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Liver steatosis in hepatitis C patients

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Abstract

There is controversy regarding some aspects of hepatitis C virus (HCV) infection-associated liver steatosis, and their relationships with body fat stores. It has classically been found that HCV, especially genotype 3, exerts direct metabolic effects which lead to liver steatosis. This supports the existence of a so called viral steatosis and a metabolic steatosis, which would affect HCV patients who are also obese or diabetics. In fact, several genotypes exert metabolic effects which overlap with some of those observed in the metabolic syndrome. In this review we will analyse the pathogenic pathways involved in the development of steatosis in HCV patients. Several cytokines and adipokines also become activated and are involved in "pure" steatotic effects, in addition to inflammation. They are probably responsible for the evolution of simple steatosis to steatohepatitis, making it difficult to explain why such alterations only affect a proportion of steatotic patients.

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Key words: Hepatitis C virus steatosis adiponectin; Leptin; Insulin resistance; Proinflammatory cytokines; Triacylglyceride synthesis; Fatty acid oxidation

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