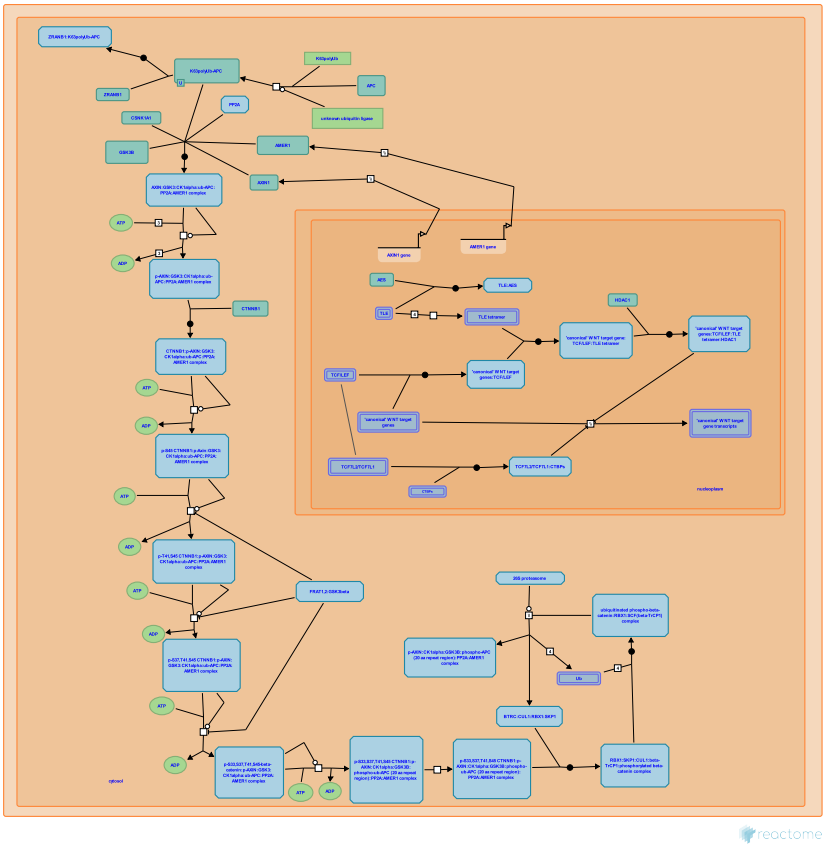
Degradation of beta-catenin by the de-

struction complex



Angers, S., Jupe, S., Kimelman, D., Matthews, L., Meldal, BH., Pagano, M., Rajakulendran, N., Rothfels, K., Salahshor, S., Woodgett, J.

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25/04/2024

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.
The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

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. ↗

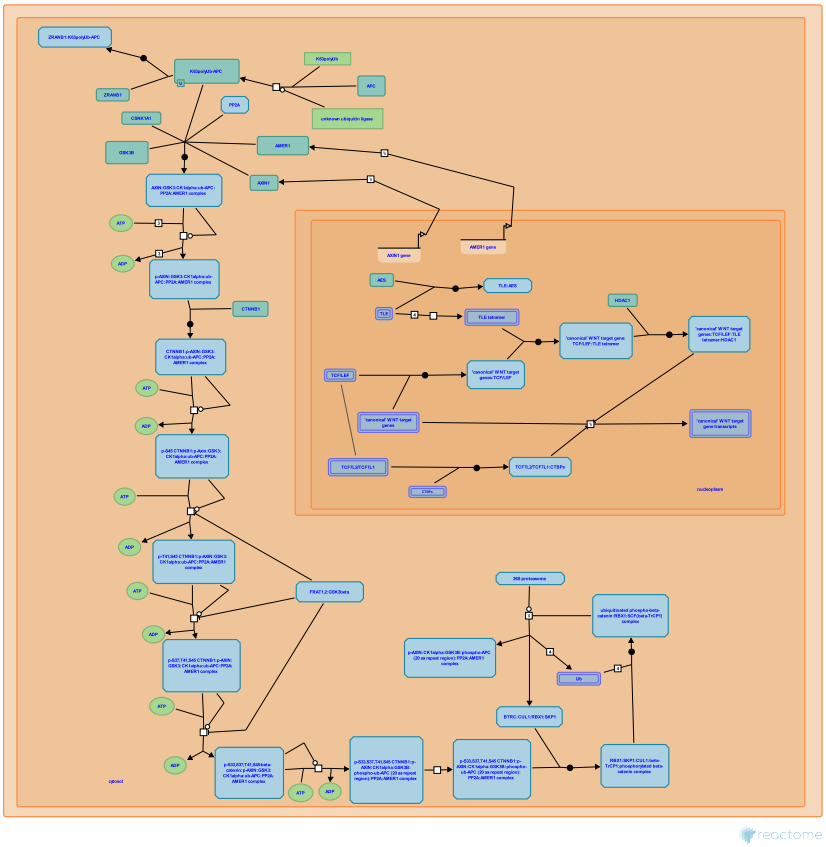
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

Degradation of beta-catenin by the destruction complex ↗

Stable identifier: R-HSA-195253



The beta-catenin destruction complex plays a key role in the canonical Wnt signaling pathway. In the absence of Wnt signaling, this complex controls the levels of cytoplamic beta-catenin. Beta-catenin associates with and is phosphorylated by the destruction complex. Phosphorylated beta-catenin is recognized and ubiquitinated by the SCF-beta TrCP ubiquitin ligase complex and is subsequently degraded by the proteasome (reviewed in Kimelman and Xu, 2006).

Literature references

Kimelman, D., Xu, W. (2006). beta-catenin destruction complex: insights and questions from a structural perspective . *Oncogene, 25*, 7482-91. ↗

Editions

2007-04-03	Authored	Kimelman, D.
2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.

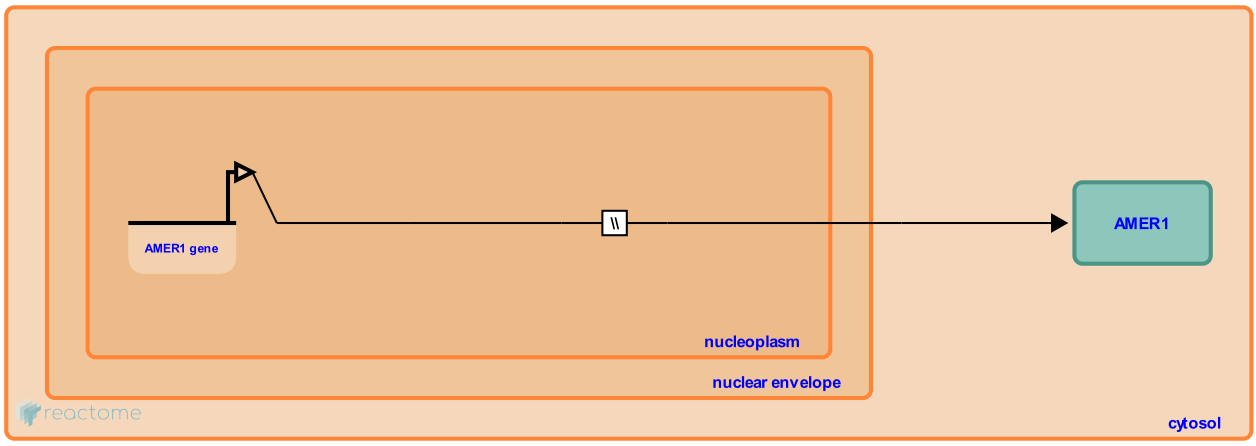
Expression of AMER1 gene ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-5251557

Type: omitted

Compartments: nucleoplasm, cytosol



AMER1 was identified as a gene mutated in a subset of Wilms tumors (Rivera et al, 2007) and the protein has been shown to be a component of the beta-catenin destruction complex (Major et al, 2007).

Followed by: Assembly of the destruction complex

Literature references

Berndt, JD., Major, MB., Maccoss, MJ., Yi, X., Camp, ND., Biechele, TL. et al. (2007). Wilms tumor suppressor WTX negatively regulates WNT/beta-catenin signaling. *Science, 316*, 1043-6. ↗

Iafrate, AJ., Wells, J., Rivera, MN., Chin, L., Han, M., Vargas, SO. et al. (2007). An X chromosome gene, WTX, is commonly inactivated in Wilms tumor. *Science, 315*, 642-5. ↗

Editions

2014-01-23	Authored	Rothfels, K.
2014-04-03	Edited	Matthews, L.
2014-05-12	Reviewed	Salahshor, S.
2014-05-22	Reviewed	Woodgett, J.

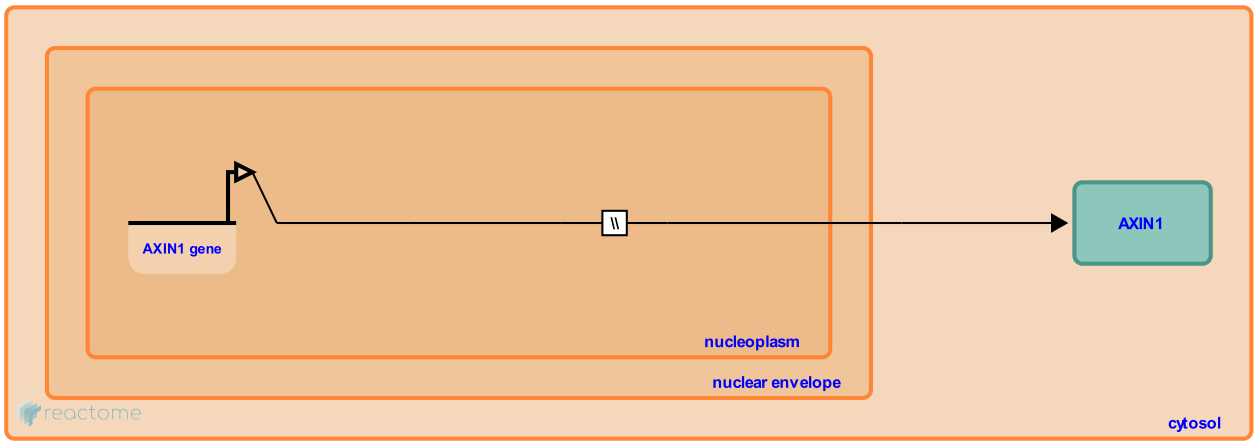
Expression of AXIN1 gene ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-5251555

Type: omitted

Compartments: nucleoplasm, cytosol



AXIN1 was first identified as the product of the mouse gene fused and has since been shown to have a key role in the degradation of beta-catenin by the destruction complex (Zeng et al, 1997; reviewed in Saito-Diaz et al, 2013). Deletion, missense and nonsense mutations that lead to activated WNT signaling have been identified in the AXIN1 gene in human cancers, making AXIN1 a tumor suppressor gene (reviewed in Salahshor and Woodgett, 2005).

Followed by: Assembly of the destruction complex

Literature references

Salahshor, S., Woodgett, JR. (2005). The links between axin and carcinogenesis. *J. Clin. Pathol., 58*, 225-36. ↗

Wang, X., Wallace, HA., Page-McCaw, A., Lee, E., Thorne, CA., Chen, TW. et al. (2013). The way Wnt works: Components and mechanism. *Growth Factors, 31*, 1-31. ↗

Gumbiner, BM., Hsu, W., Costantini, F., Zhang, T., Lee, JJ., Vasicek, TJ. et al. (1997). The mouse Fused locus encodes Axin, an inhibitor of the Wnt signaling pathway that regulates embryonic axis formation. *Cell, 90*, 181-92. ↗

Editions

2014-01-23	Authored	Rothfels, K.
2014-04-03	Edited	Matthews, L.
2014-05-12	Reviewed	Salahshor, S.
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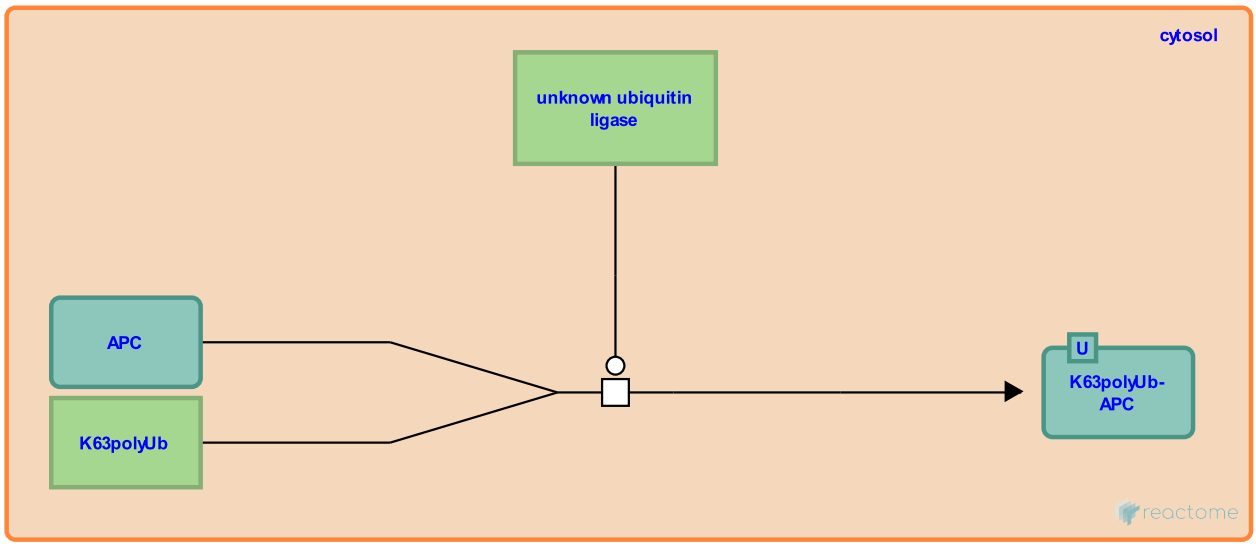
APC is K63-polyubiquitinated ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-5246693

Type: transition

Compartments: cytosol



In unstimulated cells, APC is K63 polyubiquitinated in a manner that depends on its association with AXIN. Although the precise timing of APC polyubiquitination is unclear, it is disrupted by abrogation of GSK3 kinase activity and in the presence of phosphodegron mutants of beta-catenin, suggesting that the formation of a functional destruction complex is required. Destruction complex formation is also dependent upon AXIN levels, which may be regulated at least in part by the balance of its ubiquitination and sumoylation (Kim et al, 2008).
Upon WNT3A stimulation, APC K63 polyubiquitination is lost coincident with disruption of the APC-AXIN interaction (Tran and Polakis, 2012). Interestingly, another study has shown that DVL is K63 polyubiquitinated upon WNT signaling (Tauriello et al, 2010), suggesting a possible model in which WNT signaling promotes a change in AXIN-K63 polyubiquitin binding partner to destabilize the destruction complex and promote pathway activation. Alternately, APC K63 polyubiquitination may protect beta-catenin from PP2A-mediated dephosphorylation and thus favour its degradation (Su et al, 2008).

Followed by: Assembly of the destruction complex

Literature references

Polakis, P., Tran, H. (2012). Reversible modification of adenomatous polyposis coli (APC) with K63-linked polyubiquitin regulates the assembly and activity of the ?-catenin destruction complex. *J. Biol. Chem., 287*, 28552-63. ↗

Maurice, MM., Canninga-van Dijk, MR., Henraat, M., Clevers, HC., Kessler, BM., Edelmann, MJ. et al. (2010). Loss of the tumor suppressor CYLD enhances Wnt/beta-catenin signaling through K63-linked ubiquitination of Dvl. *Mol. Cell, 37*, 607-19. ↗

Costantini, F., Kim, MJ., Chia, IV. (2008). SUMOylation target sites at the C terminus protect Axin from ubiquitination and confer protein stability. *FASEB J., 22*, 3785-94. ↗

Stella, A., Liu, B., Su, Y., Ishikawa, S., Kojima, M., Schreiber, EM. et al. (2008). APC is essential for targeting phosphorylated beta-catenin to the SCFbeta-TrCP ubiquitin ligase. *Mol. Cell, 32*, 652-61. ↗

Editions

2014-01-17	Authored	Rothfels, K.
2014-04-03	Edited	Matthews, L.
2014-05-12	Reviewed	Salahshor, S.
2014-05-22	Reviewed	Woodgett, J.

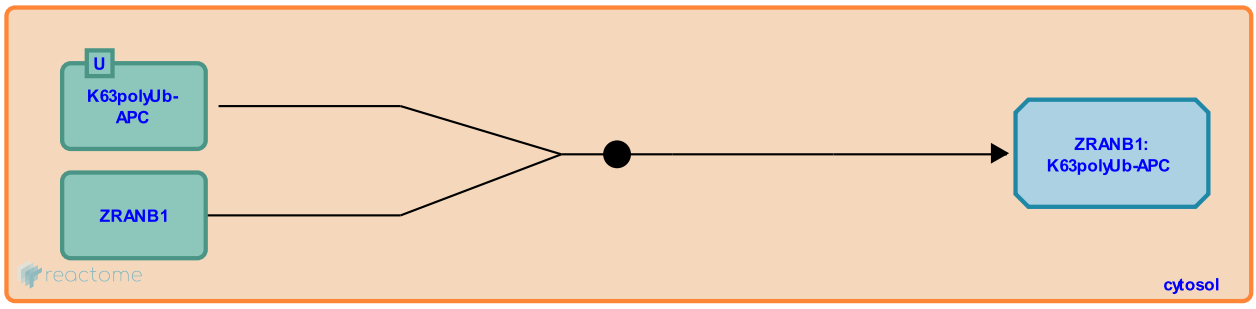
ZRANB1 binds APC ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-6781905

Type: binding

Compartments: cytosol



ZRANB1 (Trabid) binds and cleaves K63-linked ubiquitin chains. It is required for efficient TCF-mediated transcription in cells with high Wnt pathway activity, including colorectal cancer cell lines. ZRANB1 can deubiquitinate the APC tumor suppressor protein, a negative regulator of Wnt-mediated transcription (Tran et al. 2008).

Literature references

Tran, H., Schwarz-Romond, T., Bienz, M., Hamada, F. (2008). Trabid, a new positive regulator of Wnt-induced transcription with preference for binding and cleaving K63-linked ubiquitin chains. *Genes Dev., 22*, 528-42. ↗

Editions

2015-04-16	Authored	Jupe, S.
2016-05-05	Edited	Jupe, S.
2016-05-16	Reviewed	Meldal, BH.

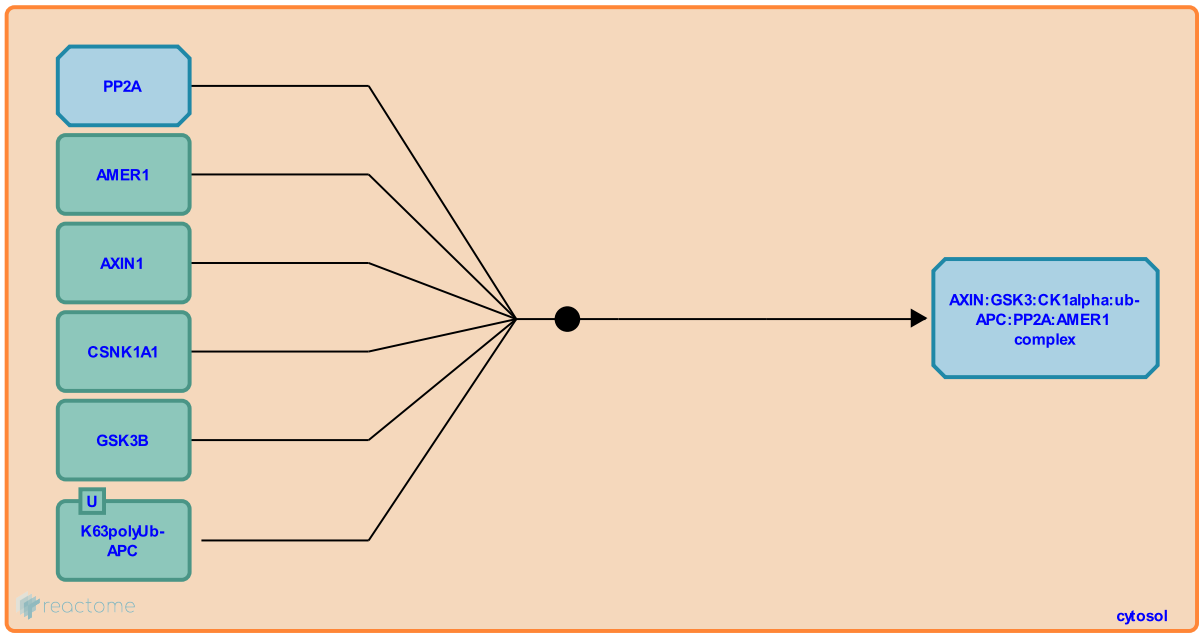
Assembly of the destruction complex ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-195251

Type: binding

Compartments: cytosol



The exact composition of the destruction complex is not known. A number of components appear to form a core complex, while others may associate with the complex transiently when a Wnt signal is present (reviewed in Kimelman and Xu, 2006). The core components include Axin, glycogen synthase kinase 3 (GSK-3), Casein kinase 1 (CKI) alpha, beta-catenin, Protein phosphatase 2A (PP2A) and Adenomatous Polyposis Coli (APC). CK1 epsilon, Diversin and PP1 may also be components of the complex.

Preceded by: Expression of AMER1 gene, APC is K63-polyubiquitinated, Expression of AXIN1 gene

Followed by: AXIN is phosphorylated in the destruction complex

Literature references

Roe, SM., Pearl, LH., Yeo, M., Fraser, E., Dajani, R., Dale, TC. et al. (2003). Structural basis for recruitment of glycogen synthase kinase 3beta to the axin-APC scaffold complex. *EMBO J, 22*, 494-501. ↗

Gil, R., Virshup, DM., Miller, JR., White, R., Seeling, JM., Moon, RT. (1999). Regulation of beta-catenin signaling by the B56 subunit of protein phosphatase 2A. *Science, 283*, 2089-91. ↗

Berndt, JD., Major, MB., Maccoss, MJ., Yi, X., Camp, ND., Biechele, TL. et al. (2007). Wilms tumor suppressor WTX negatively regulates WNT/beta-catenin signaling. *Science, 316*, 1043-6. ↗

Editions

2007-04-03	Authored	Kimelman, D.
2007-04-03	Edited	Matthews, L.
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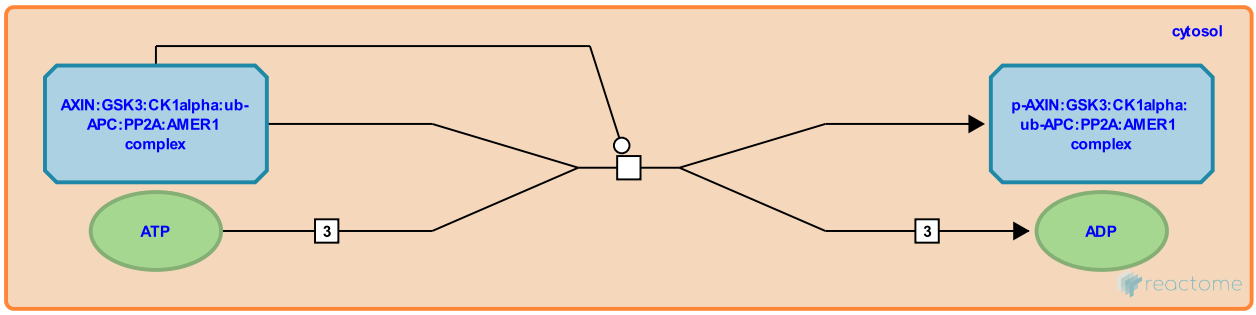
AXIN is phosphorylated in the destruction complex ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-5229343

Type: transition

Compartments: cytosol



In the absence of WNT signal, AXIN is a phosphoprotein; candidate kinases include both GSK3beta and CK1 (Ikeda et al, 1998; Willert et al, 1999; Jho et al, 1999; Yamamoto et al, 1999; Luo et al, 2007). Phosphorylation of AXIN is thought to increase its binding affinity for beta-catenin and GSK3beta, stabilizing the destruction complex and promoting efficient degradation of beta-catenin (Willert et al, 1999; Jho et al, 1999; Luo et al, 2007). A more recent model suggests that AXIN phosphorylation may disrupt an intramolecular interaction between its DIX domain and the beta-catenin binding region, which would otherwise keep AXIN in a 'closed' inactive state (Kim et al, 2013). Activation of the WNT pathway upon ligand binding favours dephosphorylation of AXIN by inactivating the kinases and allowing the steady state dephosphorylation by candidate phosphatases PP2A and PP1 to predominate (Willert et al, 1999; Luo et al, 2007; reviewed in Saito-Diaz et al, 2013).

Preceded by: Assembly of the destruction complex

Followed by: Association of beta-catenin with the destruction complex

Literature references

Wang, X., Wallace, HA., Page-McCaw, A., Lee, E., Thorne, CA., Chen, TW. et al. (2013). The way Wnt works: Components and mechanism. *Growth Factors, 31*, 1-31. ↗

Kikuchi, A., Matsumoto, S., Sato, A., Ohdan, H., Hanaki, H., Yamamoto, H. et al. (2012). An anti-Wnt5a antibody suppresses metastasis of gastric cancer cells in vivo by inhibiting receptor-mediated endocytosis. *Mol. Cancer Ther., 11*, 298-307. ↗

Takada, S., Ikeda, S., Kikuchi, A., Kishida, M., Yamamoto, H., Kishida, S. (1999). Phosphorylation of axin, a Wnt signal negative regulator, by glycogen synthase kinase-3beta regulates its stability. *J Biol Chem, 274*, 10681-4. ↗

Willert, K., Nusse, R., Shibamoto, S. (1999). Wnt-induced dephosphorylation of axin releases beta-catenin from the axin complex. *Genes Dev, 13*, 1768-73. ↗

Kikuchi, A., Murai, H., Ikeda, S., Kishida, S., Koyama, S., Yamamoto, H. (1998). Axin, a negative regulator of the Wnt signaling pathway, forms a complex with GSK-3beta and beta-catenin and promotes GSK-3beta-dependent phosphorylation of beta-catenin. *EMBO J., 17*, 1371-84. ↗

Editions

2014-01-17	Authored	Rothfels, K.
2014-01-22	Reviewed	Rajakulendran, N.
2014-02-15	Edited	Rothfels, K.
2014-05-12	Revised	Salahshor, S.

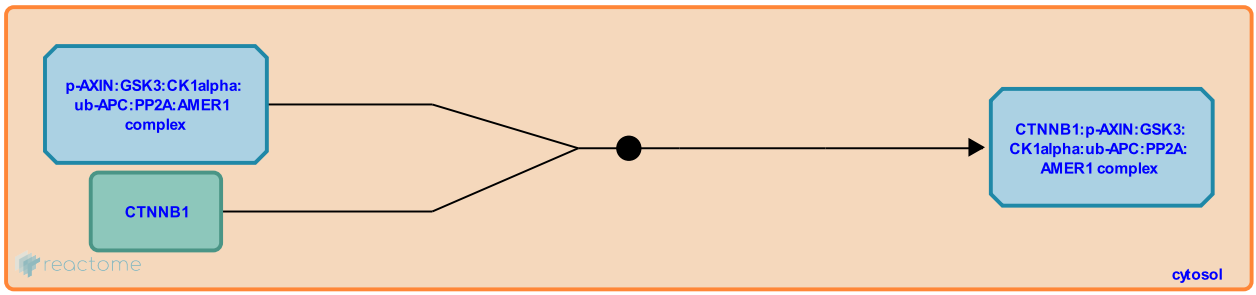
Association of beta-catenin with the destruction complex ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-195304

Type: binding

Compartments: cytosol



Beta-catenin associates with the destruction complex through an interaction with Axin and or APC. This association may also involve interactions with the 15 aa repeats in APC (Spink et al., 2001) or the third APC 20aa repeat and its N-terminal flanking residues (Ha et al., 2004, Xing et al., 2004; Liu et al., 2006).

Preceded by: AXIN is phosphorylated in the destruction complex

Literature references

Kimelman, D., Stenkamp, R., Hinds, TR., Xing, Y., Clements, WK., Le Trong, I. et al. (2004). Crystal structure of a beta-catenin/APC complex reveals a critical role for APC phosphorylation in APC function. *Mol Cell, 15*, 523-33. ↗

Editions

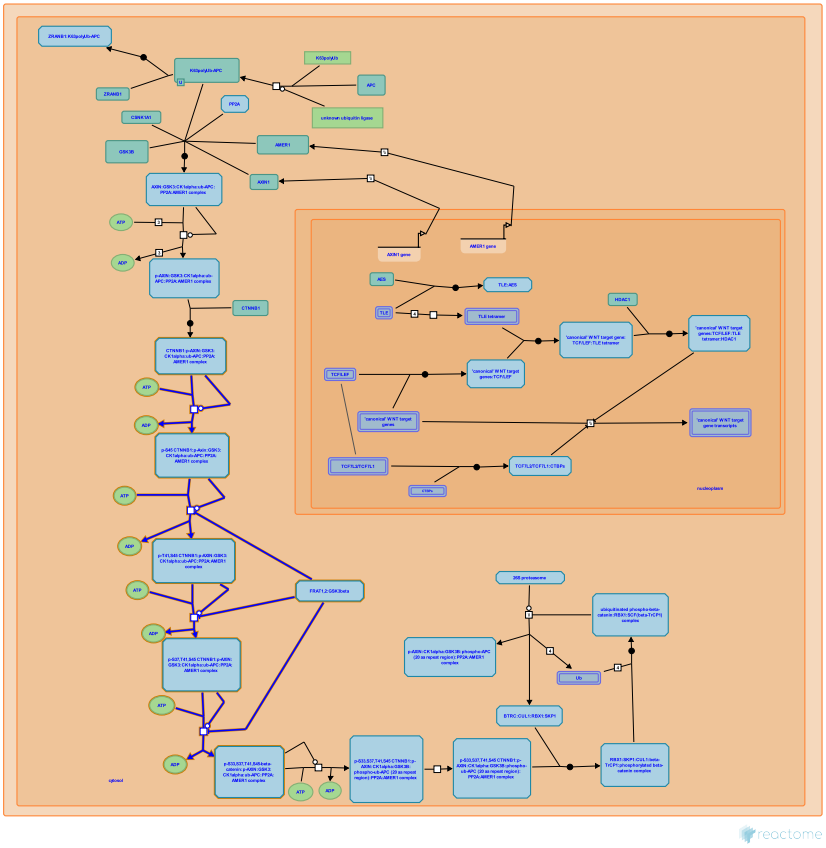
2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
2014-01-22	Revised	Rajakulendran, N.
2014-05-12	Revised	Salahshor, S.

Beta-catenin phosphorylation cascade ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-196299

Compartments: cytosol



Degradation of beta-catenin is initiated following amino-terminal serine/threonine phosphorylation. Phosphorylation of B-catenin at S45 by CK1 alpha primes the subsequent sequential GSK-3-mediated phosphorylation at Thr41, Ser37 and Ser33 (Amit et al., 2002 ; Lui et al., 2002).

Literature references

Andersen, JS., Birman, Y., Mann, M., Ben-Shushan, E., Alkalay, I., Hatzubai, A. et al. (2002). Axin-mediated CKI phosphorylation of beta-catenin at Ser 45: a molecular switch for the Wnt pathway. *Genes Dev, 16*, 1066-76. ↗

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.
2007-04-19	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.

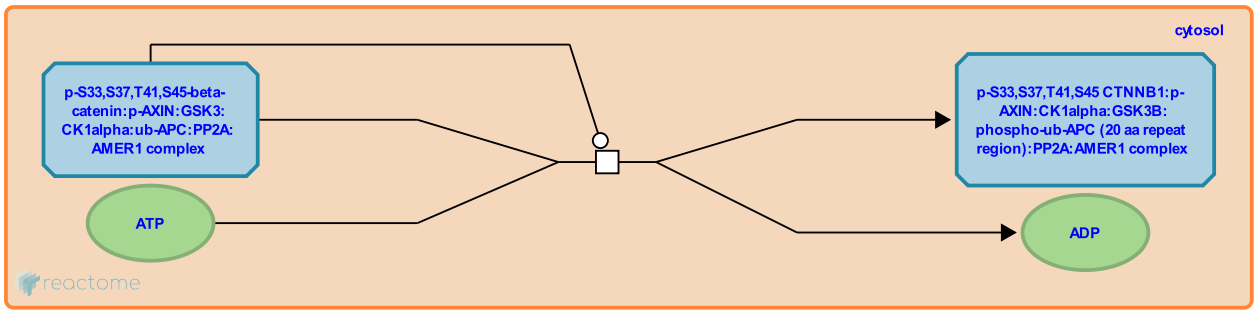
Phosphorylation of APC component of the destruction complex ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-195275

Type: transition

Compartments: cytosol



APC is phosphorylated on the 20 aa repeats by CK1 and potentially GSK-3. This significantly increases the binding affinity of the APC 20 aa repeats for beta-catenin, causing one of them to bind b-catenin in the same region as beta-catenin binds Axin, thus displacing beta-catenin from Axin (Step 5 above) (Reviewed in Kimelman, 2006).

Followed by: Dissociation of beta-catenin from Axin and association of beta catenin with phospho-(20 aa) APC in the detruction complex

Literature references

Liu, J., Zheng, J., Hinds, TR., Xing, Y., Xu, W. (2006). The third 20 amino acid repeat is the tightest binding site of APC for beta-catenin. *J Mol Biol, 360*, 133-44. ↗

Editions

2007-04-03	Authored	Kimelman, D.
2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
2014-01-22	Revised	Rajakulendran, N.
2014-05-12	Revised	Salahshor, S.

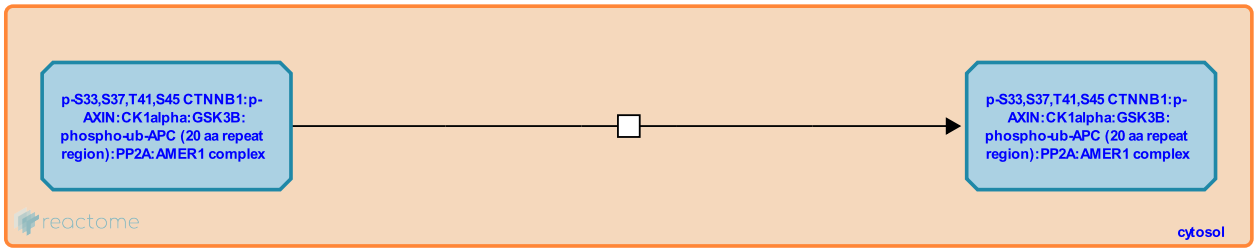
Dissociation of beta-catenin from Axin and association of beta catenin with phospho-(20 aa) APC in the detruction complex ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-195280

Type: transition

Compartments: cytosol



The phosphorylation of the 20 aa repeats in APC results in an increase in affinity for beta-catenin (Ha et al., 2004, Xing et al., 2004; Liu et al., 2006). The binding site of phospho -(20 aa) APC on beta-catenin overlaps the binding site of Axin on beta catenin. In addition, phosphorylated APC prevents the association of Axin with beta-catenin (Ha et al., 2004, Xing et al., 2004). In this model, phosphorylated APC may compete with Axin for beta-catenin binding, resulting in dissociation of the Axin:beta-catenin interaction in the destruction complex (see Kimelman and Xu 2006).

Preceded by: Phosphorylation of APC component of the destruction complex

Followed by: Association of beta-catenin with the RBX1:SCF(beta-TrCP1) ubiquitin ligase complex

Literature references

Kimelman, D., Stenkamp, R., Hinds, TR., Xing, Y., Clements, WK., Le Trong, I. et al. (2004). Crystal structure of a beta-catenin/APC complex reveals a critical role for APC phosphorylation in APC function. *Mol Cell, 15*, 523-33. ↗

Editions

2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
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2014-05-12	Revised	Salahshor, S.

Association of beta-catenin with the RBX1:SCF(beta-TrCP1) ubiquitin ligase complex

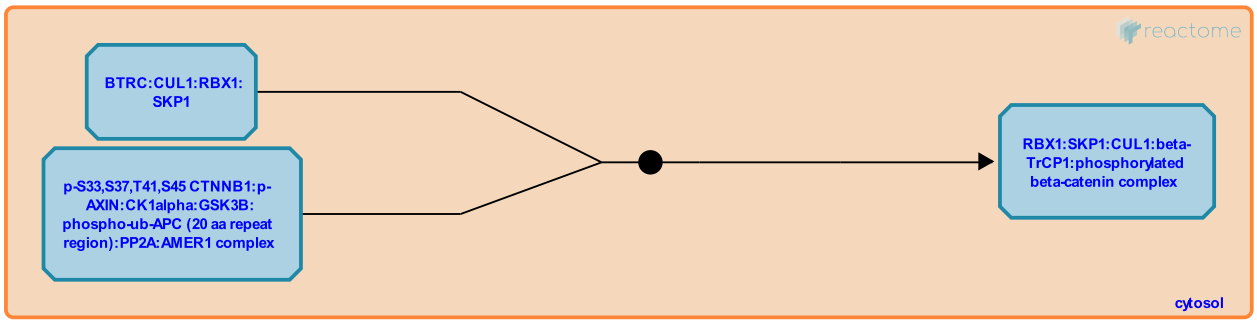
↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-2130279

Type: binding

Compartments: cytosol



B-TrCP associates with phosphorylated beta-catenin through the B-TrCP WD40 repeat region. Currently, it is unclear whether the ubiquitin ligase binds beta-catenin after it leaves the complex. It is equally possible that it binds beta-catenin while beta-catenin is still bound to Axin.

Preceded by: Dissociation of beta-catenin from Axin and association of beta catenin with phospho-(20 aa) APC in the detruction complex

Followed by: Multi-ubiquitination of phospho-beta-catenin by RBX1:SCF(beta-TrCP1)

Literature references

Chiaur, DS., Latres, E., Pagano, M. (1999). The human F box protein beta-Trcp associates with the Cul1/Skp1 complex and regulates the stability of beta-catenin. *Oncogene, 18*, 849-54. ↗

Strack, P., Elledge, SJ., Beer-Romero, P., Chu, CY., Winston, JT., Harper, JW. (1999). The SCFbeta-TRCP-ubiquitin ligase complex associates specifically with phosphorylated destruction motifs in IkappaBalpha and beta-catenin and stimulates IkappaBalpha ubiquitination in vitro. *Genes Dev, 13*, 270-83. ↗

Editions

2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
2012-02-16	Revised	Angers, S.
2012-02-16	Edited	Rothfels, K.
2013-05-30	Authored	Rothfels, K.
2014-01-22	Revised	Rajakulendran, N.
2014-05-12	Revised	Salahshor, S.

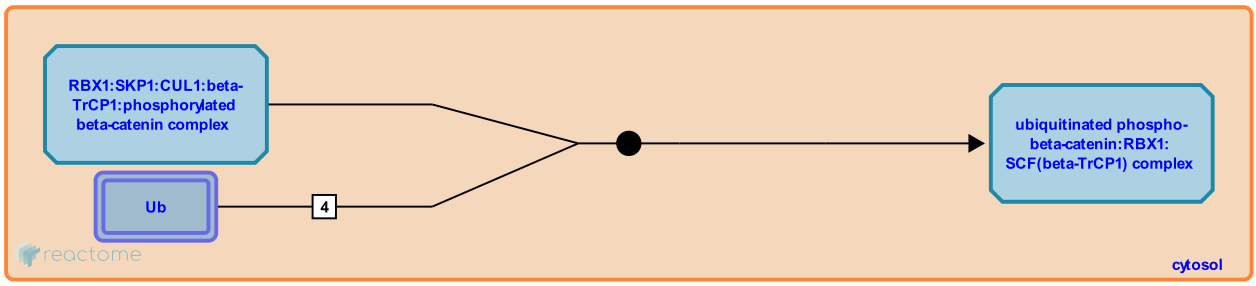
Multi-ubiquitination of phospho-beta-catenin by RBX1:SCF(beta-TrCP1) ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-2130286

Type: binding

Compartments: cytosol



Beta-catenin is ubiquitinated by the SCF-B-TrCP1 complex.

Preceded by: Association of beta-catenin with the RBX1:SCF(beta-TrCP1) ubiquitin ligase complex

Followed by: Degradation of ubiquitinated beta catenin by the proteasome

Literature references

Pavletich, NP., Wu, G., Schulman, BA., Harper, JW., Jeffrey, PD., Xu, G. (2003). Structure of a beta-TrCP1-Skp1-beta-catenin complex: destruction motif binding and lysine specificity of the SCF(beta-TrCP1) ubiquitin ligase. *Mol Cell, 11*, 1445-56. ↗

Editions

2007-04-03	Authored	Kimelman, D.
2007-04-03	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
2012-02-16	Revised	Angers, S.
2012-02-16	Edited	Rothfels, K.
2014-01-22	Revised	Rajakulendran, N.
2014-05-12	Revised	Salahshor, S.

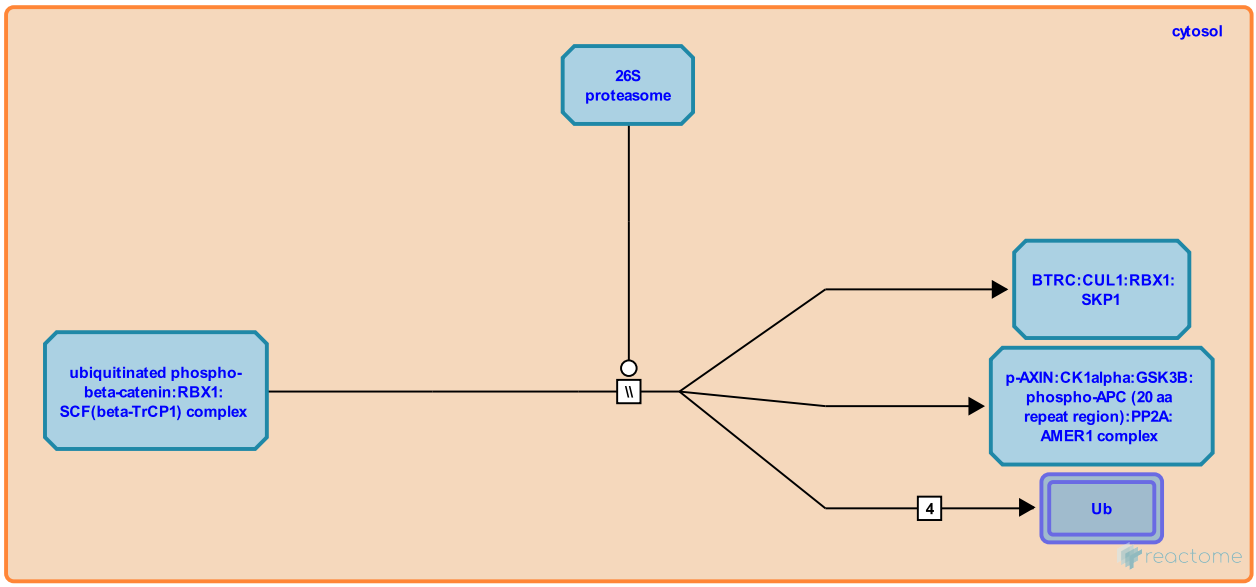
Degradation of ubiquitinated beta catenin by the proteasome ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-2130282

Type: omitted

Compartments: cytosol



Ubiquitinated beta-catenin is degraded by the proteasome.

Preceded by: Multi-ubiquitination of phospho-beta-catenin by RBX1:SCF(beta-TrCP1)

Literature references

Piccioni, E., Serini, S., Resci, F., Monego, G., Calviello, G., Palozza, P. et al. (2006). Docosahexaenoic Acid Induces Proteasome-dependent Degradation of {beta}-catenin, Down-regulation of Survivin and Apoptosis in Human Colorectal Cancer Cells not expressing COX-2. *Carcinogenesis.* ↗

Editions

2007-04-03	Authored	Matthews, L.
2007-04-19	Edited	Matthews, L.
2007-04-27	Reviewed	Pagano, M.
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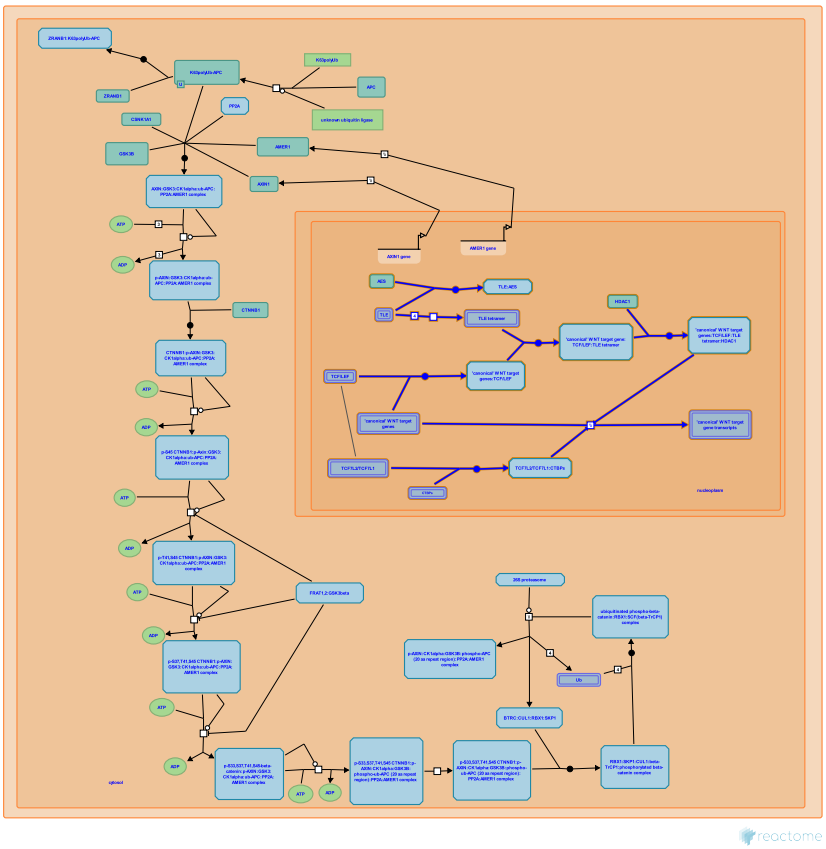
2014-05-12	Revised	Salahshor, S.

Repression of WNT target genes ↗

Location: Degradation of beta-catenin by the destruction complex

Stable identifier: R-HSA-4641265

Compartments: nucleoplasm



In the absence of a WNT signal, many WNT target genes are repressed by Groucho/TLE. Groucho was initially identified in Drosophila, where it has been shown to interact with a variety of proteins to repress transcription (reviewed in Turki-Judeh and Courey, 2012). Groucho proteins, including the 4 human homologues (transducin-like enhancer of split (TLE) 1-4), do not bind DNA directly but instead are recruited to target genes through interaction with DNA-binding transcription factors including TCF/LEF (Brantjes et al, 2001; reviewed in Chen and Courey, 2000). Groucho proteins are believed to oligomerize in a manner that depends on an N-terminal glutamine-rich Q domain, and oligomerization may be important for function (Song et al, 2004; Pinto and Lobe, 1996). Groucho/TLE proteins affect levels of gene expression by interacting with the core transcriptional machinery as well as by modfiying chromatin structure through direct interaction with histones and recruitment of histone deacetylases, among other mechanisms (reviewed in Turki-Judeh and Courey, 2012). In addition to the four TLE proteins, human cells also include a truncated TLE-like protein called amino-terminal enhancer of split (AES) which contains the N-terminal Q domain but lacks much of the C-terminal sequence of TLE proteins, including the WD domain which is important for many protein-protein interactions. AES is believed to act as a dominant negative, since it is able to heter-oligomerize with full-length TLE proteins to form non-functional complexes (Brantjes et al, 2001; reviewed in Beagle and Johnson, 2010).

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Editions

2013-09-23	Authored	Rothfels, K.
2014-01-22	Reviewed	Rajakulendran, N.
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