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Hepatitis C: From inflammatory pathogenesis to anti-inflammatory/hepatoprotective therapy

Li H *et al.* Anti-inflammatory/hepatoprotective therapy of HCV

Hu Li, Meng-Hao Huang, Jian-Dong Jiang, Zong-Gen Peng

Abstract

Hepatitis C virus (HCV) infection commonly causes progressive liver diseases that deteriorate from chronic inflammation to fibrosis, cirrhosis and even to hepatocellular carcinoma. A long-term, persistent and uncontrolled inflammatory response is a hallmark of these diseases and further leads to hepatic injury and more severe disease progression. The levels of inflammatory cytokines and chemokines change with the states of infection and treatment, and therefore, they may serve as candidate biomarkers for disease progression and therapeutic effects. The mechanisms of HCV-induced inflammation involve classic pathogen pattern recognition, inflammasome activation, intrahepatic inflammatory cascade response, and oxidative and endoplasmic reticulum stress. Direct-acting antivirals (DAAs) are the first-choice therapy for effectively eliminating HCV, but DAAs alone are not sufficient to block the uncontrolled inflammation and severe liver injury in

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