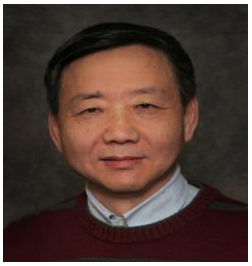
Frontiers in Bioscience-Landmark

Special Issue

MAPKs and Oncogenesis

Guest Editor



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Message from the Guest Editor

Dear Colleagues,

Kinases can amplify oncogenic signals by phosphorylating substrates and thereby play a decisive role in oncogenesis. Deregulated kinases in cancer are druggable and represent attractive targets for therapeutic intervention, and their precise roles in oncogenesis, however, are mostly not well-established. Mitogen-activated protein kinases (MAPKs) play a fundamental role in relaying and processing extracellular and intracellular signals to regulate cell growth, cell death and cell transformation through substrates, target genes, interacting partners, and cross-talking with other pathways. For example, extracellular-signal regulated kinases (ERKs) are major proliferative pathways for Ras and other oncogenes, whereas p38 and c-Jun N-terminal kinases (JNKs) are activated by stress signaling and inflammation stimulus and can either promote or inhibit inflammation-associated cancer. Recent genetic studies show that p38 MAPK family proteins play a cell-type dependent and isoform-specific role in inflammation and in inflammation-associated cancer. This knowledge is critical for understanding basic mechanisms of oncogenesis and is fundamentally important for precision oncology. This special issue aims to update recent developments in the field of MAPKs and oncogenesis and discuss potential approaches to effectively intervene tumorigenesis by targeting a specific MAPK pathway.

Prof. Guan Chen, MD, PhD

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