TGFBR2 phosphorylates TGFBR1

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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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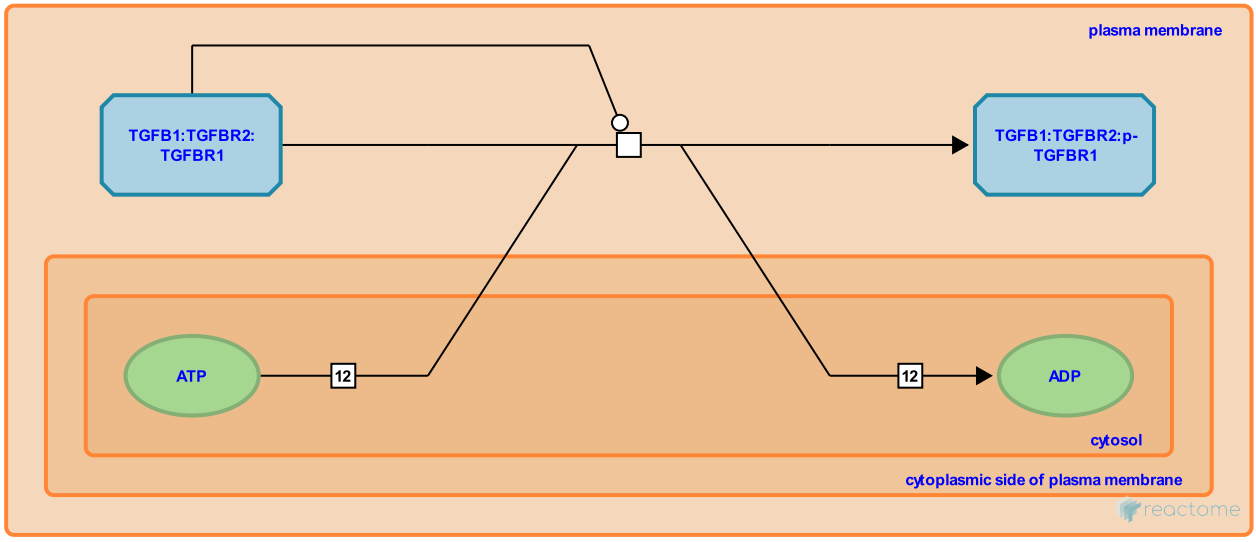
Stable identifier: R-HSA-170843

Type: transition

Compartments: cytosol, plasma membrane



Formation of the hetero-tetrameric TGF-beta-1 receptor complex induces receptor rotation, so that TGFBR2 and TGFBR1 cytoplasmic kinase domains face each other in a catalytically favourable configuration. The constitutively active type II receptor kinase (which auto-phosphorylates in the absence of ligand), trans-phosphorylates specific serine residues at the conserved Gly-Ser-rich juxtapositioned domain (GS domain) of the type I receptor (Wrana et al. 1994, Souchelnytskyi et al. 1996).

In addition to phosphorylation, TGFBR1 may also be sumoylated in response to TGF-beta-1 stimulation. Sumoylation enhances TGFBR1 function by facilitating recruitment and phosphorylation of SMAD3 (Kang et al. 2008).

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