14-3-3 zeta binding allows recruitment of PI3K

Hercus, TR., Jupe, S., Lopez, AF., Ray, KP.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

15/05/2024

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

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

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

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. ↗

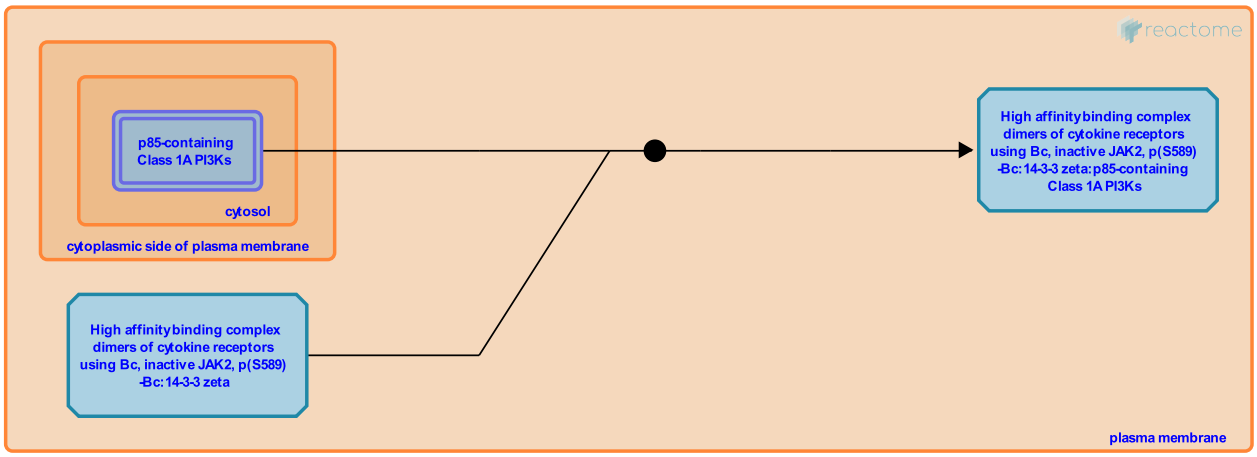
Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

14-3-3 zeta binding allows recruitment of PI3K ↗

Stable identifier: R-HSA-914182

Type: binding

Compartments: cytosol, plasma membrane



Immunoprecipitation and kinase activity experiments demonstrated that Ser-585 phosphorylation of the common beta chain (Bc) was required for activation of PI3K activity in response to IL-3 and co-precipitation of Bc, 14-3-3 zeta and the p85 subunit of Class 1A PI3 kinases (Guthridge et al. 2000). Subsequent experiments confirmed that Ser-585 phosphorylation and PI3K activation are required to promote cell survival in response to GM-CSF, but not for proliferation responses, and that this mechanism is independent of Bc tyrosine phosphorylation (Guthridge et al. 2004). This is one of two mechanisms described for the recruitment of PI3K to the IL-3/IL-5/GM-CSF receptors; the other involves Bc tyrosine-593 phosphorylation-mediated recruitment of SHC1, GRB2 and GAB2.

Literature references

Barry, EF., Berndt, MC., Stomski, FC., McClure, BJ., Lopez, AF., Dottore, M. et al. (2000). Site-specific serine phosphorylation of the IL-3 receptor is required for hemopoietic cell survival. *Mol Cell, 6*, 99-108. ↗

Editions

2010-05-17	Authored	Ray, KP.
2010-08-06	Edited	Jupe, S.
2010-09-06	Reviewed	Lopez, AF., Hercus, TR.