Interaction of PECAM-1 and SHP-2

Garapati, P V., Trowsdale, J., de Bono, 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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Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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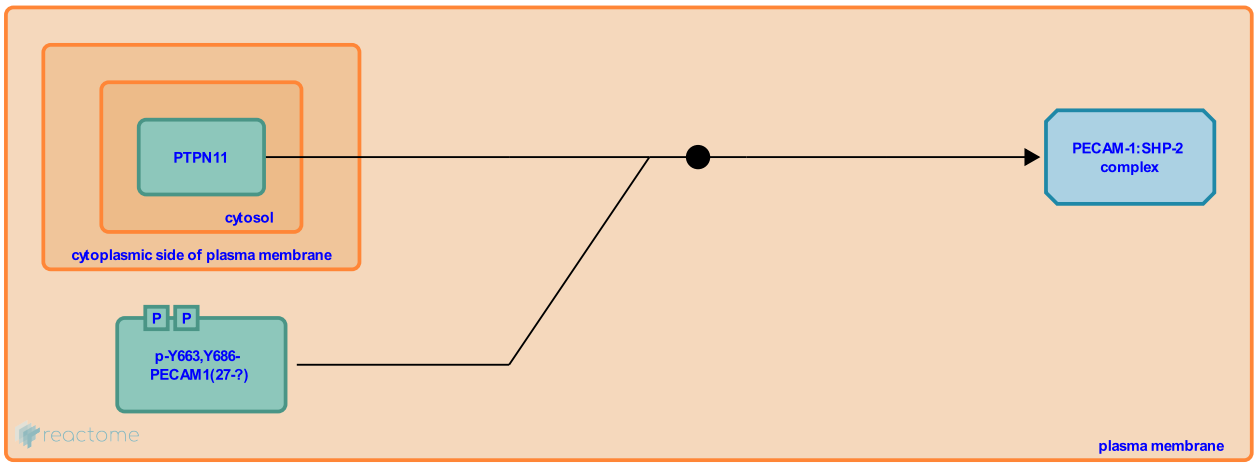
Stable identifier: R-HSA-210294

Type: binding

Compartments: cytosol, plasma membrane



PECAM-1 becomes tyrosine-phosphorylated during the platelet aggregation process; the phosphorylation of two tandem tyrosine residues (Y663 and Y686) within the cytoplasmic domain is required for downstream signalling events. Phosphorylation creates docking sites for the protein-tyrosine phosphatase SHP-2. The interaction between SHP-2 and PECAM-1 is dependent upon integrin-mediated platelet/platelet interactions and occurs via the Src homology 2 (SH2) domains of the phosphatase and highly conserved phosphatase-binding motifs encompassing phosphotyrosines 663 and 686 within the cytoplasmic domain of PECAM-1.

Literature references

Ward, CM., Wang, R., Newman, PJ., Jackson, DE. (1997). The protein-tyrosine phosphatase SHP-2 binds platelet/endothelial cell adhesion molecule-1 (PECAM-1) and forms a distinct signaling complex during platelet aggregation. Evidence for a mechanistic link between PECAM-1- and integrin-mediated cellular signaling. *J Biol Chem, 272*, 6986-93. ↗

Editions

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