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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


**Stable identifier:** R-MMU-9007137

**Type:** omitted

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

Grb2 associated binding protein 2 (Gab2) is phosphorylated in response to Interleukin-2 (IL2) and Interleukin-15 (IL15) stimulation. Its phosphorylation is greatly diminished by mutation of the site in the Interleukin-2 Receptor beta chain (Y338F) (IL2rb, IL2rβ) that recruits SHC transforming protein 1 (Shc1) (Gadina et al. 2000, Wöhrle et al. 2009, Gesbert et al. 1998, Brockdorff et al. 2001). The IL15 receptor complex consists mainly of IL15, Interleukin-15 receptor alpha subunit (IL15ra, IL15rα), IL2rb, which is associated with Jak1 and Interleukin receptor 2 gamma subunit, which is associated with Jak3 (Johnston et al. 1995). In this stage, it also includes a phosphorylated molecule of Shc1, a Grb2 and Gab2. This is a black box event because the kinase responsible for Gab2 phosphorylation has not been demonstrated.

More in detail, human and mouse IL15 have 70.2% amino acid sequence similarity and exhibit similar trans-presentation mechanism, signal transduction machinery and biological activities. Similarly, human IL15 shows cross-reactivity with mouse cells and it was demonstrated that human and mouse IL15 showed similar responses in mouse models (Stoklasek et al. 2006) (Patidar et al. data not published).

**Literature references**


**Editions**

| 2017-08-07 | Authored, Edited | Duenas, C. |
| 2017-08-07 | Reviewed | Patidar, M. |

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