G-protein beta:gamma signalling

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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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references


Reactome database release: 69

This document contains 5 pathways (see Table of Contents)

https://www.reactome.org
The classical role of the G-protein beta/gamma dimer was believed to be the inactivation of the alpha subunit, Gbeta/gamma was viewed as a negative regulator of Galpha signalling. It is now known that Gbeta/gamma subunits can directly modulate many effectors, including some also regulated by G alpha.

**Literature references**


**Editions**

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PI3K gamma (PI3KG) is a heterodimer consisting of a p110 catalytic subunit associated with a regulatory p101 or p84 subunit. PI3KG is most highly expressed in neutrophils, where the p101 form predominates (approximately 95%). G beta:gamma recruits PI3KG to the plasma membrane, both activating PI3KG and providing access to its substrate PIP2, which is converted to PIP3.

**Literature references**


Phospholipase C beta (PLCbeta) isoforms are activated by G-protein beta:gamma in the order PLCB3 > PLCB2 > PLCB1. Gbeta:gamma binds to the pleckstrin homology domain of PLC beta, increasing phospholipase activity and leading to increased hydrolysis of PIP2 to DAG and IP3.

**Literature references**


**Editions**

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G beta:gamma signalling through BTK

Location: G-protein beta:gamma signalling

Stable identifier: R-HSA-8964315

Compartments: cytoplasmic side of 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. Here, the G-protein beta-gamma complex activates BTK. Subsequently, active BTK dissociates from the complex to phosphorylate downstream substrates. Physiologically, BTK plays a key role in B lymphocyte development, differentiation and signalling.

Literature references


Editions

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G beta:gamma signalling through CDC42

**Location:** G-protein beta:gamma signalling

**Stable identifier:** R-HSA-8964616

**Compartments:** plasma membrane, cytosol

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. Serine/threonine-protein kinase PAK 1 binds with Rho guanine nucleotide exchange factor 6 (ARHGEF6, PIX-Alpha) in the cytosol and is subsequently translocated by the G-protein beta-gamma complex to the plasma membrane. Here, ARHGEF6 activates Cell division control protein 42 homolog (CDC42) by acting as a GEF. Once active, CDC42 can facilitate the activation of PAK1. CDC42 is known to be involved in epithelial cell polarization processes.

**Literature references**


**Editions**

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