IL17RA and IL17RC bind IL17A dimer, IL17F dimer, IL17A:IL17F

Jupe, S., Meldal, BH., Messina, F.
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


Reactome database release: 82

This document contains 1 reaction (see Table of Contents)
IL17RA and IL17RC bind IL17A dimer, IL17F dimer, IL17A:IL17F

Stable identifier: R-HSA-447246

Type: omitted

Compartments: extracellular region, plasma membrane

The Interleukin-17 receptor A/C complex is a dimer of Interleukin-17 receptor A (IL17RA) and Interleukin-17 receptor C (IL17RC). It binds interleukin-17A (IL17A) dimers, IL17F dimers and IL17A:IL17F heterodimers (IL17A:IL17F) (Yao et al. 1995, Toy et al. 2006). IL17A and IL17F homodimers tend to disfavor binding of a second molecule of IL17RA to the receptor/ligand complex (Liu et al. 2013). Engagement of IL17RA or IL17RC by IL17A or IL17F brings about an allosteric 'preference' for the second receptor-biding site to engage a different receptor to form a heterodimeric receptor complex (Ely et al. 2003) This is a Black Box event because the order of receptor binding is unclear.

Secreted SARS-CoV-2 ORF8 (8) binds to IL17RA and activates IL17 signaling pathway leading to an increased secretion of cytokines/chemokines thus contributing to cytokine storm during SARS-CoV-2 infection (Lin X et al. 2021).

Literature references


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