IRAK1/or IRAK2 binds to the activated IRAK4 :oligo MyD88:activated TLR5 or 10 complex

Gillespie, ME., Li, L., Shamovsky, V.
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: 83

This document contains 1 reaction (see Table of Contents)

https://reactome.org
IRAK1/or IRAK2 binds to the activated IRAK4:oligo MyD88:activated TLR5 or 10 complex

**Stable identifier:** R-HSA-975852

**Type:** binding

**Compartments:** plasma membrane, cytosol

IRAK2 has been implicated in IL1R and TLR signaling by the observation that IRAK2 can associate with MyD88 and Mal (Muzio et al. 1997). Like IRAK1, IRAK2 is activated downstream of IRAK4 (Kawagoe et al. 2008). It has been suggested that IRAK1 activates IRAK2 (Wesche et al. 1999) but IRAK2 phosphorylation is observed in IRAK1−/− mouse macrophages while IRAK4 deficiency abrogates IRAK2 phosphorylation (Kawagoe et al. 2008), suggesting that activated IRAK4 phosphorylates IRAK2 as it does IRAK1. IL6 production in response to IL1beta is impaired in embryonic fibroblasts from IRAK1 or IRAK2 knockout mice and abrogated in IRAK1/2 dual knockouts (Kawagoe et al. 2007) suggesting that IRAK1 and IRAK2 are both involved in IL1R signaling downstream of IRAK4.

MYD88 recruits unphosphorylated, inactive IRAK1 to the IL1 receptor complex.

**Literature references**


**Editions**

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