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

Ibrahim Guillermo Castro-Torres1*, Elia Brosla Naranjo-Rodríguez2 and Minerva Hernández-Lozano3

1Escuela Nacional Colegio de Ciencias y Humanidades, Colegio de Ciencias y Humanidades, Plantel Sur, Universidad Nacional Autónoma de México, Ciudad de México, México
2Departamento de Farmacia, Facultad de Química, Universidad Nacional Autónoma de México, Ciudad de México, México
3Facultad de Química Farmacéutica Biológica, Universidad Veracruzana, Xalapa de Enríquez, Veracruz, México

*Corresponding Author: Ibrahim Guillermo Castro-Torres, Escuela Nacional Colegio de Ciencias y Humanidades, Colegio de Ciencias y Humanidades, Plantel Sur, Universidad Nacional Autónoma de México, Ciudad de México, México.

Received: June 03, 2021; Published: June 26, 2021

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


Volume 8 Issue 7 July 2021
©All rights reserved by Ibrahim Guillermo Castro-Torres, et al.