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Editorial

Cholesterol Crystallization: A Little Understood Topic in the Pathogenesis of Gallstones

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Cholesterol gallstones formation involves many physiological factors, among them are: alteration in biliary lipid secretion, gallbladder stasis, cholesterol crystallization, and bile sludge formation [1]. The crystallization of cholesterol is referred to as the key process for the formation of a gallstone [2]. When the gallbladder is supersaturated with cholesterol, the molecules of this lipid collide themselves, causing the nucleation and growth of cholesterol crystals. The monohydrated crystals produce inflammatory reactions in the gallbladder, there is secretion of biliary sludge, which later forms the matrix where cholesterol is deposited and the gallstone is formed [3]. A key research in gallstone therapy is the analysis of the molecular mechanisms that prevent the nucleation of cholesterol. There are antinucleating agents, for example, osteopontin, a phosphorous protein that can bind calcium and slow down the nucleation rate of cholesterol [4]. This protein is abundantly expressed in healthy biliary epithelium; however, its expression decreases in the presence of gallstones.

Cholesterol crystals produce the activation of mucin proteins in the gallbladder, for this reason, it is very important to investigate some physicochemical mechanisms to prevent cholesterol from modifying its state in the bile. Concomitant therapies could be considered, where antinucleating agents are used and, at the same time, some substance that can transport excess cholesterol in the bile to avoid its supersaturation. In this section, it is feasible to consider the participation of ursodeoxycholic acid, since it promotes the formation of bile salts, which are assembled within the mixed micelles, to transport and emulsify excess cholesterol.

Bibliography

1. Portincasa P., *et al*. "The physical presence of gallstone modulates ex vivo cholesterol crystallization pathways of human bile". *Gastroenterology Reports* 7.1 (2019): 32-41.

2. Castro-Torres IG., *et al*. "Future therapeutic targets for the treatment and prevention of cholesterol gallstones". *European Journal of Pharmacology* 765 (2015): 366-374.

3. Grebe A and Latz E. "Cholesterol crystals and inflammation". *Current Rheumatology Reports* 15 (2019): 313.

4. Yang L., *et al*. "Osteopontin and integrin are involved in cholesterol gallstone formation". *Medical Science Monitor* 8 (2012): 16-23.

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