STAT3 as a molecular target for cancer prevention and therapy

STAT3 comprises a family of cytoplasmic transcription factors that transmit signals, mediate intracellular signaling usually generated at cell surface receptors and transmitted to the nucleus. Numerous studies have demonstrated constitutive activation of STAT3 in a wide variety of human tumors, including blood malignancies (leukemias, lymphomas and multiple myeloma) as well as solid tissues (such as head and neck, breast, lung, gastric, hepatocellular and prostate cancers). There is strong evidence to suggest that aberrant STAT3 signaling promotes development and progression of human cancers by either inhibiting apoptosis or inducing cell proliferation, angiogenesis, invasion and metastasis. However, the development of novel drugs for the targeting STAT3 that is both safe and efficacious remains an important scientific and clinical challenge. We will present the data that shows that novel small molecule inhibitors of STAT3/JAK2 pathway can suppress the expression of genes involved in cancer initiation and promotion both in vitro and in vivo.

Biography

Gautam Sethi has completed his Postdoctoral training at University of Texas MD Anderson Cancer Center and joined Department of Pharmacology, Yong Loo Lin School of Medicine, National University of Singapore in 2008 as an Assistant Professor and was promoted to Associate Professor in 2015. The focus of his research over the past few years has been to elucidate the mechanism(s) of activation of oncogenic transcription factors such as NF-κB/STAT3 by carcinogens and inflammatory agents and the identification of novel inhibitors of these proteins for prevention of and therapy for cancer. He has more than 150 scientific publications in high impact factor peer reviewed journals and has several international awards to his credit. He currently serves as an Academic Editor for PLOS ONE, Editorial Board Member of Scientific Reports and ad-hoc Reviewer for several other international journals.

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