CDK5:p25 phosphorylates lamin A

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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CDK5:p25 phosphorylates lamin A ↗

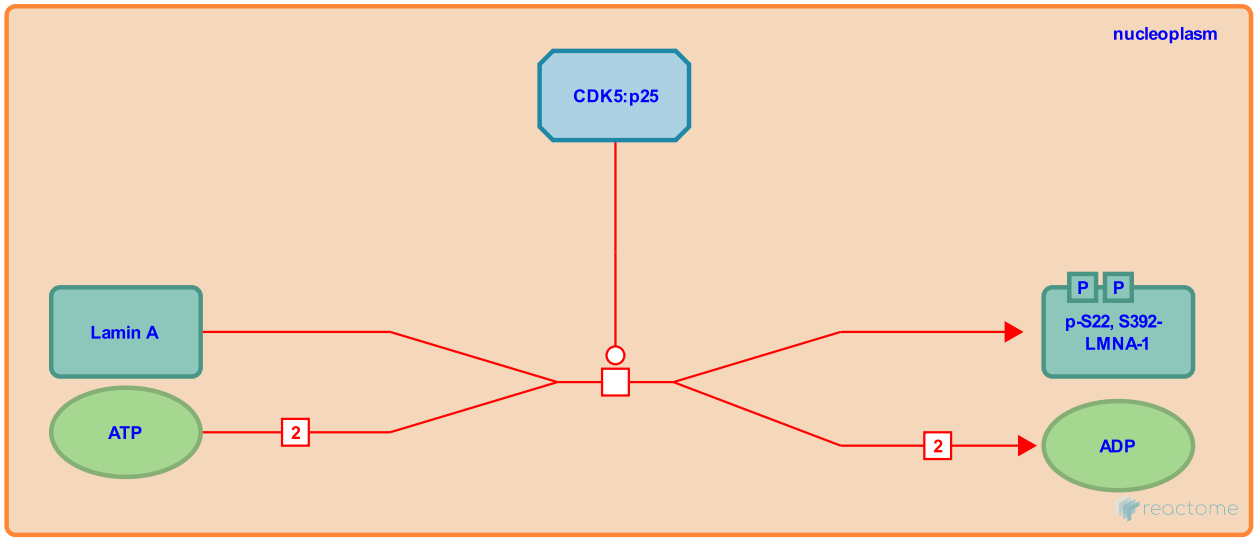
Stable identifier: R-HSA-8868344

Type: transition

Compartments: nucleoplasm

Diseases: Alzheimer's disease

Inferred from: Cdk5:p25 phosphorylates lamin A (Mus musculus)



Alzheimer's disease (AD), like many other neurodegenerative diseases, is characterized by nuclear envelope fragmentation. Based on a mouse AD model, nuclear fragmentation is initiated by phosphorylation of nuclear lamins by p25-activated CDK5. The CDK5:p25 complex phosphorylates lamin A (LMNA-1) at serine residues S22 and S392, with S392 being the major CDK5 target site. Nuclear envelope fragmentation increases access of the CDK5:p25 complex to nuclear proteins and precedes neuronal death (Chang et al. 2011).

Editions

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