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# Role of inflammatory response in liver diseases: Therapeutic strategies

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**Abstract**

Inflammation and tumorigenesis are tightly linked pathways impacting cancer development. Inflammasomes are key signalling platforms that detect pathogenic microorganisms, including hepatitis C virus (HCV) infection, and sterile stressors (oxidative stress, insulin resistance, lipotoxicity) able to activate pro-inflammatory cytokines interleukin-1β and IL-18. Most of the inflammasome complexes that have been described to date contain a NOD-like receptor (NLR) sensor molecule. Redox state and autophagy can regulate inflammasome complex and, depending on the conditions, can be either pro- or anti-apoptotic. Acute and chronic liver diseases are cytokine-driven diseases as several proinflammatory cytokines [IL-1α, IL-1β, tumor necrosis factor-alpha (TNFα), and IL-6] are critically involved in inflammation, steatosis, fibrosis, and cancer development. NLRP3 inflammasome gain-of-function aggravates liver

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