SHC1 bound to the common beta chain becomes tyrosine phosphorylated

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03/05/2024

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. ↗

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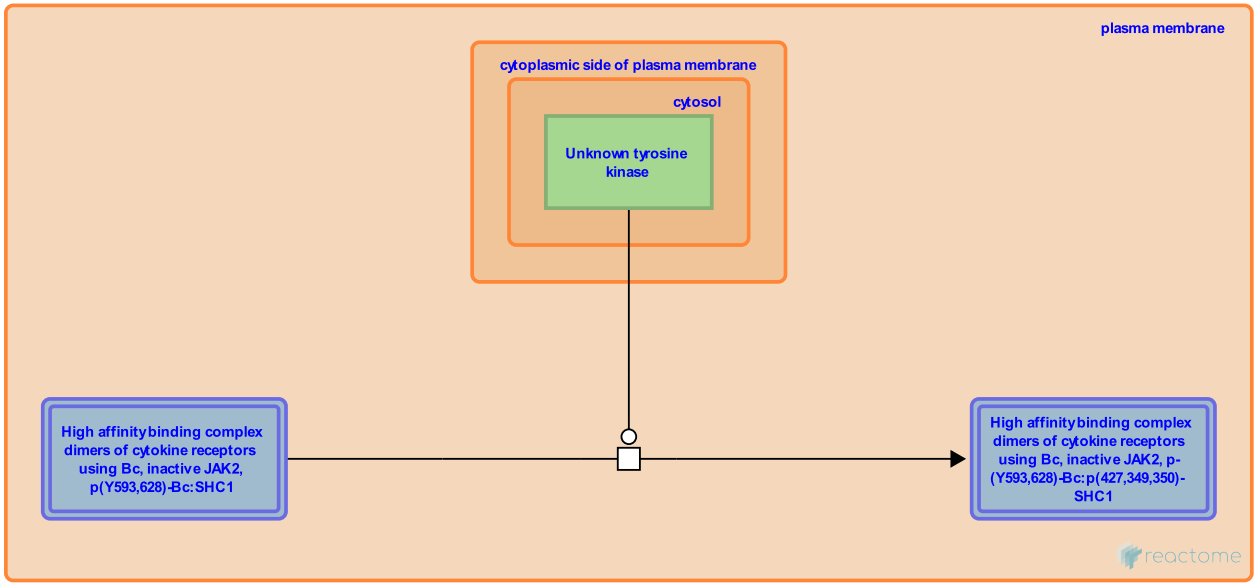
Stable identifier: R-HSA-879925

Type: transition

Compartments: plasma membrane



IL-3, IL-5 and GM-CSF all induce tyrosine phosphorylation of Shc (Dorsch et al. 1994). Three sites are known to mediate specific downstream associations; tyrosine Y427 (Salcini et al. 1994) mediates the subsequent association of Shc with Grb2 (Salcini et al. 1994). The identity of the kinase is unknown. Y349 and Y350 phosphorylation is not required for Ras-MAPK signaling but are involved in IL-3-induced cell survival (Gotoh et al. 1996).
Residue numbering used here refers to Uniprot P29353 where the p66 isoform has been selected as the canonical form. Literature references given here refer to the p52 isoform which lacks the first 110 residues, so Y427 is referred to as Y317 in Salcini et al. 1994, Y349 and Y350 as Y239 and Y240 in Gotoh et al. 1996.

Literature references

Tojo, A., Shibuya, M., Gotoh, N. (1996). A novel pathway from phosphorylation of tyrosine residues 239/240 of Shc, contributing to suppress apoptosis by IL-3. *EMBO J, 15*, 6197-204. ↗

Pawson, T., Pelicci, G., Salcini, AE., McGlade, J., Nicoletti, I., Pelicci, PG. (1994). Formation of Shc-Grb2 complexes is necessary to induce neoplastic transformation by overexpression of Shc proteins. *Oncogene, 9*, 2827-36. ↗

Diamantstein, T., Dorsch, M., Hock, H. (1994). Tyrosine phosphorylation of Shc is induced by IL-3, IL-5 and GM-CSF. *Biochem Biophys Res Commun, 200*, 562-8. ↗

Editions

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