Special Issue

Liver Fibrosis: Mechanisms, Targets, Assessment and Treatment

Message from the Guest Editors

Fibrosis is a double-edged sword. On the one hand, it can be the final state of healed inflammation as scar tissue; on the other hand, it is frequently associated with a reduction in or loss of organ function. Moreover, especially in liver disease, it is a surrogate parameter that indicates the progression of the disease to liver cirrhosis or even hepatocellular carcinoma. This is especially true for non-alcoholic fatty liver disease, a pandemic disorder associated with Western lifestyles and diets. To influence organ fibrosis, it is important to better understand its induction, perpetuation and termination at the molecular level. The induction of liver fibrosis may be metabolic (e.g., alcohol, diet and drugs), infectious (e.g., viruses), autoimmune (e.g., primary biliary cholangitis) or due to monogenetic defects (e.g., increased iron storage). The molecular mechanisms leading to final-stage fibrosis are very different—dependent on its pathogenesis. It is the aim of this Special Issue to provide more insight into these processes.

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