

RB1 binds condensin II

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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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Reactome database release: 88

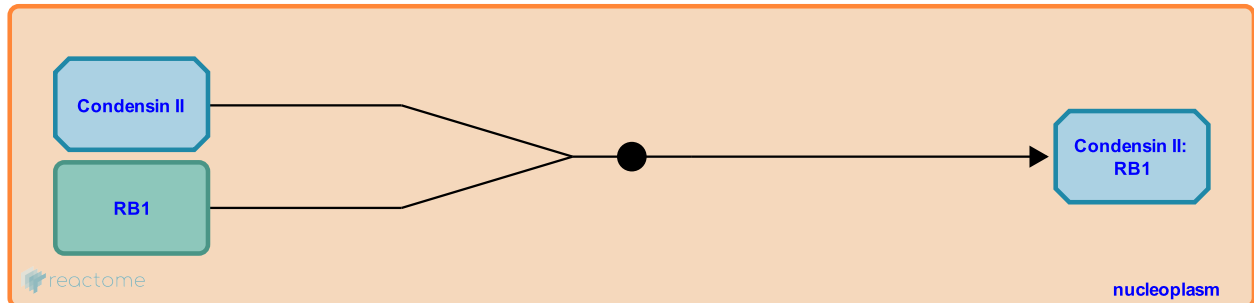
This document contains 1 reaction ([see Table of Contents](#))

RB1 binds condensin II [↗](#)

Stable identifier: R-HSA-2172666

Type: binding

Compartments: nucleoplasm



RB1 binds the condensin II complex through interaction with the NCAPD3 subunit of condensin II. This interaction is E2F independent and is important for targeting of the condensin II complex to chromatin (Longworth et al. 2008). RB1 may be particularly important for targeting of the condensin II complex to centromeres (Manning et al. 2010). RB1 deficient cells exhibit chromosome condensation defects and are prone to aneuploidy caused by aberrant chromosomal segregation. Therefore, tumor suppressor role of RB1 is based both on E2F-dependent control of G1/S transition, as well as on E2F-independent maintenance of genomic stability through regulation of mitotic chromosome condensation (Longworth et al. 2008, Coschi et al. 2010, Manning et al. 2010).

The role of RB1 in the maintenance of genomic stability is supported by studies of the childhood eye cancer retinoblastoma and its precursor, retinoma. Retinoma, a quiescent precursor of malignant retinoblastoma with functional loss of both RB1 alleles, is genomically unstable (Dimaras et al. 2008). Also, while the majority of retinoblastoma tumors are caused by the loss-of-function of the tumor suppressor gene RB1, ~2% of retinoblastoma tumors in unilaterally affected patients are initiated by a high level amplification of MYCN gene, in the presence of two functional, unmutated RB1 alleles. These tumors, with normal RB1 and amplified MYCN show a much lower level of genomic instability than retinoblastoma tumors with RB1 loss-of-function (Rushlow et al. 2013).

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Editions

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