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Mitogen-Activated Protein Kinase (MAPK) Signaling Pathway and Mammalian Cells

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Short Communication

The mitogen-activated protein kinase (MAPK) pathway has been implicated as a key signaling system that mediates extracellular signals through a cascade of phosphorylation in mammalian cells. There are three distinct tiers in the MAPK pathway that are stimulated by different stress signals, ERK, JNK, and p38. Identifying cell signaling molecules as a response to stress is of extreme importance, since signal transduction provides the necessary link between a stimulus from the external environment of a cell and the events that occur among its intracellular components. Modulation of biomolecules in the MAPK family is a key mechanism of action and has the potential to cause cell cycle progression, proliferation, and differentiation. However, the onset of cancer is influenced by mutated regulatory genes. Therefore, when gene mutations occur, their products interact with biomolecules of the MAPK pathway. This interaction cause molecular events such as tumorigenesis to manifest.

Many human cancers are connected to inactivated tumor suppressor genes or the transcriptional activation of multiple oncogenes. Although the causes of cancer are diverse, most involve the transformation of cells by altering genes that control cell division. Notably, the initiation- or progression phase of carcinogenesis is the recurring loss of function in tumour suppressor genes. Mammalian hepatocytes respond to a variety of environmental stress via an intricate signaling mechanism that coordinate long-term cell adaptation or programmed cell death. Intracellular signal transduction pathways, aggravated by stress, are thought to be directly responsible for regulating cell proliferation, differentiation, and survival in mammalian hepatocytes. Further, cytotoxic stresses can influence the synthesis of protective proteins that are associated with biotransformation activity, cell cycle progression, DNA-impairment, proliferative machinery, oxidative damage, and protein perturbations.

Mammalian hepatocytes are characterized by their high rate of metabolic activity and are useful models in toxicogenomics. Further, mammalian hepatocytes are good models to investigate cell signaling pathways. The role of the MAPK biomolecules with regard to growth, cell proliferation, and differentiation has not been clearly established in hepatocellular cancer. The search for anti-cancer therapy treatment has led the scientific community to actively seek information about impact of MAPK inhibitors on tumorigenesis. Chemicals that inhibit proliferation and migration of hepatocytes represent a potential pharmacological tool for inhibiting solid tumor growth as well as hepatocellular malignancies. Understanding the behavior of biomolecules in the MAPK family is of key importance to develop anti-cancer agents.

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