BTK in PIP3:BTK:G beta-gamma complex is activated

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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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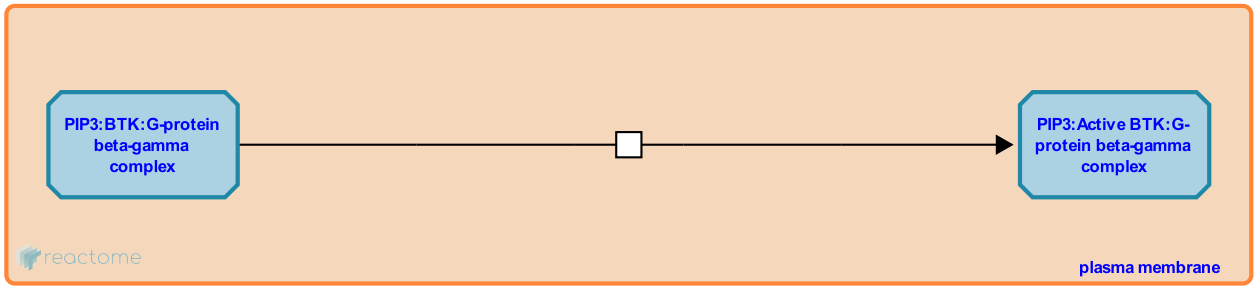
Stable identifier: R-HSA-8964271

Type: transition

Compartments: plasma membrane



G-Protein Coupled Receptors (GPCR) sense extracellular signals and activate different Guanine nucleotide binding proteins (G-proteins) that have alpha, beta and gamma subunits. Upon activation, the alpha subunit of G-proteins dissociates from beta-gamma and the both are then free to regulate downstream effectors. G-protein beta-gamma complex, along with phosphatidylinositol 3,4,5-trisphosphate (PIP3), recruits the non-receptor Tyrosine-protein kinase BTK to the cell membrane. In the membrane, the G-protein beta-gamma complex binds to the catalytic domain of BTK and activates it. Active BTK is then released to the cytoplasm. Physiologically, BTK plays a key role in B lymphocyte development, differentiation and signalling.

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Editions

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