

CDK1 phosphorylates MAPK6

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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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Reactome database release: 88

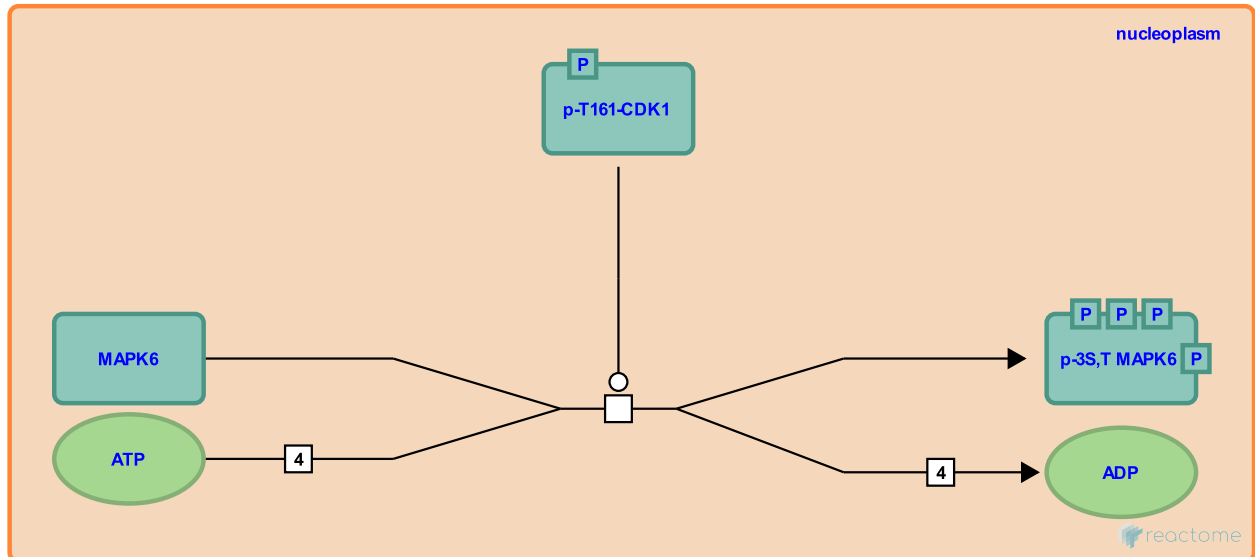
This document contains 1 reaction ([see Table of Contents](#))

CDK1 phosphorylates MAPK6 [↗](#)

Stable identifier: R-HSA-5692755

Type: transition

Compartments: nucleoplasm



MAPK6 is hyperphosphorylated by CDK1 at multiple sites in the C-terminal extension, and this phosphorylation is associated with the stabilization of MAPK6 protein in mitosis. Residues S684, S688, T698 and S705 have been identified as in vitro targets of CDK1, and phosphorylation of T698 has also been demonstrated in vivo (Tanguay et al, 2010). The role of hyperphosphorylated MAPK6 during mitosis has not been established, and although the CDK1-dependent phosphorylation of MAPK6 is depicted as occurring in the nucleus, the site of action has also not been determined. CDK1-dependent hyperphosphorylation of the C-terminal tail is reversed by the phosphatases CDC14A and B (Tanguay et al, 2010; Hansen et al, 2008).

Literature references

Meloche, S., Rodier, G., Tanguay, PL. (2010). C-terminal domain phosphorylation of ERK3 controlled by Cdk1 and Cdc14 regulates its stability in mitosis. *Biochem. J.*, 428, 103-11. [↗](#)

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Editions

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