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Editorial

MapK Pathway: A Novel Research

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Dear Readers

The Journal of cell Signaling is an open access journal which publishes peer reviewed articles to engage the viewers with the latest research work in an elaborative way. As an Editorial member, I express my heartfelt gratitude to the entire editorial board for strengthening its base on significant and relevant topics. The following up is an overview of the major cell signaling pathway, MapK Pathway.

A mitogen-activated protein kinase (MAPK) cascade plays an important role in transduction of extracellular signals to cellular responses that regulates cell proliferation, differentiation and death [1]. In mammalian cells, the MAP kinase is characterized into three families: extracellular signal- regulated kinase (ERK), C-Jun-N- terminal kinase (JNK) and p38 kinase/ stress activated protein kinase (SAPK).Each cascade is initiated by specific extracellular signal leading to the activation of a particular MAPK followed by the successive activation of a MAPK kinase kinase (MAPKKK) and a MAPK kinase (MAPKK) [2 The MAPKKK is usually activated by interactions with a small GTPase and/or phosphorylation by protein kinases downstream from cell surface receptors. The MAPKK is directly phosphorylated and activated byMAPKKK, which in turn activates the MAPK by dual phosphorylation of a conserved tripeptide TxY motif in the activation segment.Upon activation, MAPK phosphorylates a variety of substrates in the cytoplasm and nucleus thus generating appropriate biological response. Activation of ERK is mediated by upstream regulators that include cell surface receptors, such as receptor tyrosine kinases (RTKs), G-protein-coupled receptors (GPCRs), and integrins, as well as the small GTPases,Ras and Rap. In mammalian cells, the chief role of the ERK pathway obtained from the studies of the Ras proteins that were first known as oncogenic proteins of tumor viruses that cause sarcomas in rats (hence the name Ras, from rat sarcoma virus) [3].Ras activation is mediated by guanine nucleotide exchange factors. Activation ofRas leads to activation of the Raf protein-serine/ threonine kinase, which phosphorylates and activates a second protein kinase called MEK (for MAP kinase/ ERK kinaase). Protein kinase C can also activate the ERK pathway, and both the Ca2+ and cAMP pathways cross with ERK signaling, either stimulating or inhibiting the ERK pathway in several kinds of cells ERK members of the family possess a TEY motif within the activation section and can be divided into two groups: the classic ERKs consisting primarily of a kinase domain (ERK 1 and ERK 2) and therefore the larger ERKs (such as ERK 5) that contain much additional extended sequence carboxy-terminal to their kinase domain.MAPKKs for the classic ERK1/2 module are MEK1 and MEK2, and therefore the MAPKKKs embody members of the Raf family, Mos, and Tpl2.

The JNK and p38 MAP kinase cascades are activated by the members of the Rho subfamily ofsmall GTP-binding proteins (together with Rae, Rho, and Cdc42) instead of using Ras. JNK family members contain TPY motif in the activation part and include JNK1, JNK2, and JNK3. The members of this family are activated in response to growth factors,environmental stress (heat, ionizing radiation, oxidative stress, and DNA damage) and inflammatory cytokines. The JNK module performs a crucial position in apoptosis, inflammation, cytokine production, and metabolism. The p38 family members contains TGY motif in the activation part and include subunits of p38- α, β, γ and δ. Like JNK family, p38 modules are preferentially activated by inflammatory cytokines and cellular stress (for example, ultraviolet irradiation).

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