

p38 MAPK activation by VEGFR

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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This document contains 1 reaction (see Table of Contents)

p38 MAPK activation by VEGFR 7

Stable identifier: R-HSA-5218804

Type: omitted

Compartments: cytosol, extracellular region, plasma membrane



In primary cultured human umbilical vein endothelial cells (HUVECs) VEGF-induced activation of SAPK2/p38MAPK, and pharmacological inhibition of p38MAPK attenuated VEGF-induced cell migration (Rouseseau et al. 1997, 2000). The p38MAPK pathway conveys the VEGF signal to microfilaments inducing rearrangements of the actin cytoskeleton. These actin structures are thought to generate the contractile force within cells that is required for endothelial cell migration. Activation of p38 requires the activity of FYN and PAK2 (Lamalice et al. 2004). However, little is known of the exact molecular events that follow activation of PAK2 and lead to p38 activation. Like all MAP kinases, p38 MAP kinases are activated by dual kinases termed the MAP kinase kinases (MKKs). There are two main MAPKKs that are known to activate p38, MKK3 and MKK6 (Zarubin & Han 2005). Along with FYN and PAK these MKKs might contribute to the activation of p38. Activation of p38 resulted in activation of MAP kinase activated protein kinase 2/3 (MAPK 2/3) and phosphorylation of the F-actin polymerization modulator, heat shock protein 27 (HSP27) (Rousseau et al. 1997).

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Editions

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