Tentative Outline

Special Thematic Issue for Current Molecular Medicine

Title of Thematic Issue

Fibrosis: from cellular and molecular targets to therapeutic strategies.

Guest Editor:

Chiara Sassoli

Aims & Scope:

Fibrosis represents a pathological condition frequently occurring in different organs as an aberrant maladaptive reparative response to severe, repeated or chronic damage, irrespective of the underlying etiology. It is characterized by an excessive tissue scarring due to an overproduction and deposition of extracellular matrix (ECM) mainly attributable to the imbalance between synthesis and degradation of ECM components, particularly collagens, often in association with uncoordinated detrimental contractures. Such process often leads to the morpho-functional impairment of the organ and, in the worst cases, to end-stage organ disease. Severe fibrosis-related pathologies are estimated to account for up to 50% of all causes of death in industrialized countries and, on the other hand, effective therapeutic tools for elimination of the causing noxa are not always available. In addition, it must be considered that most human fibrotic diseases are often multifactorial in origin, so that a direct action on the underlying etiology may be virtually impossible. The current antifibrotic therapeutic tools are of limited efficacy and, at present, organ transplantation, when possible, represents the only option, even if all the related critical issues and concerns have to be considered. Therefore, the identification of alternative and effective therapies aimed to prevent and even more counteract and revert excessive tissue scarring is a current scientific challenge and an urgent medical need with high impact on health care system. To accomplish this task it appears imperative to decipher the cellular and molecular mechanisms underpinning the pathogenesis of fibrosis which may be regarded as potential and preferential therapeutic targets. To date the recognized “core cellular mechanisms of fibrosis” is the generation of myofibroblasts and their persistence in activated functional state. These cells derive mainly from the differentiation of resident fibroblasts in the ECM, promoted by the action of several soluble pro-fibrogenic mediators, such as transforming growth factor (TGF)-β1 released by infiltrating inflammatory cells and other local cell types at the site of the injury, integrated with mechanical stimuli coming from the injured microenvironment. However, recently, other cells different from fibroblasts, such as epithelial cells, endothelial cells, adipocyte/adipose-derive stem cells, pericytes, monocytes/macrophages have been identified as potential myofibroblast progenitors and as modulators of tissue scarring.

In this Special Issue, contributions are encouraged to further and deeper investigate into the cell types and the molecular mechanisms involved in the onset and progression of fibrosis in different organs and into the definition of potential therapeutic approaches.

We hope that this Special Issue will highlight the relevance of this topic and the current trends and will provide new insights into cellular players and molecular signaling pathways driving fibrosis potentially

able to accelerate the development of effective treatment for those diseases where fibrosis plays a major etiological role.

Subtopics:

Myofibroblasts

Epithelial-mesenchymal transition

Endothelial-mesenchymal transition

Adipocyte-myofibroblast transition

Extracellular matrix remodelling

Inflammasomes

Pro-fibrotic and anti-fibrotic factors and signaling pathways

Pharmacological therapy

Photobiomodulation/Low level laser therapy

Cell based therapy

Blood products-platelet rich plasma

Contacts:

Guest Editor: Dr. Chiara Sassoli

Affiliation: Department of Experimental and Clinical Medicine - Section of Anatomy and Histology University of Florence Largo Brambilla 3, 50134 Florence, Italy Tel +39 552758063 Fax +39 554379500, Italy

E-mail chiara.sassoli@unifi.it

Any queries should be addressed to cmm@benthamscience.net