Special Issue

Molecular and Cellular Mechanisms of Treating Fibrosis

Message from the Guest Editor

Fibrosis, defined as excessive extracellular matrix (ECM) deposition through persisting myofibroblast activation, is a common and resistant pathologic process, contributing widely to morbidity through organ involvement and increased mortality through the eventual failure of organ systems. Current approaches involve the empirical use of anti-inflammatory or immunosuppressive therapies given during any inflammatory phase, or more specific approaches such as the use of nintedanib which targets receptors for growth factors and angiogenic cytokines, or perfenidone which may inhibit ECM assembly outside of the cell. Understanding of the cellular and molecular mechanisms driving complex fibrosis. Future therapies likely involve targeting key cellular players such as M2-like macrophages, myofibroblasts themselves, or adaptive immune cells, or modification of the ECM itself such as preventing its secretion-stable assembly of fibrillar collagen matrix. Taken together, the clinical problem of fibrosis presents an intriguing conundrum which is likely solvable through modern research approaches and the delivery of targeted therapy.

Guest Editor

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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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