

Phosphorylation of IL2RB Y338, Y392 or Y510 enables STAT recruitment

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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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Reactome database release: 75

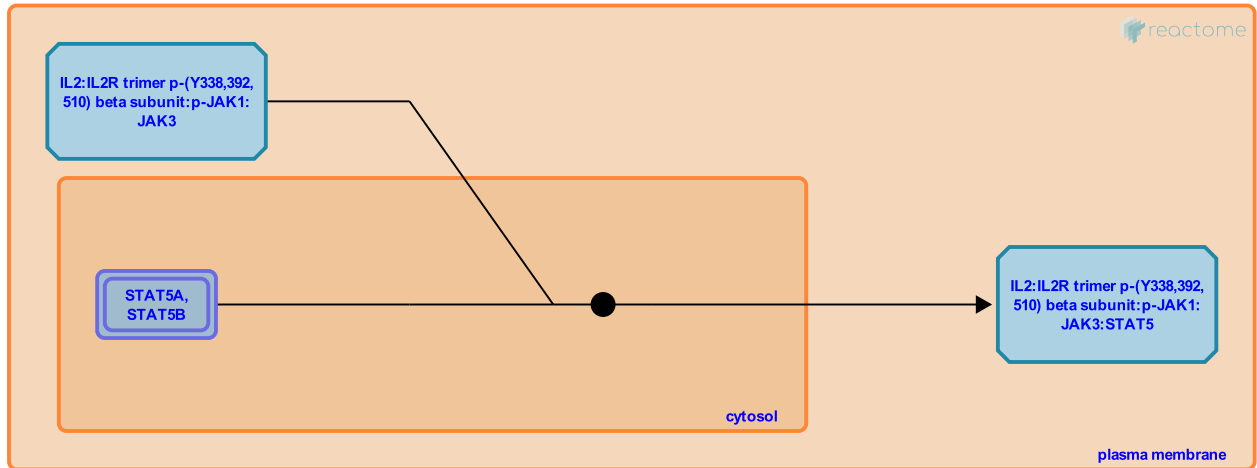
This document contains 1 reaction ([see Table of Contents](#))

Phosphorylation of IL2RB Y338, Y392 or Y510 enables STAT recruitment [↗](#)

Stable identifier: R-HSA-452108

Type: binding

Compartments: cytosol, plasma membrane



Mutation analysis has shown that Y338, Y392 and Y510 are involved in IL-2-induced STAT protein binding. Phospho-tyrosines 338, 392 and 510 can each promote STAT5 activation (Gaffen et al. 1996), though Y510 appears to be the primary site for STAT5 binding (Gesbert et al. 1998). STAT3 may also be recruited to phospho-tyrosines on IL2RB and studies have shown defective IL-2 responses in STAT3^{-/-} T cells, thereby supporting a functional role for STAT3 downstream of IL-2 signaling (Akaishi et al. 1998).

Literature references

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Gaffen, SL., Lai, SY., Ha, M., Liu, X., Hennighausen, L., Greene, WC. et al. (1996). Distinct tyrosine residues within the interleukin-2 receptor beta chain drive signal transduction specificity, redundancy, and diversity. *J Biol Chem*, 271, 21381-90. [↗](#)

Editions

2010-05-17	Authored	Ray, KP.
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