

p-Y-STAT1,3,5 dimerize

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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. 7
- Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics*, 33, 3461-3467. A
- 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. *オ*

This document contains 1 reaction (see Table of Contents)

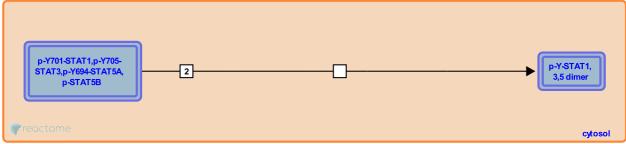
p-Y-STAT1,3,5 dimerize 🛪

Stable identifier: R-HSA-9674542

Type: transition

Compartments: cytosol

Inferred from: p-Y-Stat1,3,5 dimerize (Mus musculus)



After being phosphorylated, STAT1, STAT3, and STAT5 (STAT5A and STAT5B) form homodimers (Tian et al. 1994, Tian et al. 1996, Ward et al. 1999, also inferred from mouse homologs). Phospho-STAT1 and phospho-STAT3 can also form heterodimers (Tian et al. 1994, Ward et al. 1999, also inferred from mouse homologs).

Literature references

- Tian, SS., Seidel, HM., Rosen, J., Stein, RB., Lamb, P. (1994). Rapid activation of the STAT3 transcription factor by granulocyte colony-stimulating factor. *Blood*, *84*, 1760-4.
- Schelen, AM., Smith, L., Ward, AC., Touw, IP., Hermans, MH., Antonissen, C. et al. (1999). Tyrosine-dependent and independent mechanisms of STAT3 activation by the human granulocyte colony-stimulating factor (G-CSF) receptor are differentially utilized depending on G-CSF concentration. *Blood*, *93*, 113-24.
- Tian, SS., Rosen, J., Tapley, P., Stein, RB., Sincich, C., Lamb, P. (1996). Multiple signaling pathways induced by granulocyte colony-stimulating factor involving activation of JAKs, STAT5, and/or STAT3 are required for regulation of three distinct classes of immediate early genes. *Blood, 88*, 4435-44.

Editions

2020-01-13	Authored, Edited	May, B.
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