VEGFR2 autophosphorylates

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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


Reactome database release: 83

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https://reactome.org
Binding of VEGFA to VEGFR2 induces receptor dimerization and autophosphorylation, leading to the recruitment of downstream signalling molecules. Once the two VEGFR2 receptors are cross-linked to each other, via simultaneous interaction with VEGFA dimer, their membrane-proximal Ig-like domain 7s are held in close proximity so that low-affinity homotypic interactions between these domains further stabilise the receptor dimers. This allows for the exact positioning of the intracellular kinase domains resulting in VEGFR2 autophosphorylation (Ruch et al. 2007, Holmes at al. 2007). The major tyrosine residues known to be autophosphorylated are Y801 and Y951 in the kinase-insert domain, Y1054 and Y1059 within the kinase domain, and Y1175 and Y1214 in the C-terminal tail of VEGFR (Dougher-Vermazen et al. 1994, Cunningham et al. 2007, Kendall et al. 1999, Matsumoto et al. 2005). The Y1175 (mice Y1173) is crucial for endothelial and haemopoietic cell development. Mice with mutation Y1173F die between E8.5 and E9.5 from lack of endothelial and haemopoietic development (Sakurai et al. 2005).

**Literature references**


**Editions**

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