Topical Collection

TGF-Beta Signaling in Tissue Fibrosis and Cancer

Message from the Collection Editors

- TGF-β-mediated transcriptional (both SMAD and non-SMAD) networks, genetic reprogramming, and phenotypic responses (e.g., cell plasticity/stemness, cell cycle arrest, proliferation, migration) related to the onset or progression of fibrotic and neoplastic diseases.
- Non-transcriptional (e.g., microRNA, lncRNA, epigenetic) control of TGF-β1 signaling.
- TGF-β crosstalk with other receptors (e.g., tyrosine kinases and serine/threonine kinases) or tumor suppressors (e.g., p53, PTEN) in promoting or suppressing fibrotic and oncogenic behavior.
- Novel positive (e.g., inducers) and negative regulators (e.g., suppressors) of TGF-β1 pathways.
- Novel or potential therapeutic approaches (TGF-β ligand traps and neutralizing antibodies, signaling networks, or TGF-β collateral networks) to target aberrant TGF-β signaling in organ fibrosis and cancer.
- TGF-β1-induced metabolic alterations (e.g., glycolysis, Krebs cycle, oxidative phosphorylation, fatty acid oxidation) in tissue fibrosis and cancer.
- TGF-β1 control of inflammatory networks.
- Tissue or organ specificity of TGF-signaling.

Collection Editors

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Biomolecules is a multidisciplinary open-access journal that reports on all aspects of research related to biogenic substances, from small molecules to complex polymers. We invite manuscripts of high scientific quality that pertain to the diverse aspects relevant to organic molecules, irrespective of the biological question or methodology. We aim for a competent, fair peer review and rapid publication. Please look at some of the exciting work that has been published in *Biomolecules* so far. We would be delighted to welcome you as one of our authors.

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