Special Issue

Exploration of Drug Targets for Liver Fibrosis Based on Hepatic Stellate Cells and Extracellular Matrix Remodeling

Message from the Guest Editor

Liver fibrosis (LF) is a pathological wound-healing response manifested through the excessive buildup of extracellular matrix (ECM) in the liver as a result of prolonged inflammation and injury. It is the common denominator of most types of chronic liver disease caused by different etiologies (alcohol consumption, viral hepatitis, and steatosis related to metabolic dysfunction being the most common). If it progresses, LF can lead to severe, irreversible damage called cirrhosis, causing liver failure, portal hypertension with its complications (ascites, jaundice, and bleeding), and the development of HCC (hepatocellular carcinoma). Activation of hepatic stellate cells (HSCs) into a myofibroblast phenotype represents a key event in LF, as these cells are the primary source of ECM. Of note, although improved management of the underlying diseases can halt progression and, to an extent, even reverse LF, there are currently no specific approved antifibrotic therapies. Recent research has focused on HSCs and, specifically, ECM remodeling as therapeutic targets.

Guest Editor

Prof. Dr. Nadezda Apostolova
Department of Pharmacology, University of Valencia, avda. Blasco Ibañez n.15, 46010 Valencia, Spain

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Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland
Tel: +41 61 683 77 34
biomolecules@mdpi.com

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