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The effect of lipid metabolism on biological characteristics of hepatic stellate cell

JING Xinyan, YANG Xuefeng *

Department of the Affiliated Nanhua Hospital, the University of South China, Hengyang 421000, Hunan, China

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Abstract

Hepatic stellate cells(HSCs) are a kind of fat-storing cells, the lipid droplets are rich in the Cytoplasm, in which retinyl ester accounts for 42%, triglyceride occupies 28% , cholesterol (total) occupies 13% , phospholipids occupies 4% respectively. Studies have confirmed that thetransforms of HSC phenotype follows the changing of the cell lipid. After the activation of HSC, with HSC phenotype changing from fat-storing cells into myofibroblast, the lipid droplets decreased or disappeared gradually, which means HSCs are under the differentiating process of removing adipose, meawhile triglyceride, and the main content of lipid droplets, also obviously reduced. It was ever declined that during the process of HSC re-fating, the activated HSC would turn into quiescent state. Therefore this shows HSCs fat metabolism is closely related to the biological activity.

Keywords: Hepatic stellate cells; Lipid metabolism; Cell proliferation; Apoptosis; Liver fibrosis

*Corresponding author.

E-mail address: yxf9988@126.com（YANG X.）

1. Introdution

Hepatic stellate cells (HSCs) is a sort of fat-storing cells, and the lipid droplets are rich in its Cytoplasm. In 1987, Yamada et al [1]. analyzed lipid droplets in rat HSCs which consiste consist with 42% retinyl ester, 28% triglyceride, 13% cholesterol (total), and 4 % phospholipids. Moriwaki et al [2]. isolated HSCs by pronase- collagenase dissociation method and separated lipid droplets of HSCs by differential centrifugation, their result of composition of lipid droplets is similar to the result of Yamada, of which retinyl-ester accounted for 39.5%, triglyceride 31.7%, cholesterol ester 15.4%, cholesterol 4.7%, phospholipids 6.3%, and free fatty acids 2.4%.

Activation of hepatic stellate cells (HSC), a resident pericytic cell in liver, into a proliferative and fibrogenic cell type, is the principal event underlying hepatic fibrosis following injury [3]. Release of lipid droplets containing retinyl esters and triglyceride is a defining feature of HSC activation but the foundation for release of lipid droplets remained unclear. Another literature says that the number of lipid droplets increased in culture-activated HSC, which is transected by the peroxisome proliferator-activated receptor gamma (PPAR-γ), while phenotype of HSC was re-fatting and switching from the active to the quiescent [4-5]. Thus it can be seen that HSCs fat metabolism is closely associated with its biological activity.

2. Effect of triglyceride metabolism on biological activity of hepatic stellate cell

In review of previous researches, we can conclude that there is some relationship between triglyceride metabolism and HSCs' state. Gäbele et al [6]. investigated that after high fat diet ,such as pathological processes are associated with too much extracellular matrix deposition. The activation of HSCs in the rat's liver, and the multiplication of collagen type Ⅰ, transforming growth factor beta (TGF-β), and tumor necrosis factor which indicates high fat diet have the function of inducing the occurrence of hepatic fibrosis. Lun-Gen Lu et al [7-9]. found that low-density lipoprotein (LDL), high-density lipoprotein (HDL) receptors exist on the membrane surface of the rat's HSC, and triglyceride (TG) and very low density lipoprotein (VLDL) can increase the affinity of LDL to LDL receptor, and decrease the affinity of HDL to HDL receptor, and promote the expression of procollagen type Ⅱ and Ⅲ mRNA in HSC, TG, VLDL and Kupffer cell-conditioned medium can advance the proliferation of HSC. Now studies [10-13] have demonstrated that LDL could activate HSC, but curcumin could repress this process through