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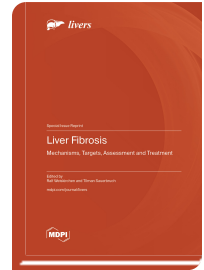
Liver Fibrosis

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Fibrosis is a double-edged sword. On one hand, it can be the final stage of healed inflammation as scar tissue. On the other hand, it is often linked to a decrease in organ function or complete organ failure. Additionally, in liver disease, fibrosis serves as an indicator of disease progression towards liver cirrhosis or hepatocellular carcinoma. This is particularly true for non-alcoholic fatty liver disease, a widespread disorder associated with Western lifestyles and diets. To interfere with organ fibrosis, it is crucial to have a better understanding of its initiation, perpetuation, and resolution at the molecular level. The development of liver fibrosis can be caused by metabolic factors (such as alcohol, diet, and drugs), infections (such as viruses), autoimmune conditions (like primary biliary cholangitis), or genetic abnormalities (such as increased iron storage). The molecular pathways that lead to advanced fibrosis vary depending on the underlying cause. The goal of this Special Issue is to dissect these processes and provide more clarity on the subject.



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