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TGF-Beta Signaling in Tissue Fibrosis and Cancer

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Message from the Collection Editors

1. 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.
2. Non-transcriptional (e.g., microRNA, lncRNA, epigenetic) control of TGF-β1 signaling.
3. 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.
4. Novel positive (e.g., inducers) and negative regulators (e.g., suppressors) of TGF-β1 pathways.
5. 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.
6. TGF-β1-induced metabolic alterations (e.g., glycolysis, Krebs cycle, oxidative phosphorylation, fatty acid oxidation) in tissue fibrosis and cancer.
7. TGF-β1 control of inflammatory networks.
8. Tissue or organ specificity of TGF-signaling.

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Topical Collection

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