CASP9 is phosphorylated at T412

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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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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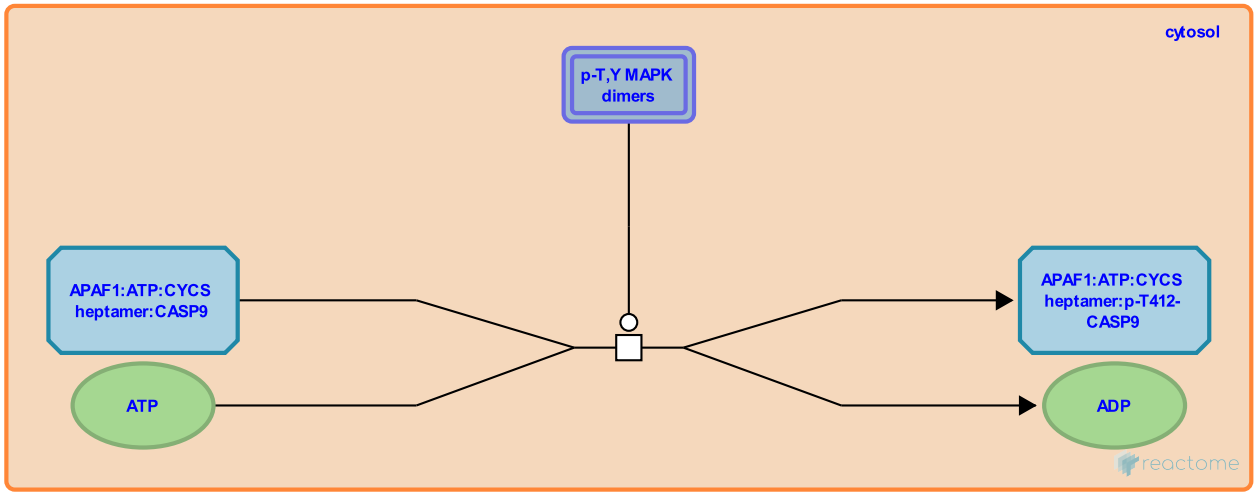
Stable identifier: R-HSA-9627089

Type: transition

Compartments: cytosol



Phosphorylation of caspase 9 (CASP9) may contribute to the suppression of apoptosis. A major inhibitory phosphorylation site in CASP9 is Thr412, which forms part of a Thr-Pro motif (Allan LA & Clarke PR 2007; Martin MC et al. 2008). This motif is targeted by multiple proline-directed kinases such as ERK1/2 in response to extracellular growth/survival signals or CDK1-cyclin B1 during mitosis (Allan LA et al. 2003; Allan LA & Clarke PR 2007; Martin MC et al. 2008). Thr412 is also phosphorylated by DYRK1A, which regulates apoptosis during development (Seifert A et al. 2008).

Literature references

Allan, LA., Clarke, PR., Mancini, EJ., Martin, MC. (2008). The docking interaction of caspase-9 with ERK2 provides a mechanism for the selective inhibitory phosphorylation of caspase-9 at threonine 125. *J. Biol. Chem., 283*, 3854-65 . ↗

Editions

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