JAK3 in IL7:p-Y449-IL7R:JAK1:IL2RG:JAK3 is phosphorylated

Duenas, C., Kumar, U.
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


Reactome database release: 83

This document contains 1 reaction (see Table of Contents)
Interleukin-7 (IL7) signaling is believed to resemble that of other gammaC family receptors, based on detailed studies of the Interleukin-2 receptor. Extending this model to IL7 suggested a series of events that bring Tyrosine-protein kinase JAK1 (JAK1) and Tyrosine-protein kinase JAK3 (JAK3) into proximity within the complex IL7:IL7R:JAK1:IL2RG:JAK3. Cytoplasmic domains of the receptor chains re-orient so that their associated kinases (JAKs and possibly phosphatidylinositol 3-kinases) can phosphorylate sequence elements on the cytoplasmic domains (Jiang et al. 2005). Tyrosine-449 (Y449) in the cytoplasmic domain of Interleukin-7 receptor is required for T-cell development in vivo and for activation of the JAK/STAT5 and PI3K/Akt pathways (Jiang et al. 2004, Pallard et al. 1999).

It has been suggested that JAK1 phosphorylates IL7R (Jiang et al. 2004) and it is believed that JAK3, associated with IL2RG, phosphorylates the tyrosine residues in the cytoplasmic portion of IL7R that lead to recruitment of STATs (Fry & Mackall 2002). This is consistent with the lack of intrinsic tyrosine kinase activity in IL7R:JAK1 in the absence of IL2RG:JAK3 (Lai et al. 1996).

**Literature references**


**Editions**

<table>
<thead>
<tr>
<th>Edition</th>
<th>Action</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>2017-07-20</td>
<td>Authored</td>
<td>Duenas, C.</td>
</tr>
<tr>
<td>2017-07-26</td>
<td>Edited</td>
<td>Duenas, C.</td>
</tr>
<tr>
<td>2017-07-26</td>
<td>Reviewed</td>
<td>Kumar, U.</td>
</tr>
</tbody>
</table>