Myosin phosphatase dephosphorylates

PLK1

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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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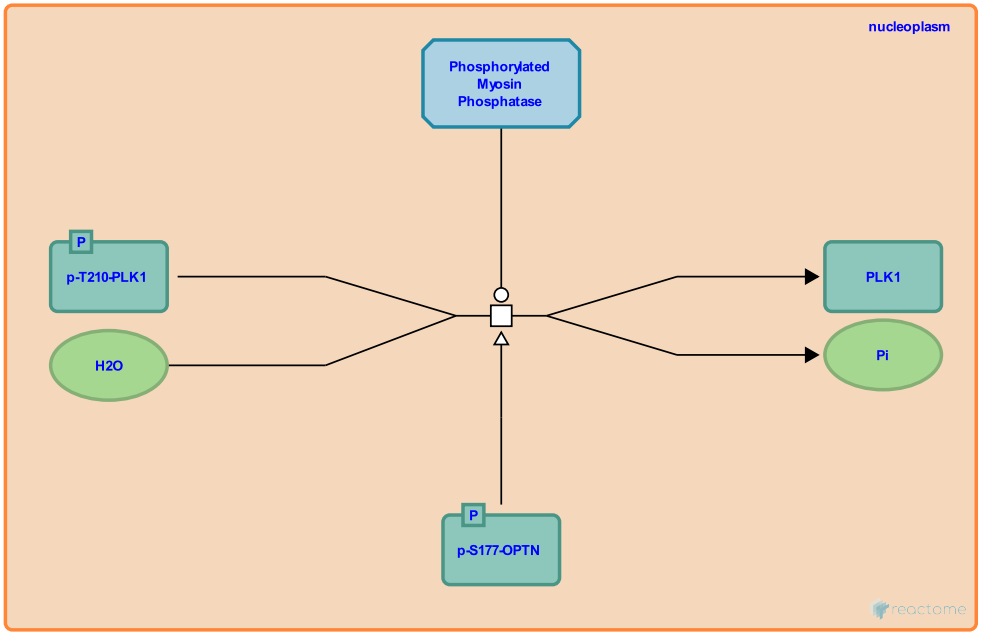
Stable identifier: R-HSA-3002811

Type: transition

Compartments: nucleoplasm



The myosin phosphatase complex can dephosphorylate PLK1 threonine residue T210 and inactivate PLK1 (Yamashiro et al. 2008). Myosin phosphatase is activated through phosphorylation of its PPP1R12A (MYPT1) subunit. Several kinases, including CDK1 (Yamashiro et al. 2008) and LATS1 (Chiyoda et al. 2012) have been implicated in myosin phosphatase activation, but the position and temporal order of key PPP1R12A phosphorylations need to be investigated further. Phosphorylated OPTN (optineurin) is able to bind PPP1R12A (MYPT1) and positively regulates PLK1 dephosphorylation by myosin phosphatase, posibly by facilitating PPP1R12A phosphorylation and myosin phosphatase activation (Kachaner et al. 2012).

Literature references

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Editions

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