Recruitment of STAT2 to p-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

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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. ↗

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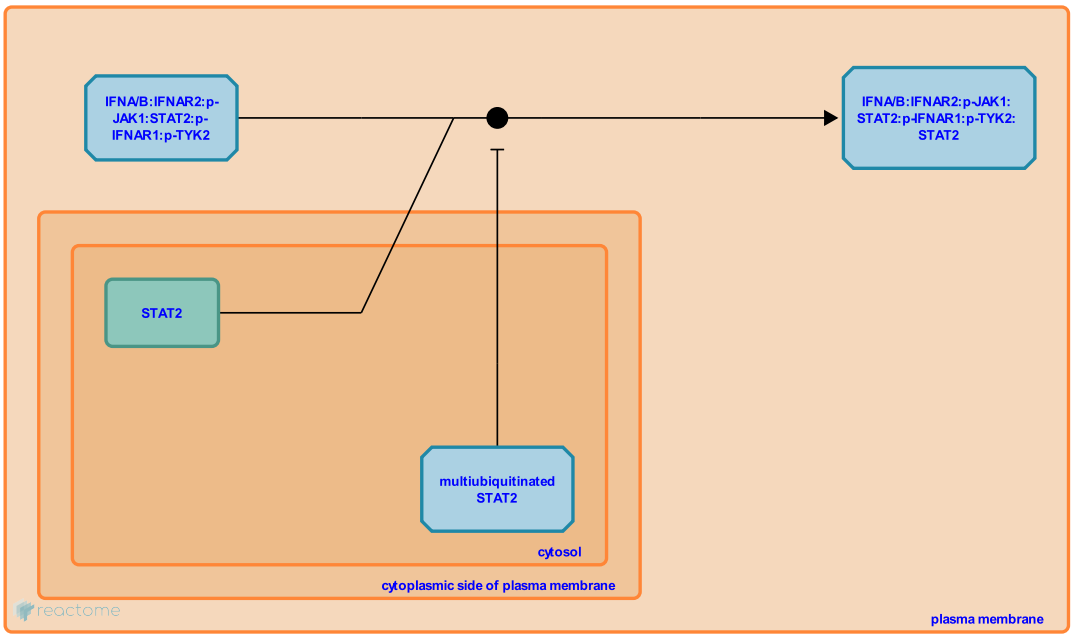
Stable identifier: R-HSA-909719

Type: binding

Compartments: cytosol, plasma membrane



Phosphorylated tyrosine residue 466 on IFNAR1 acts as a docking site for STAT2. Latent STAT2 is recruited to this phosphotyrosine residue via its SH2 domain (Yan et al, 1996). Infection by human respiratory syncytial virus (hRSV) leads to loss of STAT2 by ubiquitination, catalyzed by a hRSV NS1 complex with elongin C (ELOC) and cullin-5 (CUL5) acting as an E3 ubiquitin ligase, and subsequent proteasomal STAT2 degradation (Elliott et al, 2007).

Literature references

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Gupta, S., Greenlund, AC., Krolewski, JJ., Yan, H., Schreiber, RD., Schindler, CW. et al. (1996). Phosphorylated interferon-alpha receptor 1 subunit (IFNaR1) acts as a docking site for the latent form of the 113 kDa STAT2 protein. *EMBO J, 15*, 1064-74.

Editions

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