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Interactions Between Bile Salts, Gut Microbiota, and Hepatic Innate Immunity

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Abstract

Bile salts are the water-soluble end products of hepatic cholesterol catabolism that are released into the duodenum and solubilize lipids due to their amphipathic structure. Bile salts also act as endogenous ligands for dedicated nuclear receptors that exert a plethora of biological processes, mostly related to metabolism. Bile salts are actively reclaimed in the distal part of the small intestine, released into the portal system, and subsequently extracted by the liver. This enterohepatic cycle is critically dependent on dedicated bile salt transporters. In the intestinal lumen, bile salts exert direct antimicrobial activity based on their detergent property and shape the gut microbiota. Bile salt metabolism by gut microbiota serves as a mechanism to counteract this toxicity and generates bile salt species that are distinct from those of the host. Innate immune cells of the liver play an important role in the early recognition and effector response to invading microbes. Bile salts signal primarily via the membrane receptor TGR5 and the intracellular farnesoid-x receptor, both present in innate immune cells. In this review, the interactions between bile salts, gut microbiota, and hepatic innate immunity are discussed.

Keywords: FXR; bile salt metabolism; bile salt signaling; bile salt toxicity; innate immunity; microbiota:host interaction.

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