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Role of mitogen- and stress-activated kinases in inflammatory arthritis

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Abstract

Lysophosphatidic acid (LPA) is a pleiotropic lipid mediator that promotes motility, survival, and the synthesis of chemokines/cytokines in human fibroblast-like synoviocytes (FLS) from patients with rheumatoid arthritis (RA). LPA activates several proteins within the MAP Kinase signaling network, including ERK1/2 and p38 MAPK. Upon docking to mitogen- and stress-activated kinases (MSKs), ERK1/2 and p38 MAPK phosphorylate serine and threonine residues within its C-terminal domain and cause autophosphorylation of MSKs. Activated MSKs can then directly phosphorylate CREB at Ser133 in FLS. Phosphorylation of CREB by MSKs is essential for the production of pro-inflammatory and anti-inflammatory cytokines. However, other downstream effectors of MSK1/2 such as NF- κ B, histone H3, and HMGN1 may also regulate gene expression in immune cells involved in disease pathogenesis. MSKs are master regulators of cell function that integrate signals induced by growth factors,

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