Recruitment of IFNAR1

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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

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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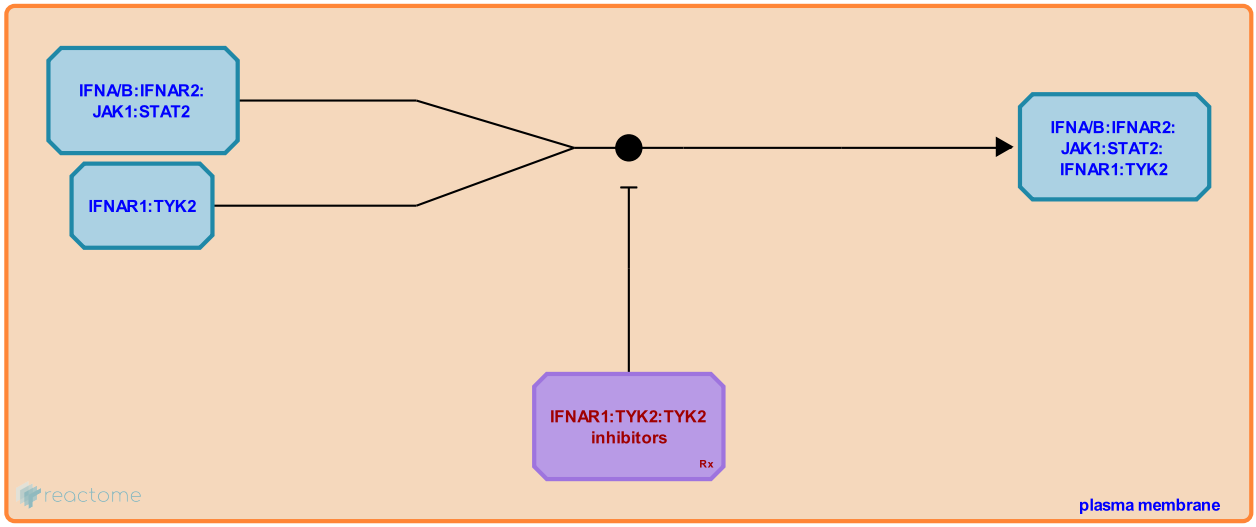
Stable identifier: R-HSA-909724

Type: binding

Compartments: plasma membrane



The extracellular domain of IFNAR1 is atypical, consisting of a tandem array of four FNIII domains and the first three N-terminal FNIII domains are involved in ligand recognition. IFNAR1 is recruited to the binary complex (IFNA/B:IFNAR2) on the membrane to form the ternary complex (IFNAR2:IFNA/B:IFNAR1). TYK2 kinase is pre-associated with IFNAR1 and JAK1 with IFNAR2. The binding of IFNA/B to IFNA receptors brings these JAK kinase together, allowing cross-phosphorylation and kinase activation.

Literature references

Jaitin, DA., Schreiber, G., Roisman, LC., Baker, DP. (2005). Mutational analysis of the IFNAR1 binding site on IFNalpha2 reveals the architecture of a weak ligand-receptor binding-site. *J Mol Biol, 353*, 271-81.

Sempé, P., Gallet, X., Nardeux, PC., Guymarho, J., Eid, P., Tovey, MG. et al. (2004). Identification of residues of the IFNAR1 chain of the type I human interferon receptor critical for ligand binding and biological activity. *Biochemistry, 43*, 12498-512.

Pellegrini, S., Piehler, J., Schreiber, G., Uzé, G. (2007). The receptor of the type I interferon family. *Curr Top Microbiol Immunol, 316*, 71-95.

Piehler, J., Gavutis, M., Lata, S., Lamken, P. (2004). Ligand-induced assembling of the type I interferon receptor on supported lipid bilayers. *J Mol Biol, 341*, 303-18.

Editions

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