

**p-Y701-STAT1, p-Y705-STAT3, p-Y649-  
STAT5 dissociates from IL9:p-Y407-  
IL9R:JAK1:IL2RG:p-904,939-JAK3:p-Y705-  
STAT3**

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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. [↗](#)

Reactome database release: 88

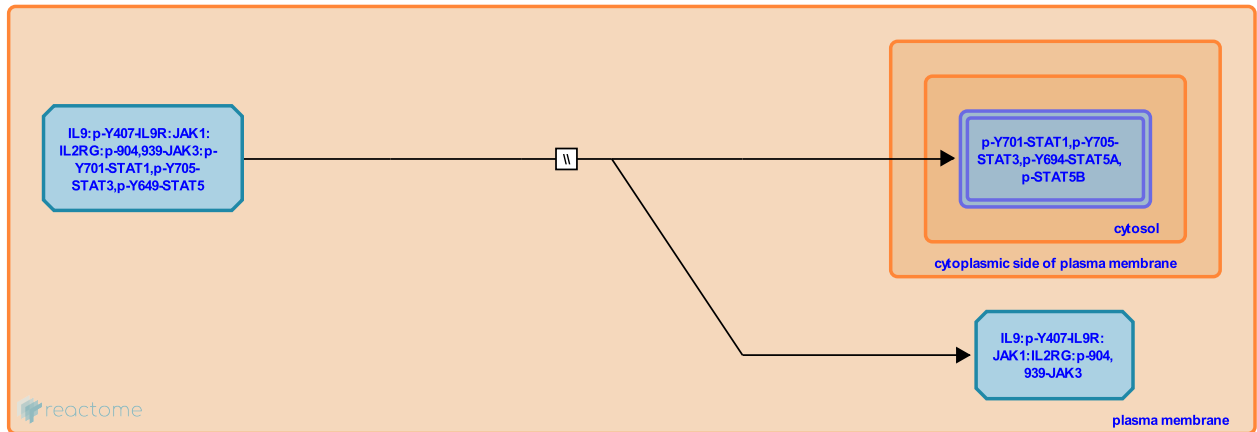
This document contains 1 reaction ([see Table of Contents](#))

## p-Y701-STAT1, p-Y705-STAT3, p-Y649-STAT5 dissociates from IL9:p-Y407-IL9R:JAK1:IL2RG:p-904,939-JAK3:p-Y705-STAT3 [↗](#)

**Stable identifier:** R-HSA-8985900

**Type:** omitted

**Compartments:** cytosol, extracellular region, plasma membrane



Signal transducer and activator of transcription 1-alpha/beta (STAT1), STAT3 and STAT5A/STAT5B (collectively termed STAT5) are believed to dissociate from the Interleukin 9:Interleukin 9 receptor complex.

STAT1 (Demoulin et al, 1996), STAT3 (Yamasaki et al. 2010) and STAT5 (Demoulin et al. 2000) have been identified as parts of complexes associated with DNA in the nucleus following IL9 stimulation. All 3 STATs are believed to follow the standard model for STAT signaling, namely dissociation from the receptor complex, dimerization and translocation to the nucleus (Demoulin et al. 2000, Demoulin et al. 2000, Levy & Darnell 2002).

### Literature references

- Uyttenhove, C., Demoulin, JB., Van Snick, J., Van Roost, E., Donckers, D., DeLestré, B. et al. (1996). A single tyrosine of the interleukin-9 (IL-9) receptor is required for STAT activation, antiapoptotic activity, and growth regulation by IL-9. *Mol. Cell. Biol.*, 16, 4710-6. [↗](#)
- Uyttenhove, C., Groner, B., Demoulin, JB., Renauld, JC., Lejeune, D., Mui, A. (2000). STAT5 activation is required for interleukin-9-dependent growth and transformation of lymphoid cells. *Cancer Res.*, 60, 3971-7. [↗](#)
- Demoulin, JB., Van Snick, J., Renauld, JC. (2001). Interleukin-9 (IL-9) induces cell growth arrest associated with sustained signal transducer and activator of transcription activation in lymphoma cells overexpressing the IL-9 receptor. *Cell Growth Differ.*, 12, 169-74. [↗](#)
- Koussih, L., Yamasaki, A., Halayko, AJ., Saleh, A., Muro, S., Gounni, AS. (2010). IL-9 induces CCL11 expression via STAT3 signalling in human airway smooth muscle cells. *PLoS ONE*, 5, e9178. [↗](#)

### Editions

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