APC truncation mutants have impaired AX-IN binding

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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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Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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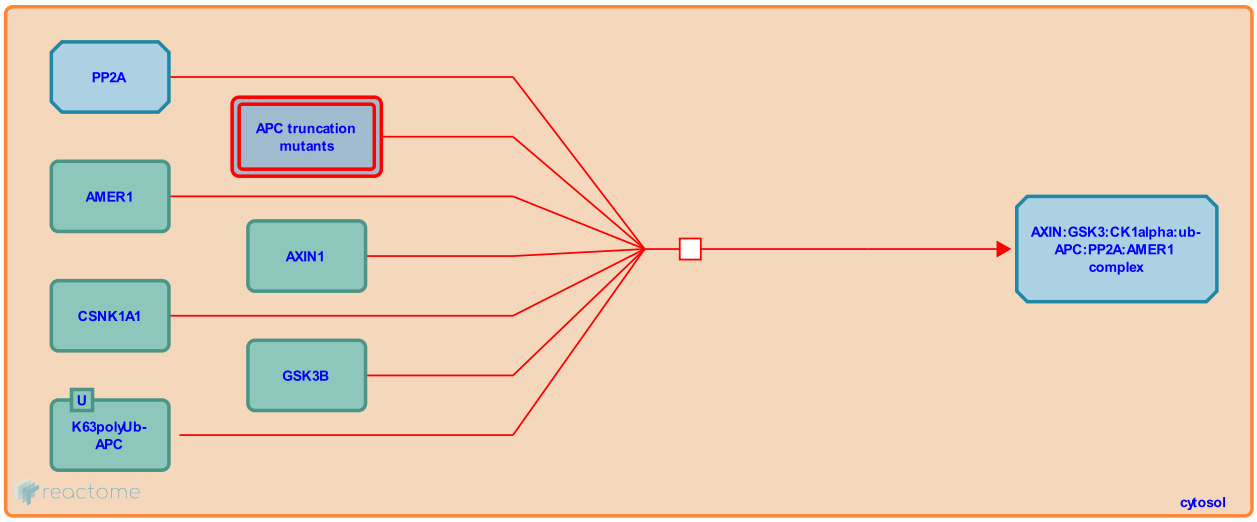
APC truncation mutants have impaired AXIN binding ↗

Stable identifier: R-HSA-4791278

Type: transition

Compartments: cytosol

Diseases: colorectal cancer, cancer



The tumor suppressor APC is a central component of the destruction complex that makes direct, functionally important contacts with a number of other destruction complex members, including AXIN, beta-catenin and GSK3 (Behrens et al, 1998; Kishida et al, 1998; Hart et al, 1998; Rubinfeld et al, 1993; Su et al, 1993; Rubinfeld et al, 1996). These protein-protein interactions are critical to APC's role in promoting the proteasome-mediated degradation of beta-catenin, and are abrogated by APC truncations that are common in the vast majority of colorectal cancers (reviewed in Saito-Diaz et al, 2013; Polakis, 2000; Polakis, 2012). Cancer cells with expressing truncated forms of APC have high levels of free beta-catenin and aberrant WNT target gene expression (Morin et al, 1997; Shih et al, 2000; Roh et al, 2001).

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Editions

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