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Special Issue

Inflammation and fibrosis: from molecular mechanisms to therapeutic opportunities

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Dear Colleadues,

Inflammation and fibrosis are critical pathological processes involved in many human diseases. These include inflammatory pulmonary fibrosis, uterine fibroids, kidney fibrosis, liver fibrosis, cardiac fibrosis, pancreas fibrosis, hypertrophic/keloid scars, scleroderma and systemic sclerosis, dupuytren's and peyronie's disease, atherosclerosis and acute coronary syndromes, as well as many tumors and cancers. Inflammation is the body's first response to injury. Fibrosis is the result of defective repair processes often seen after chronic injury and/or inflammation that is associated with excessive accumulation of ECM proteins leading to disruption of normal tissue architecture.

Quiescent cells are activated by tissue injury, and/or inflammation. Cytokines, chemokines, and growth factors are produced at the site of injury and contribute to fibroblast activation and differentiation into myofibroblasts. Angiogenic growth factors also take part in this process. To restore homeostatic condition, myofibroblasts produce ECM proteins and maintain MMPs (matrix metalloproteinases)-TIMPs (inhibitors of metalloproteinases) balance, resulting in wound healing and myofibroblasts are eliminated by apoptosis. However, under a chronic inflammatory state, myofibroblasts become resistant to apoptosis, producing excessive amounts of ECM proteins, and therefore MMP-TIMP imbalance which induces cells to undergo fibrotic transformation.

In recent years, significant progress has been made in the understanding of the contribution of inflammation and fibrosis in human diseases as well as the development of new therapeutic compounds. Therefore, the purpose of this special issue of Frontiers in Bioscience-Landmark is to collect original research articles and reviews on the role of inflammation and fibrosis in human diseases as well as the current status of therapeutic compounds (from natural to synthetic compounds) that target inflammation and fibrosis.

Md Soriful Islam and Most Mauluda Akhtar

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