CDK5 binds p25

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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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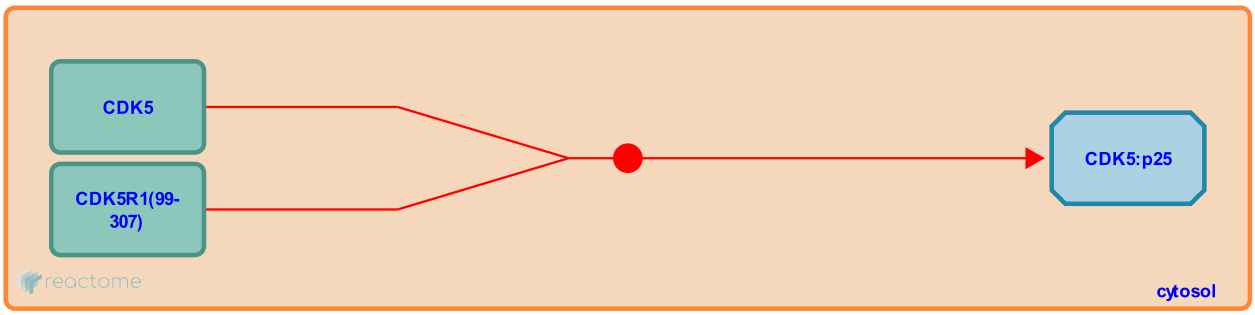
CDK5 binds p25

Stable identifier: R-HSA-8863013

Type: binding

Compartments: cytosol

Diseases: Alzheimer's disease



p25 has a 5-10 fold longer half-life compared to p35 and lacks the membrane anchoring signal, which results in its constitutive activation and mislocalization of the CDK5:p25 complex to the cytoplasm and the nucleus (Patrick et al. 1999). As CDK5 deregulation triggers nuclear envelope dispersion (Chang et al. 2011), with timing being uncertain, all phosphorylation events catalyzed by the CDK5:p25 complex are shown to occur in the cytosol.

Literature references

Dikkes, P., Patrick, GN., Nikolic, M., Zukerberg, L., Tsai, LH., de la Monte, S. (1999). Conversion of p35 to p25 deregulates Cdk5 activity and promotes neurodegeneration. *Nature, 402*, 615-22.

Editions

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