Special Issue

Molecular Mechanisms of Fibrosis in Chronic Liver Diseases

Message from the Guest Editors

Liver fibrosis is a progressive condition marked by excessive extracellular matrix (ECM) accumulation due to chronic liver injury. It often stems from metabolic dysfunction-associated steatohepatitis (MASH), viral hepatitis, alcohol misuse, and autoimmune or cholestatic diseases. At its core, fibrosis is driven by the persistent activation of hepatic stellate cells (HSCs), which transform into myofibroblast-like cells that produce ECM proteins, gradually impairing liver structure and function. Its progression involves complex interactions among cytokine signaling, immune cell communication, oxidative stress, and metabolic changes. Recent studies highlight the roles of immune–metabolic pathways, gut–liver axis imbalance, and epigenetic modifications in fibrogenesis. Emerging tools like single-cell transcriptomics and spatial omics are uncovering the cellular diversity and regulatory networks in the fibrotic liver. This Special Issue aims to explore the molecular mechanisms of hepatic fibrosis and their translational potential. We welcome original research and reviews on fibrosis biology, biomarker development, and antifibrotic therapies. We look forward to your contributions.

Guest Editors

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Message from the Editorial Board

Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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