Calpain 1 or Calpain 2 cleaves Cdk5r1 (p35)

Orlic-Milacic, M., Shah, K.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

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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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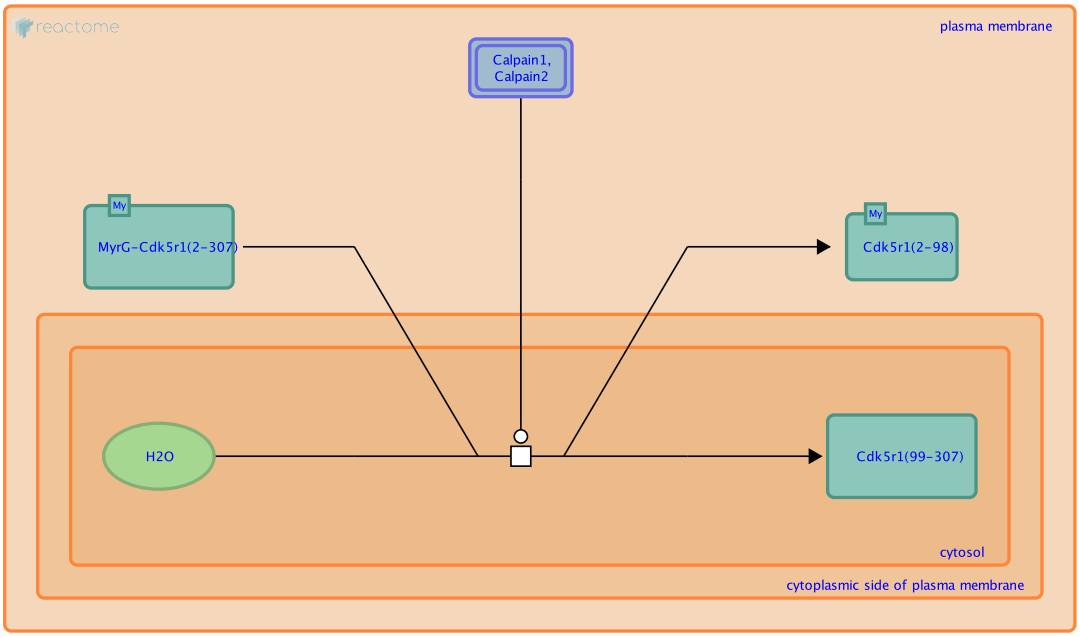
Stable identifier: R-MMU-8863416

Type: transition

Compartments: cytosol, plasma membrane



In mouse brain, neurotoxicity induces increase in neuronal cell calcium (Ca2+) levels, triggering calpain activation and calpain-mediated cleavage of p35 (Cdk5r1) to produce the cytosolic p25 fragment (Lee et al. 2000). Mouse Cdk5r1 shows 98% sequence identity to the human ortholog, and the calpain complex subunits are also highly conserved between the two species. Two calpain catalytic subunits expressed in neurons, Capn1 (mu-calpain) and Capn2 (M-calpain) are 89% and 94% identical, respectively, to human counterparts. The regulatory subunits Capns1 and Capns2 are 94% and 92% identical to human proteins, respectively.

Literature references

Lee, MS., Kwon, YT., Li, M., Peng, J., Friedlander, RM., Tsai, LH. (2000). Neurotoxicity induces cleavage of p35 to p25 by calpain. *Nature, 405*, 360-4.

Editions

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