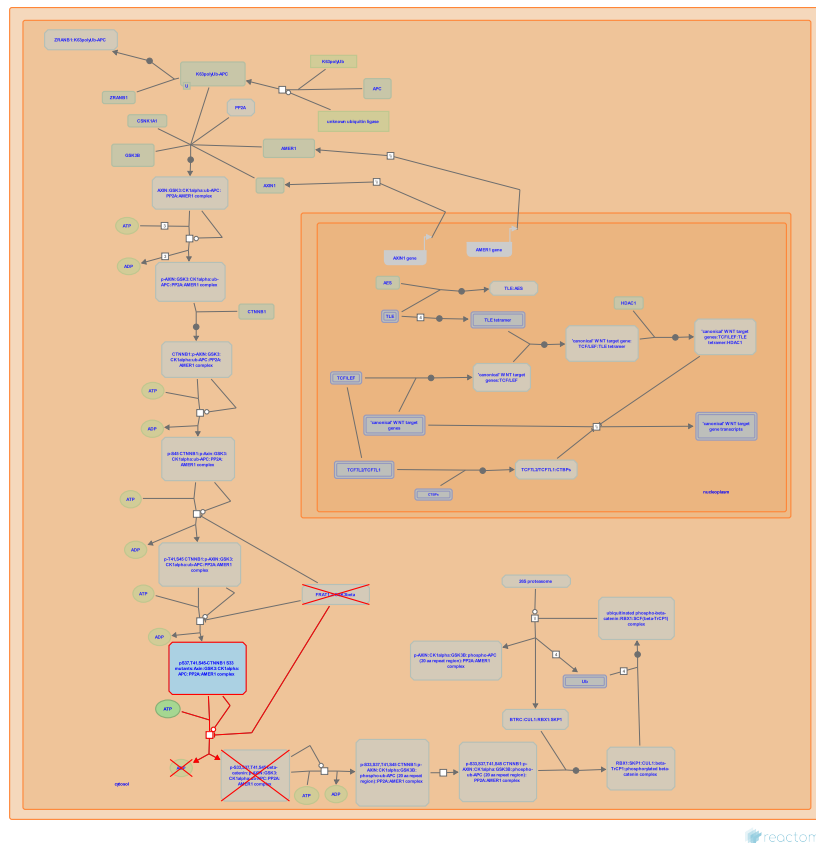


CTNNB1 S33 mutants aren't phosphorylated



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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the [Reactome Textbook](https://reactome.org/textbook/).

30/04/2024

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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Reactome database release: 88

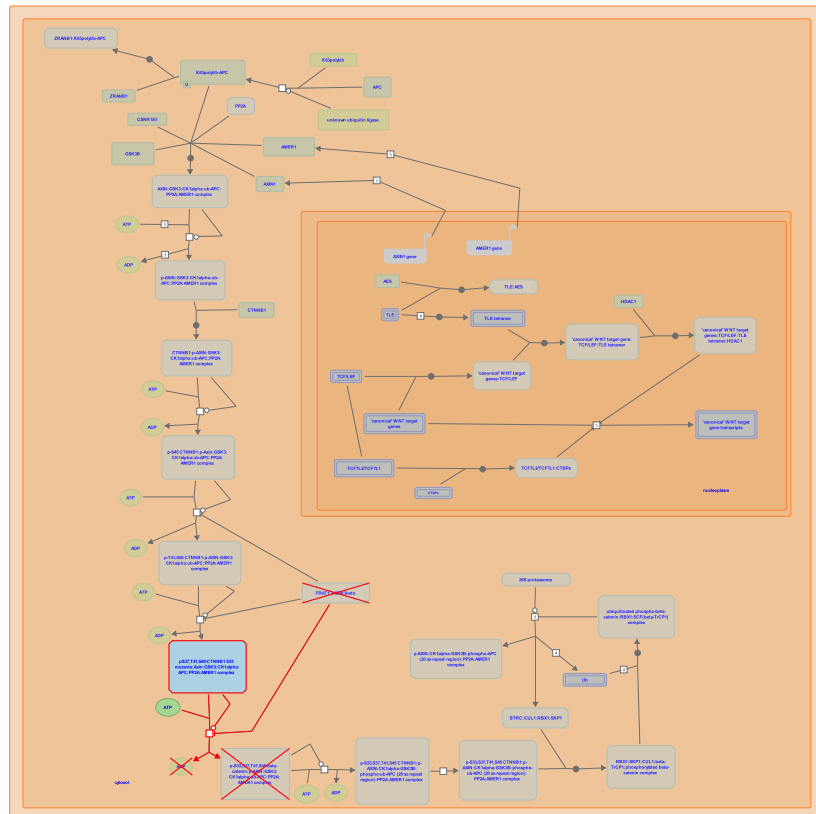
This document contains 1 pathway and 1 reaction ([see Table of Contents](#))

CTNNB1 S33 mutants aren't phosphorylated ↗

Stable identifier: R-HSA-5358747

Compartments: cytosol

Diseases: cancer



S33 mutations of beta-catenin interfere with GSK3 phosphorylation and result in stabilization and nuclear localization of the protein and enhanced WNT signaling (Groen et al, 2008; Nhieu et al, 1999; Clements et al, 2002; reviewed in Polakis, 2000). S33 mutations have been identified in cancers of the central nervous system, liver, endometrium and stomach, among others (reviewed in Polakis, 2000).

Literature references

Zafrani, ES., Renard, CA., Wei, Y., Cherqui, D., Nhieu, JT., Buendia, MA. (1999). Nuclear accumulation of mutated beta-catenin in hepatocellular carcinoma is associated with increased cell proliferation. *Am. J. Pathol.*, 155, 703-10. ↗

Polakis, P. (2000). Wnt signaling and cancer. *Genes Dev.*, 14, 1837-51. ↗

Schilder-Tol, EJ., Spaargaren, M., Oud, ME., Overdijk, MB., Pals, ST., Groen, RW. et al. (2008). Illegitimate WNT pathway activation by beta-catenin mutation or autocrine stimulation in T-cell malignancies. *Cancer Res.*, 68, 6969-77. ↗

Sarnaik, A., Wang, J., Lowy, AM., MacDonald, J., Kim, OJ., Fenoglio-Preiser, C. et al. (2002). beta-Catenin mutation is a frequent cause of Wnt pathway activation in gastric cancer. *Cancer Res.*, 62, 3503-6. ↗

Editions

2014-04-02	Authored	Rothfels, K.
2014-04-03	Edited	Matthews, L.
2014-05-12	Reviewed	Salahshor, S.
2014-05-22	Reviewed	Woodgett, J.

CTNNB1 S33 mutants aren't phosphorylated by GSK3beta ↗

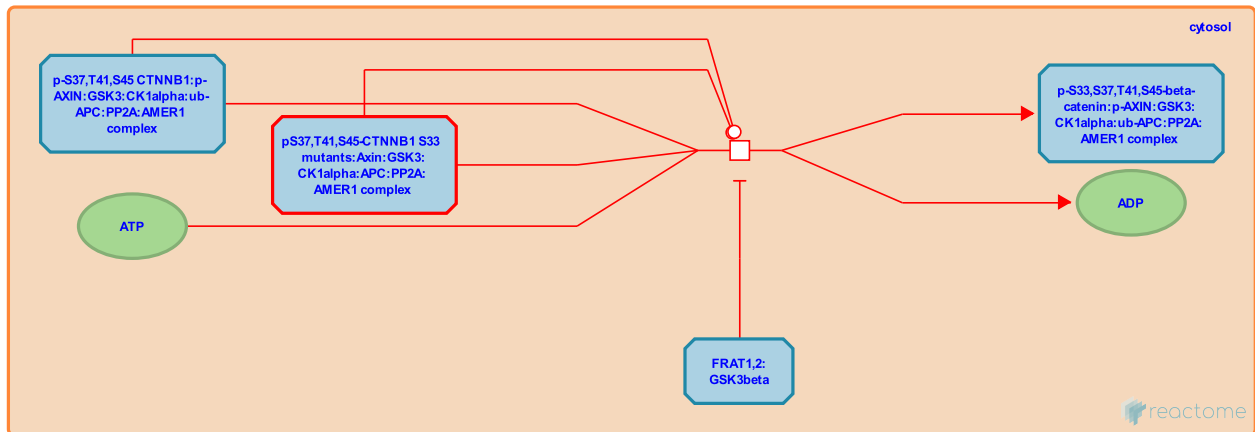
Location: CTNNB1 S33 mutants aren't phosphorylated

Stable identifier: R-HSA-4839634

Type: transition

Compartments: cytosol

Diseases: cancer



S33 mutations of beta-catenin interfere with GSK3 phosphorylation and result in stabilization and nuclear localization of the protein and enhanced WNT signaling (Groen et al, 2008; Nhieu et al, 1999; Clements et al, 2002; reviewed in Polakis, 2000). S33 mutations have been identified in cancers of the central nervous system, liver, endometrium and stomach, among others (reviewed in Polakis, 2000).

Literature references

- Zafrani, ES., Renard, CA., Wei, Y., Cherqui, D., Nhieu, JT., Buendia, MA. (1999). Nuclear accumulation of mutated beta-catenin in hepatocellular carcinoma is associated with increased cell proliferation. *Am. J. Pathol.*, 155, 703-10. ↗
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- Schilder-Tol, EJ., Spaargaren, M., Oud, ME., Overdijk, MB., Pals, ST., Groen, RW. et al. (2008). Illegitimate WNT pathway activation by beta-catenin mutation or autocrine stimulation in T-cell malignancies. *Cancer Res.*, 68, 6969-77. ↗
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