

p100:RELB binds active NIK:p-IKKA dimer

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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: 74

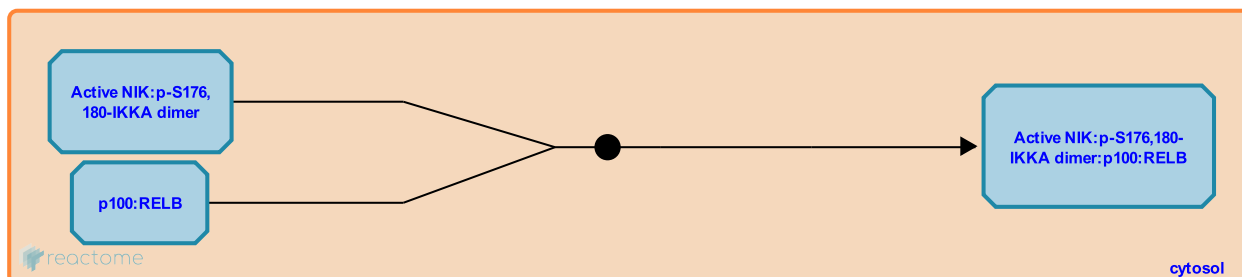
This document contains 1 reaction ([see Table of Contents](#))

p100:RELB binds active NIK:p-IKKA dimer ↗

Stable identifier: R-HSA-5607720

Type: binding

Compartments: cytosol



NFKB2 (also known as p100) is a member of the NF- κ B family of transcription factors. It is synthesised as large precursor with an N-terminal RHD (Rel homology domain) and a C-terminal series of ankyrin repeats that masks the nuclear localization signal of NFKB2/p100 localising it to the cytosol. In resting cells, p100 is associated with RELB (Transcription factor RelB) in the cytosol. Upon cell stimulation, the I κ B-like C terminus of p100 is proteolyzed, resulting in RELB-p52 dimers that translocate to the nucleus (Senftleben et al. 2001, Hayden & Ghosh 2004). IKKA (I kappa-B kinase alpha) does not associate directly with p100 but in the presence of NIK (NF- κ B-inducing kinase), IKKA stably binds to p100. Serine residues 866 and 870 of p100 are essential for the recruitment of IKKA to p100 by NIK. This interaction is required for p100 phosphorylation and subsequent processing by IKKA (Xiao et al. 2001, 2004).

Literature references

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Editions

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