Ptk2 autophosphorylates at Y397

Garapati, P V., Ip, NY.

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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Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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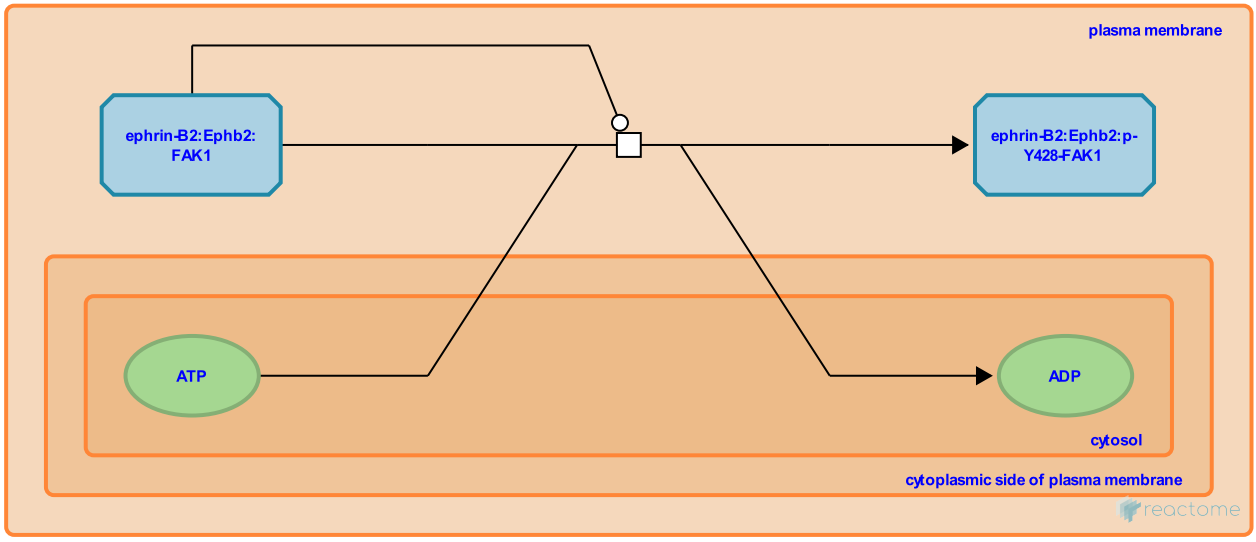
Stable identifier: R-MMU-3928599

Type: transition

Compartments: plasma membrane, cytosol



Focal adhesion kinase 1 (PTK2, FAK, FAK1) activation plays a critical role in EPHB receptor signaling in dendritic spines. PTK2 has six tyrosine phosphorylation sites, with tyrosine 397 being the main auto-phosphorylation site present upstream of the kinase domain (Schaller et al. 1994). Activation of EPHB receptors induces long-lasting phosphorylation of PTK2 on tyrosine 397 (Shi et al. 2009). This phosphorylated tyrosine then creates a binding site for other signaling proteins that link PTK2 to downstream signaling pathways and actin cytoskeleton.

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Editions

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