# Autophosphorylation of PDGF alpha receptors

Garapati, P V., Heldin, CH., Jassal, B.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.



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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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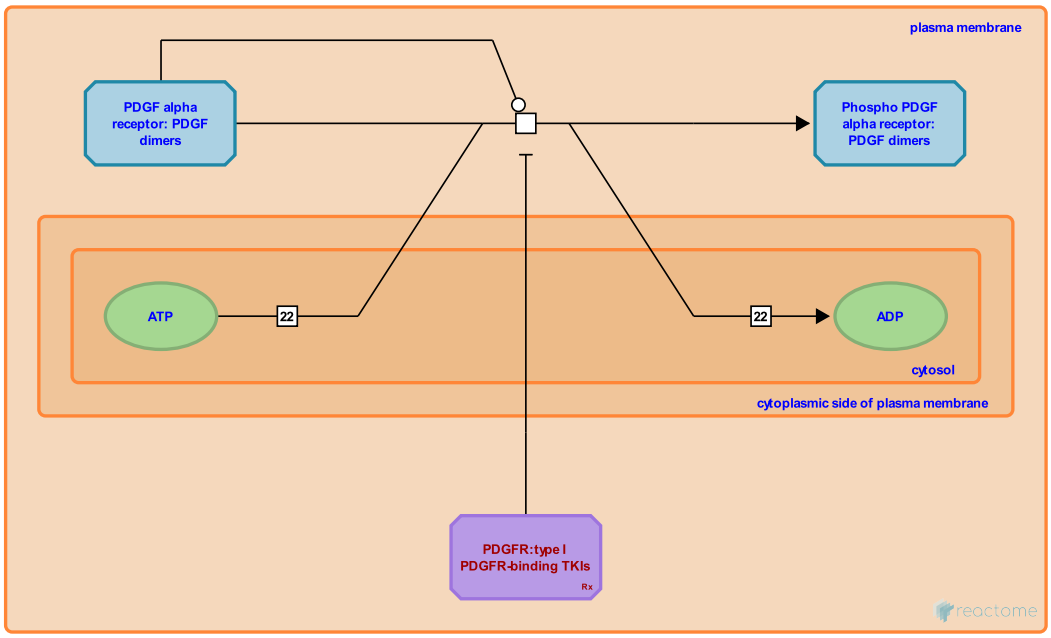
## Autophosphorylation of PDGF alpha receptors ↗

**Stable identifier:** R-HSA-389083

**Type:** transition

**Compartments:** cytosol, plasma membrane




Receptor dimerisation is key event in PDGF receptor activation. The intracellular regions of the receptors are juxtaposed which allows trans-phosphorylation between the two receptors in the complex.
The autophosphorylation site Y857 located inside the kinase domain of beta-receptor (PDGFRB) is important for activation of the kinase. This tyrosine is conserved in the alpha-receptor (PDGFRA), where it corresponsds to Y849, and in almost all other tyrosine kinase receptors. The other known autophosphorylation sites are localized outside the kinase domains of the alpha- and beta- receptors; of the 15( beta) or 16 (alpha) tyrosine residues in the intracellular, non-catalytic part of the beta- or alpha receptor, 11 and 10, respectively, are autophosphorylation sites (reviewed in Heldin et al, 1998).
PDGFRA and PDGFRB activity can be inhibited by binding to type I and type II tyrosine kinase inhibitors (reviewed in Roskoski, 2018). Type I inhibitors such as crenolanib, avripatinib and pazopanib, bind to the active conformation of the receptor and inhibit trans-autophosphorylation (Ip et al, 2018; Evans et al, 2017; Davids et al, 2009; reviewed in Roskoski, 2018; Klug et al, 2018; Papadopoulos and Lennartsson, 2016).

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## Editions

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