work of Dr Yvan-Charvet has mainly been focused on how metabolic regulation of the hematopoietic tree occurs in chronic inflammatory diseases such as atherosclerosis – a new area of research for cardiovascular diseases. More recently, a hypothesis-driven selection of biochemical pathways linking metabolism and inflammation has emerged by filtering 'Omics' studies through a 'metabolic pathways' analysis bioinformatic tools. The metabolic reprogramming of macrophage in atherosclerosis beyond cholesterol will be discussed.

Keywords

Hematometabolism, cardiometabolic diseases, atherosclerosis