

# PLK1 phosphorylates NEK9

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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

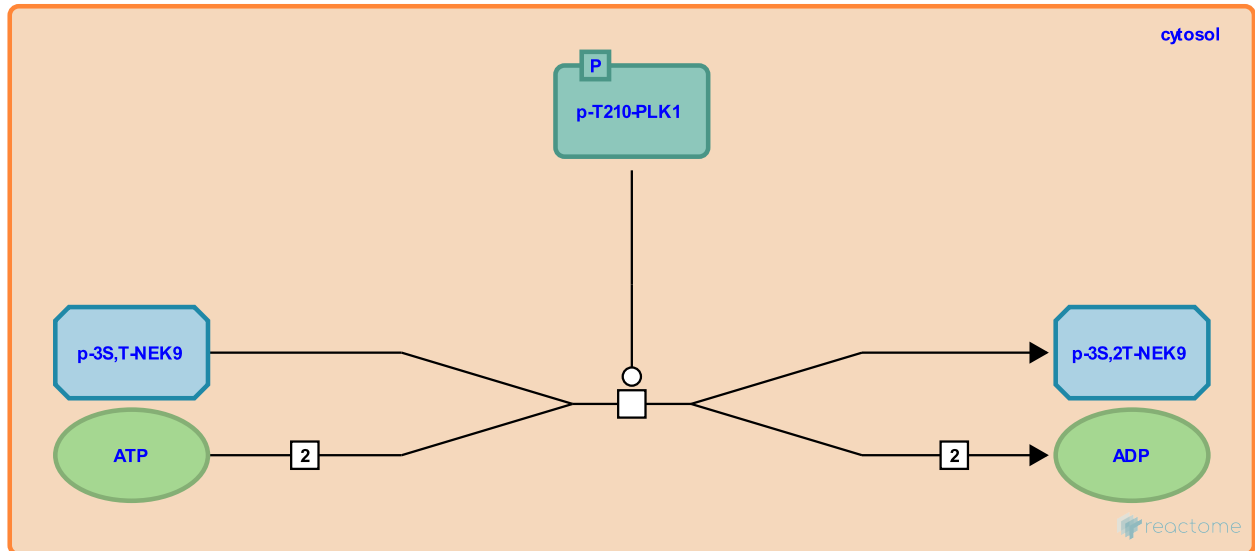
This document contains 1 reaction ([see Table of Contents](#))

## PLK1 phosphorylates NEK9 [↗](#)

**Stable identifier:** R-HSA-2984226

**Type:** transition

**Compartments:** cytosol



NEK9 serine residues S29, S750 and S869, which are likely targets of CDK1:CCNB-mediated phosphorylation in mitosis, can be recognized by the polo-box domain (PBD) of PLK1 when phosphorylated. Phosphorylation of S869 appears to be crucial for the interaction of NEK9 and PLK1 (Bertran et al. 2011). PLK1 phosphorylates threonine T210 of NEK9 in vitro. T210 is located in the kinase activation loop of NEK9 and T210 phosphorylation is necessary for NEK9 kinase activity. While T210 can be autophosphorylated in vitro, when NEK9 is incubated in the presence of excess ATP and Mg<sup>2+</sup> (Roig et al. 2005), mitotic phosphorylation of T210 requires both CDK1 and PLK1 activity (Bertran et al. 2011).

### Literature references

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### Editions

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