Evasion of Oxidative Stress Induced Senescence Due to Defective p16INK4A binding to CDK4 and CDK6

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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


Reactome database release: 72

This document contains 1 pathway and 1 reaction (see Table of Contents)
Evasion of Oxidative Stress Induced Senescence Due to Defective p16INK4A binding to CDK4 and CDK6

Stable identifier: R-HSA-9632700

Diseases: cancer

Missense and nonsense mutations in the CDKN2A gene that result in amino acid substitutions in p16INK4A or p16INK4A truncations, respectively, impairing its ability to bind to CDK4 and CDK6, interfere with p16INK4A-mediated induction of cellular senescence in response to oxidative stress (Chen 2000, Vurusaner et al. 2012).

Loss-of-function mutations in p16INK4A can also contribute to cancer by interfering with p16INK4A-mediated inhibition of NFKB signaling (Becker et al. 2005).

Literature references


Editions

<table>
<thead>
<tr>
<th>Date</th>
<th>Action</th>
<th>Author</th>
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</thead>
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p16INK4A mutants do not bind CDK4, CDK6

Location: Evasion of Oxidative Stress Induced Senescence Due to Defective p16INK4A binding to CDK4 and CDK6

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