

Phosphorylation of phospho-(Ser45) at Thr 41 by GSK-3

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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. [↗](#)

Reactome database release: 88

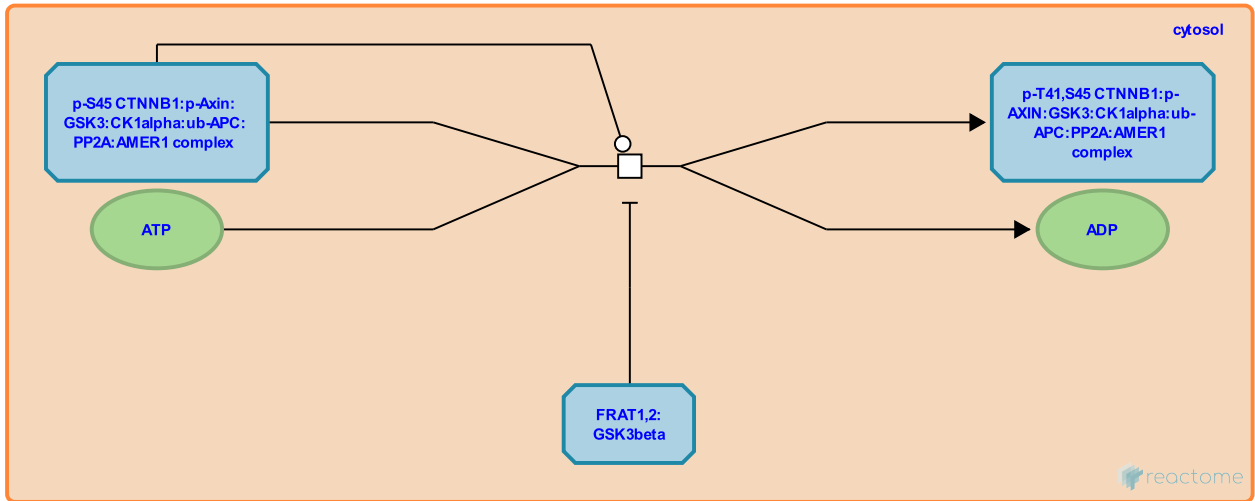
This document contains 1 reaction ([see Table of Contents](#))

Phosphorylation of phospho-(Ser45) at Thr 41 by GSK-3 ↗

Stable identifier: R-HSA-195287

Type: transition

Compartments: cytosol



Following CKI-mediated phosphorylation at Ser45, beta-catenin is phosphorylated by GSK3 at Thr41.

Literature references

Zhang, Z., Baeg, GH., Lin, X., Tan, Y., Li, Y., He, X. et al. (2002). Control of beta-catenin phosphorylation/degradation by a dual-kinase mechanism. *Cell*, 108, 837-47. ↗

Editions

2007-04-03	Authored	Kimelman, D.
2007-04-03	Edited	Matthews, L.
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