p-S MAPK6,4 phosphorylate MAPKAPK5

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Introduction

Reactome is an 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


Reactome database release: 82

This document contains 1 reaction (see Table of Contents)
Activated MAPK6 and MAPK4 promote the phosphorylation of MAPKAPK5 on threonine 182, activating it (Deleris et al, 2008; Aberg et al, 2006; Aberg et al, 2009; Seternes et al, 2004; Perander et al, 2008). Thr182 phosphorylation may result in part from autophosphorylation stimulated by MAPK6 binding, rather than direct phosphorylation by MAPK6, as an ATP-binding pocket mutant of MAPKAPK5 is not phosphorylated in response to MAPK6 (Seternes et al, 2004; Schumacher et al, 2004). There is conflicting evidence as to whether a catalytically inactive MAPK6 mutant can promote MAPKAPK5 phosphorylation (Schumacher et al, 2004; Seternes et al, 2004; Deleris et al, 2008). These conflicting results can be reconciled by the suggestion that inactive MAPK6 promotes MAPKAPK5 phosphorylation through heterodimerization with active MAPK4 (Kant et al, 2006). Phosphorylation of MAPKAPK5 in response to MAPK4/6 signaling promotes its cytoplasmic relocalization (Shumacher et al, 2004; Aberg et al, 2006; Deleris et al, 2008; Seternes et al, 2004).

**Literature references**


**Editions**

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