CTNNB1 S37 mutants aren't phosphorylated by GSK3beta

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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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

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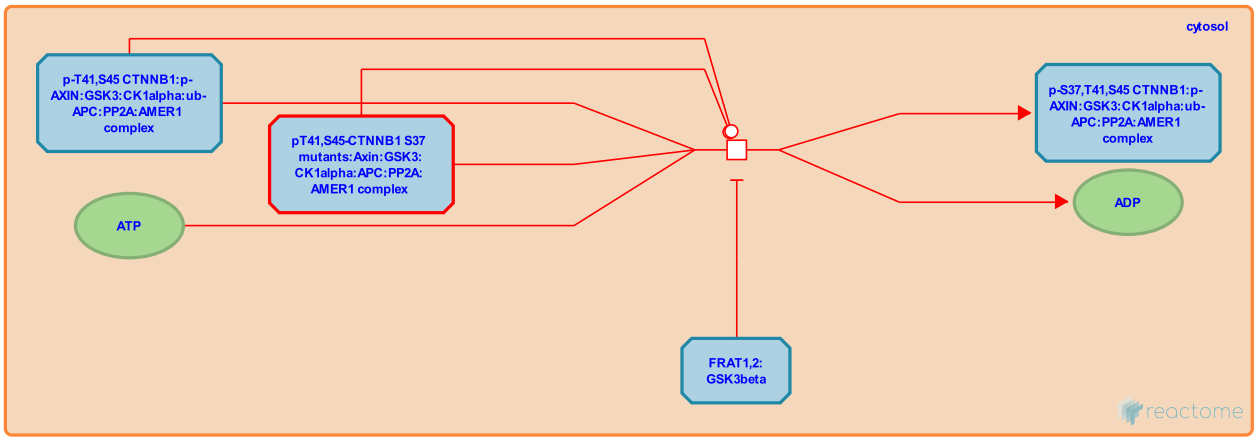
Stable identifier: R-HSA-4839635

Type: transition

Compartments: cytosol

Diseases: cancer



S37 mutations of beta-catenin interfere with GSK3 phosphorylation and stabilize the protein, resulting in enhanced WNT pathway signaling (Nhieu et al, 1999; Clements et al, 2002; reviewed in Polakis, 2000). S37 mutations have been identified in cancers of the brain, liver, ovary and large intestine, 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. ↗

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

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