FLT3 mutants recruit PIK3R1 through phosphorylated GAB2

Kazi, JU., Rothfels, K.
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


Reactome database release: 83

This document contains 1 reaction (see Table of Contents)

https://reactome.org
FLT3 mutants recruit PIK3R1 through phosphorylated GAB2

Stable identifier: R-HSA-9698179

Type: binding

Compartments: cytosol, plasma membrane

Diseases: cancer

Inferred from: Active Flt3:Grb2:p-Y-Gab2 binds Pik3r1 (Mus musculus)

FLT3 ITD mutants signal through the PI3K/AKT pathway in a GRB2-GAB2-dependent manner. As is the case for the wild-type receptor, FLT3 ITD-associated GAB2 is phosphorylated and recruits the p85 regulatory subunit of PI3K to the activated receptor (Masson et al, 2009; Zhang et al, 1999; Zhang et al, 2000). Other FLT3-interacting adaptor proteins have also been shown to bind to p85 and may additionally contribute to PI3K/AKT signaling downstream of FLT3; these alternate mechanisms are not shown in this pathway (reviewed in Kazi and Ronnstrand, 2019).

Literature references


Editions

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