

p-STAT1, p-STAT3 dissociate from IL27RA:IL12RB2 receptor

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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., 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. [↗](#)

Reactome database release: 88

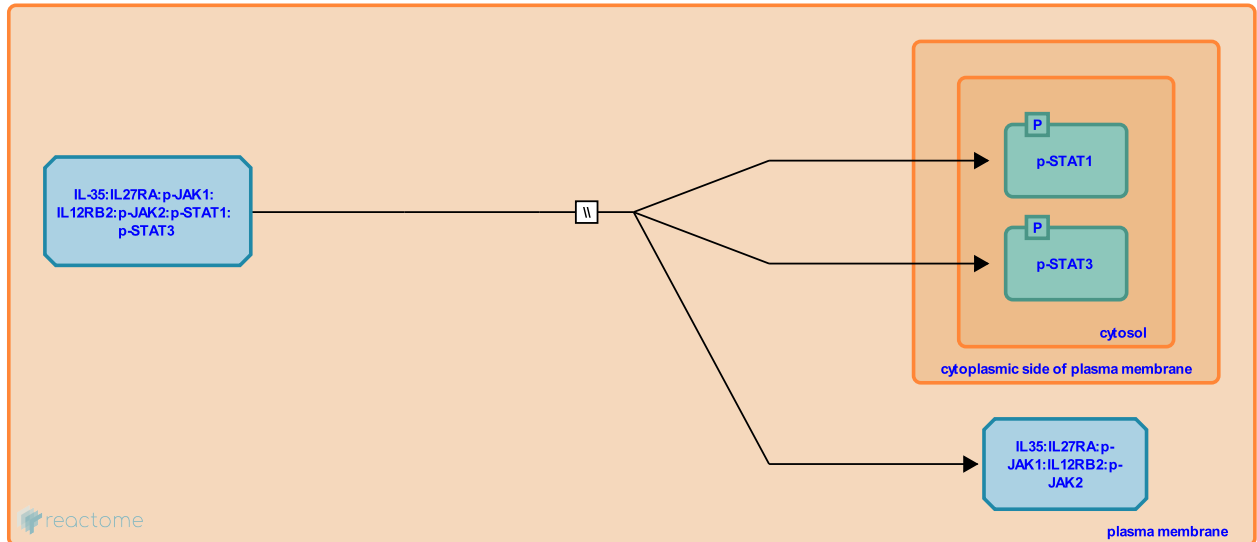
This document contains 1 reaction ([see Table of Contents](#))

p-STAT1, p-STAT3 dissociate from IL27RA:IL12RB2 receptor [↗](#)

Stable identifier: R-HSA-8984023

Type: omitted

Compartments: plasma membrane, cytosol, extracellular region



Interleukin 35 (IL35) can signal via IL35 receptors triggering the JAK/STAT pathway downstream. Signal transducer and activator of transcription 1 alpha/beta (STAT1) and Signal transducer and activator of transcription 3 alpha/beta (STAT3) bind to the JAK associated IL35 receptor complex and are activated via phosphorylations. Subsequently, phosphorylated p STAT1:p STAT3 dissociates from the receptor complex (Wang et al. 2014). This is a black box event because there is no literature evidence about the exact mechanism of the release of STAT1 and STAT3 from the receptor complex.

Literature references

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Kim, SH., Wingfield, PT., Mahdi, RM., Dambuza, IM., Yu, CR., Wang, RX. et al. (2014). Interleukin-35 induces regulatory B cells that suppress autoimmune disease. *Nat. Med.*, 20, 633-41. [↗](#)

Editions

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