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Editorial

Essential Omega-3 fatty Acids and Lipid Equilibrium Attenuation in Overexpressing Glial cell Foam Cells

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Abstract

The rates of cholesterol uptake and efflux determine the transformation of macrophages into foam cells. Conjugated linoleic acid (CLA), alpha-linolenic acid (ALA), and eicosapentaenoic acid (EPA) are examined in this study using a real-time RT-PCR method to investigate their roles in the regulation of the liver X receptor (LXR) and ATP-binding cassette A1 (ABCA1) genes, which are involved in cholesterol homeostasis. As a result, the total, free, and esterified cholesterol levels in the foam cells were significantly reduced by these fatty acids. The pharmacological LXR ligand T0901317 increased the expression of the ABCA1 and LXR genes, but CLA, ALA, and EPA had no significant effect on their mRNA expression (Rom et al., 2016). These findings suggest that, despite their influence on cholesterol homeostasis, polyunsaturated fatty acids cannot alter the expression of the ABCA1 and LXR genes. Alternately, a number of additional proteins and genes may be involved (Hampton 2017).

Keywords: THP-1; acid conjugated to linoleic; Alpha-linolenic acid; Eicosapentaenoic corrosive; The liver's X receptor The A1 ATP-binding cassette

INTRODUCTION

The pathologic condition known as atherosclerosis is caused by the accumulation of cholesterol in the walls of the arteries, which makes it possible for macrophages to absorb modified low-density lipoprotein (m-LDL). In macrophages, the formation of foam cells, which are crucial to the onset and progression of atherosclerosis, is influenced by the equilibrium of cholesterol uptake and efflux. Various qualities and proteins, for example, ATP-restricting tape A1 (ABCA1), liver X receptors (LXRs), sreol administrative component restricting proteins (SREBPs) and Peroxisome proliferator-actuated receptors (PPARs), are engaged with the course of froth cell arrangement, and investigation of these variables and their administrative systems is of extraordinary incentive for the avoidance of cardiovascular sickness (CVD). The large ATP-binding cassette transporter family includes ABCA1. Cholesterol and phospholipids are transported by these transmembrane proteins to apolipoprotein A-I (apoA-I), which is either lipid-poor or lipid-free (Rom et al., 2018). In point of fact, ABCA1, whose function is faulty in Tangier disease, is a key protein in regulating plasma HDL cholesterol levels and cellular cholesterol homeostasis.

DISCUSSION

Class II nuclear receptors known as liver X receptors (LXR) and LXR directly bind to the LXRE sequence as a heterodimer with Rethinoid X Receptor (RXR). It has been demonstrated that oxysterols, such as 24, 25-epoxycholesterol and 22(R)-hydroxycholesterol, bind to and activate the LXRs. By binding LXR/RXR heterodimers to LXRE sequences in the promoters of genes whose products are involved in hepatic bile acid synthesis, such as 7-hydroxylase (Cyp7A), this activation activates the primary pathway for cholesterol elimination. In addition, the ABCA1 and ABCG1 genes are induced to be expressed by LXR, which controls the accumulation of cholesterol esters. Joseph et al. showed that activating LXR

prevented mice from developing atherosclerosis, and some studies suggested that unsaturated fatty acids (USFAs) may have a stimulant effect on LXR but not LXR in cultivated hepatoma cells. Notwithstanding, rather than these reports, different analysts accept that USFAs stifle the declaration of the LXR quality (Caldow et al., 2016). Additionally, it has been demonstrated that PUFAs compete with oxysterols for binding to LXR and, as a result, inhibit the oxysterol-induced upregulation of SREBP-1c. Compared to palmitic, stearic, or oleic acids, it has been demonstrated that eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids reduce the cellular content of cholesterol-ester (CE) by approximately 50%. However, this rate of reduction for some other fatty acids, such as -3 linolenic, -6 linoleic, and arachidonic acids, In point of fact, arachidonic, palmitic, stearic, and linolenic acids do not have the same effect as EPA, which is a poor substrate for esterification that reduces the incorporation of oleic acid into CE by up to 50%. As needs be, when human fibroblasts were filled within the sight of EPA for five days, both FC and CE levels were decreased in correlation with control and linoleic corrosive treated cells (Heresco et al., 1999).

Based on the aforementioned studies taken as a whole, it is still unclear what role ABCA1 and LXR play in the development of foam cells. This study therefore sought to address the influence of several distinct polyunsaturated fatty acids on the transcription level of ABCA1 and LXR genes in macrophages as well as the cholesterol homeostasis in foam cells with the ultimate goal of studying the effect of expression of these two genes on the formation of foam cells.

CONCLUSION

In conclusion, although this study suggests that fatty acids play a role in reducing cholesterol accumulation within foam cells, it did not include ABCA1 and LXR's distinct roles in this process. As a result, it may be inferred that fatty acids may exert their influence through additional pathways, such as PPARs. The course of lipid digestion inside the froth cells is by all accounts extremely confounded, and this reality can make sense of the questionable outcomes that are accounted for by various examination gatherings. As a result, it will be necessary to evaluate other involved genes and factors in depth simultaneously.

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