RB1 binds and inhibits E2F1/2/3:DP1/2 complexes

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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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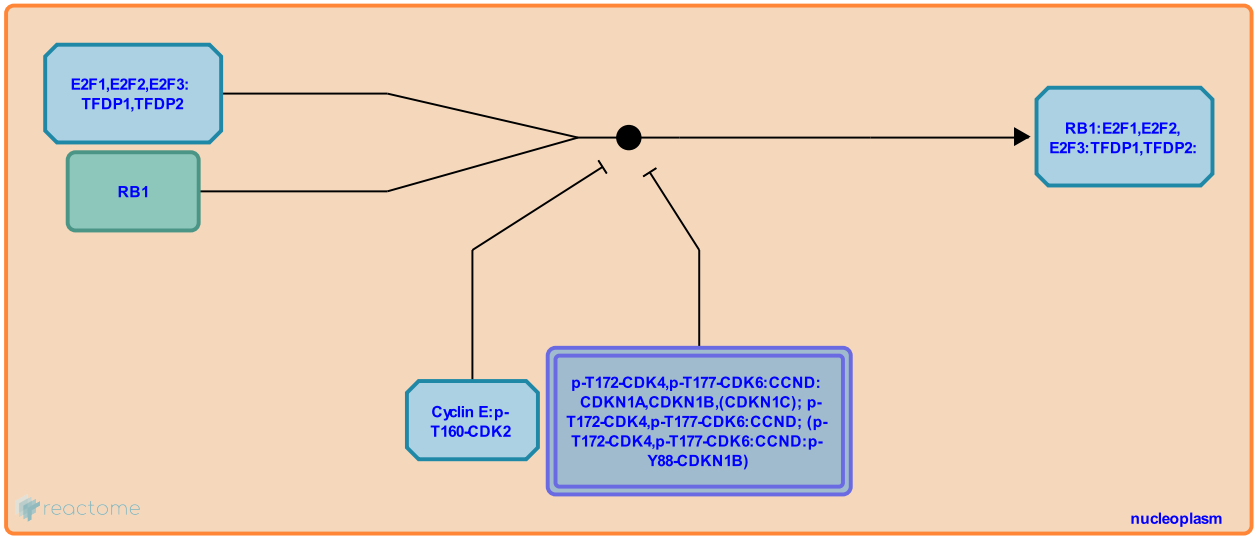
Stable identifier: R-HSA-9018017

Type: binding

Compartments: nucleoplasm



RB1 tumor suppressor, the product of the retinoblastoma susceptibility gene, binds to E2F transcription factors E2F1, E2F2 and E2F3, presumably heterodimerized with TFDP1 or TFDP2. The interaction involves the pocket domain of RB1. RB1 binding inhibits transcriptional activity of E2F1/2/3:TFDP1/2 complexes, resulting in prevention of G1/S transition (Chellappan et al. 1991, Bagchi et al. 1991, Chittenden et al. 1991, Lees et al. 1993, Hiebert 1993, Wu et al. 2001). Once RB1 is phosphorylated on serine residue S795 by Cyclin D:CDK4/6 complexes, it can no longer associate with and inhibit E2F1/2/3:TFDP1/2 complexes. Thus, CDK4/6-mediated phosphorylation of RB1 leads to transcriptional activation of E2F1/2/3 target genes needed for the S phase of the cell cycle (Connell-Crowley et al. 1997).

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Editions

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