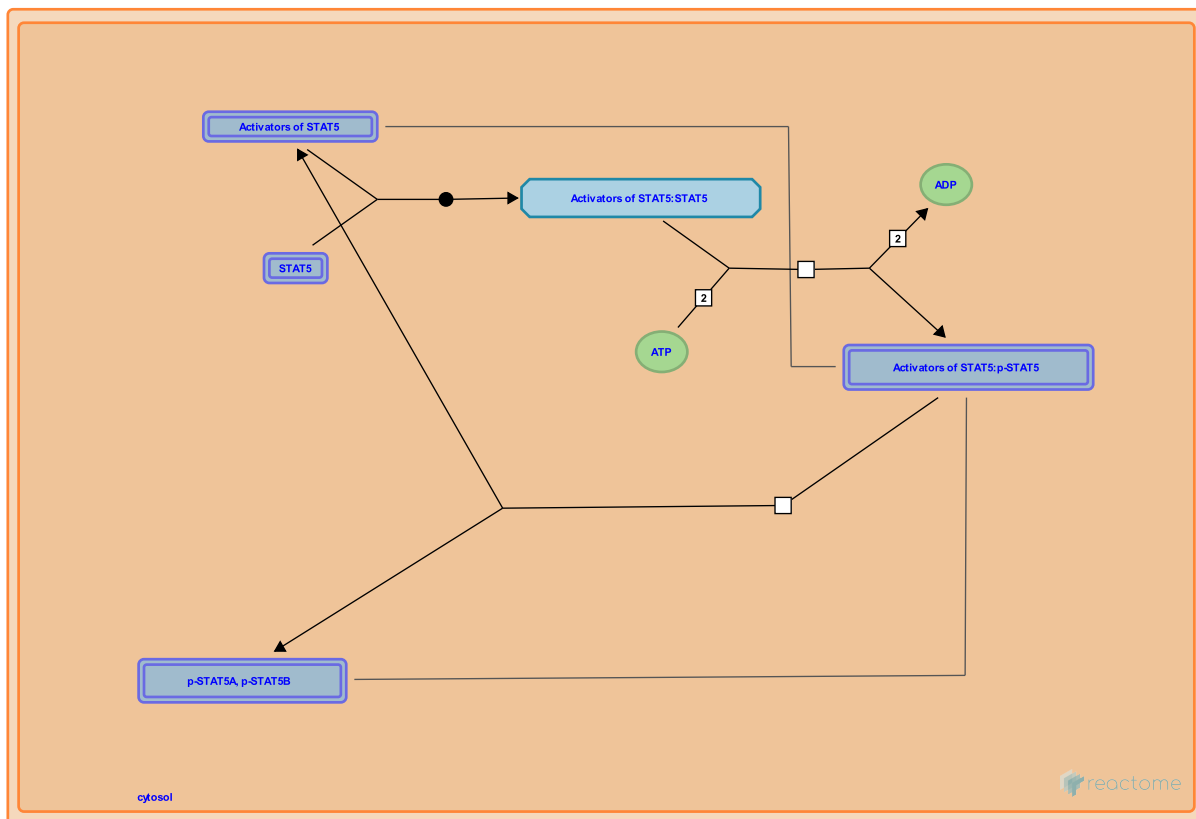


STAT5 Activation



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This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the [Reactome Textbook](https://reactome.org/textbook).

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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.

The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

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Reactome database release: 89

This document contains 1 pathway and 3 reactions ([see Table of Contents](#))

Activators bind STAT5 ↗

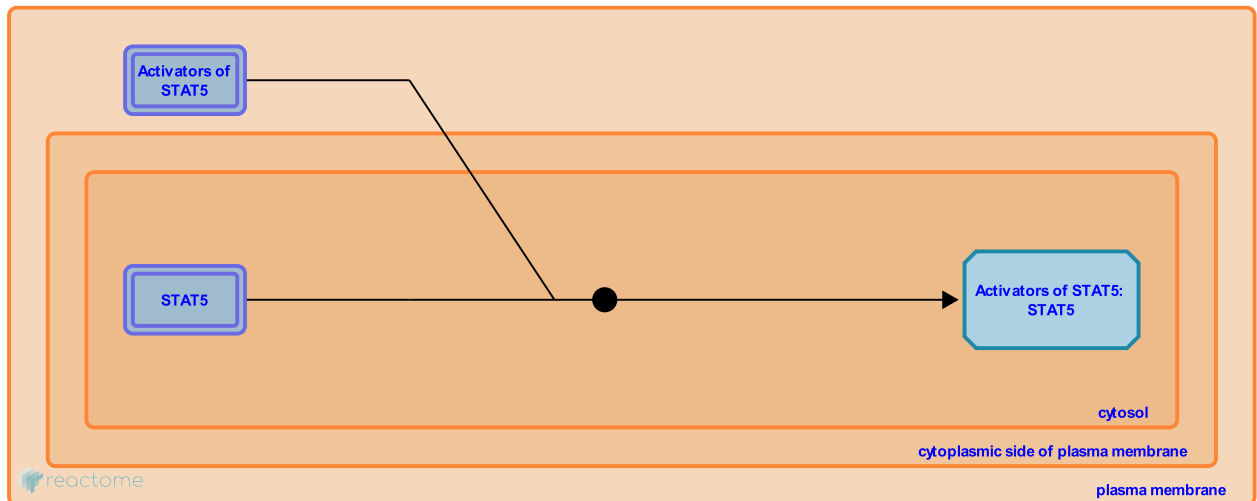
Location: [STAT5 Activation](#)

Stable identifier: R-HSA-9645126

Type: binding

Compartments: cytosol

Inferred from: [Activators bind Stat5 \(Mus musculus\)](#)



Signal transducer and activator of transcription (STAT5A/STAT5B) is a key component in several cell signaling and gene transcription process and regulated cell survival and proliferation. STAT5 is activated by multiple upstream activators including cytokines and growth factors. The process begins when an active upstream regulator binds STAT5 to initiate its phosphorylation (Nabinger et al, 2013).

Followed by: [Activator phosphorylates STAT5](#)

Literature references

Boswell, HS., Ramdas, B., Feng, GS., Zeng, L., Kapur, R., He, Y. et al. (2013). The protein tyrosine phosphatase, Shp2, positively contributes to FLT3-ITD-induced hematopoietic progenitor hyperproliferation and malignant disease in vivo. *Leukemia*, 27, 398-408. ↗

Editions

2019-02-23	Authored	Varusai, TM.
2019-05-03	Reviewed	Traer, E.
2019-05-21	Reviewed	Joshi, S.

Activator phosphorylates STAT5 ↗

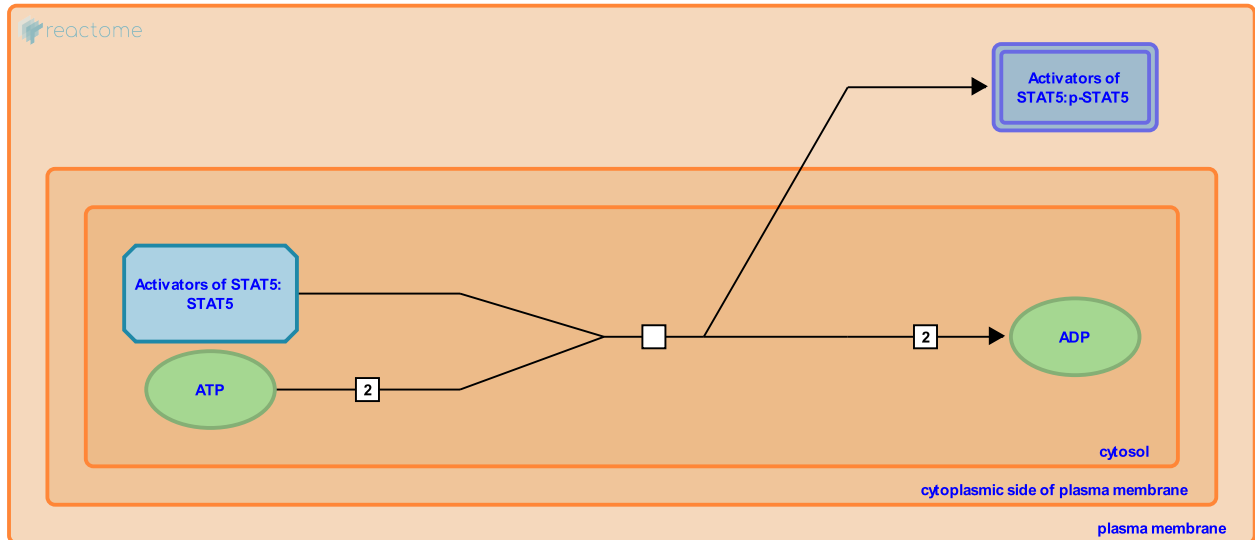
Location: [STAT5 Activation](#)

Stable identifier: R-HSA-9645137

Type: transition

Compartments: cytosol

Inferred from: [Activator phosphorylates Stat5 \(Mus musculus\)](#)



Signal transducer and activator of transcription (STAT5A/STAT5B) is regulated by extracellular factors including cytokines and growth factors. Activators bind and phosphorylate STAT5 which results in its activation (Nabinger et al, 2013).

Preceded by: [Activators bind STAT5](#)

Followed by: [p-STAT5 dissociates from activator:STAT5 complex](#)

Literature references

Boswell, HS., Ramdas, B., Feng, GS., Zeng, L., Kapur, R., He, Y. et al. (2013). The protein tyrosine phosphatase, Shp2, positively contributes to FLT3-ITD-induced hematopoietic progenitor hyperproliferation and malignant disease in vivo. *Leukemia*, 27, 398-408. ↗

Editions

2019-02-23	Authored	Varusai, TM.
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2019-05-21	Reviewed	Joshi, S.

p-STAT5 dissociates from activator:STAT5 complex ↗

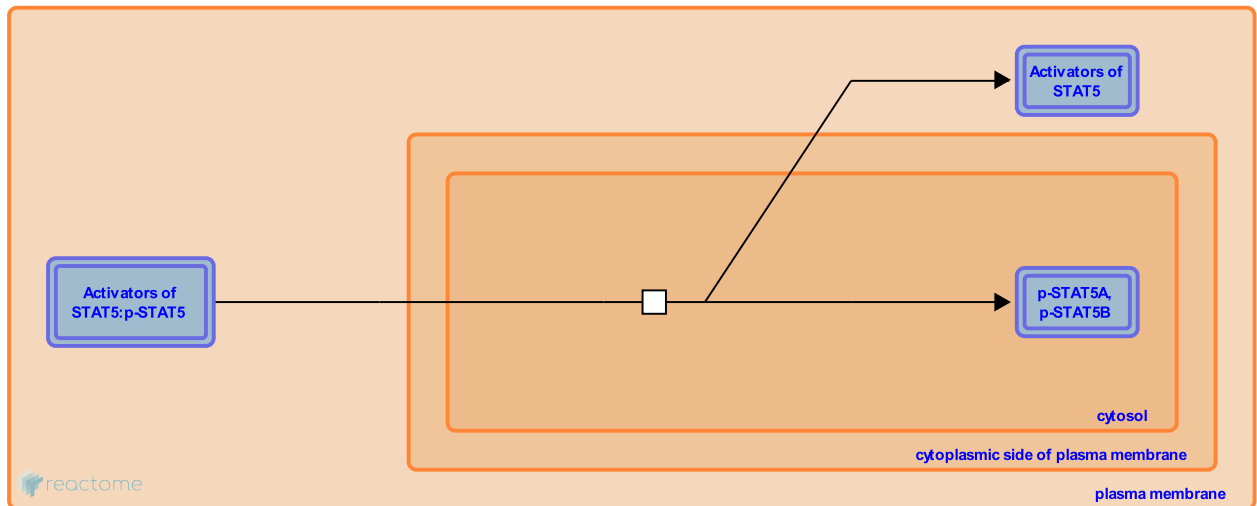
Location: [STAT5 Activation](#)

Stable identifier: R-HSA-9645134

Type: transition

Compartments: cytosol

Inferred from: [p-Stat5 dissociates from activator:Stat5 complex \(Mus musculus\)](#)



Signal transducer and activator of transcription (STAT5A/STAT5B) is activated by a range of upstream signals including cytokines and growth factors. Activators bind and phosphorylate STAT5 following which STAT5 is released from the complex and translocates to the nucleus to regulate gene transcription (Nabinger et al, 2013).

Preceded by: [Activator phosphorylates STAT5](#)

Literature references

Boswell, HS., Ramdas, B., Feng, GS., Zeng, L., Kapur, R., He, Y. et al. (2013). The protein tyrosine phosphatase, Shp2, positively contributes to FLT3-ITD-induced hematopoietic progenitor hyperproliferation and malignant disease in vivo. *Leukemia*, 27, 398-408. ↗

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