Recruitment of VAV to p-SLP-76

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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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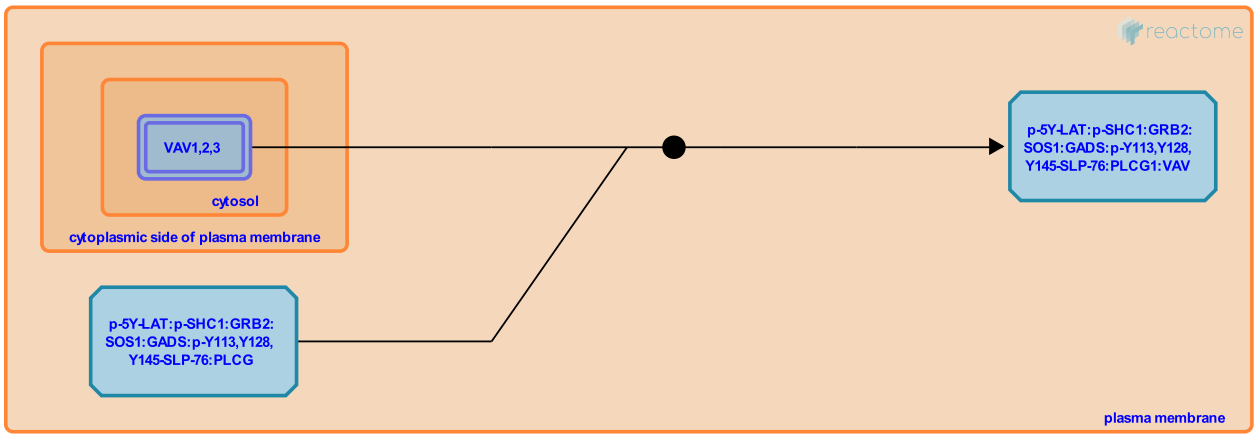
Stable identifier: R-HSA-2730892

Type: binding

Compartments: plasma membrane, cytosol



VAV an activator of RAC-GTPases, is redistributed to plasma membrane and is phosphorylated following engagement of FCERI. Phosphorylated SLP-76 tyrosines Y113 and Y128 (112Y and 128Y in mouse) provide binding sites for the SH2 domains of VAV. The binding of VAV to these phosphotyrosine residues may link SLP-76 to the Jun amino-terminal kinase (JNK) pathway and the actin cytoskeleton (Kettner et al. 2003).
In addition to its known role as guanine nucleotide exchange factor (GEF), VAV also modulates cytokine production in mast cells. VAV1-deficient bone marrow-derived mast cells exhibited reduced degranulation and cytokine production and calcium release in addition of reduced activation of c-Jun NH2-terminal kinase 1 (JNK1), although tyrosine phosphorylation of FCERI, SYK and LAT was normal (Manetz et al. 2001, Arudchandran et al. 2000, Song et al. 1999).

Literature references

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Editions

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