Activation of RAC1 by VAV

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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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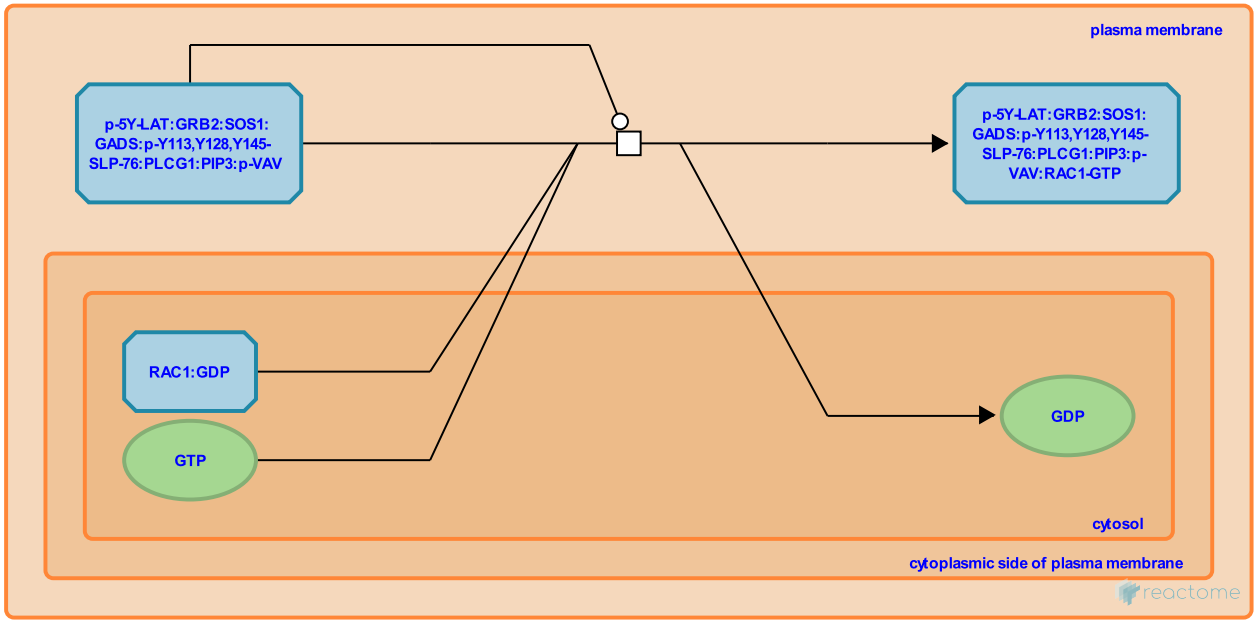
Stable identifier: R-HSA-2730840

Type: transition

Compartments: cytosol, plasma membrane



Rac1 exists in inactive state in the cytosol until the reception of extracellular signals by the cell. To be functional Rac1 is rapidly targeted to the plasma membrane upon cell stimulation. The main factors involved in this mobilisation are the Rac GEFs like VAV and phospholipids (PtdIns(4,5)P2, PtdIns(3,4,5) P3) and lipid rafts at the plasma membrane. VAV catalyses the disassociation of GDP from Rac1 by modifying the nucleotide-binding site such that GDP is released and subsequently replaced. The incoming GTP occupies the nucleotide binding site and finally displaces VAV from Rac1 (Bos et al. 2007, Bustelo et al. 2012).

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Ardouin, L., Tybulewicz, VL., Reynolds, LF., Prisco, A. (2003). Vav1: a key signal transducer downstream of the TCR. *Immunol Rev, 192,* 42-52. ↗

Editions

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