Special Issue

TGF-□ Signaling in Immunity, Inflammation, Fibrosis and Cancer

Message from the Guest Editors

Due to its role in immune homeostasis, perturbations of TGF-□ signaling underlies inflammatory diseases. Many chronic inflammatory diseases are marked by fibrosis, which concurs with excessive deposition of the extracellular matrix, resulting in the loss of normal function of the affected organs. The TGF-□ family also plays essential roles in the initiation and progression of fibrosis, through the activation of fibroblasts towards a myofibroblast phenotype. During the early stages of tumorigenesis, TGF-□ may act as a tumor suppressor by inducing cytostasis and apoptosis of preneoplastic cells. However, at later stages, when cancer cells have acquired oncogenic mutations that allow scaping from TGF-□ tumor suppressor function, it becomes a tumor promoter by stimulating tumor cells to undergo epithelial–mesenchymal Transition (EMT), which increases migration and invasion. TGF-□ is also central to immune suppression within the tumor microenvironment, and recent studies have revealed roles in tumor immune evasion and poor responses to cancer immunotherapy.

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