TRAF3 binds BAFFR:BAFF

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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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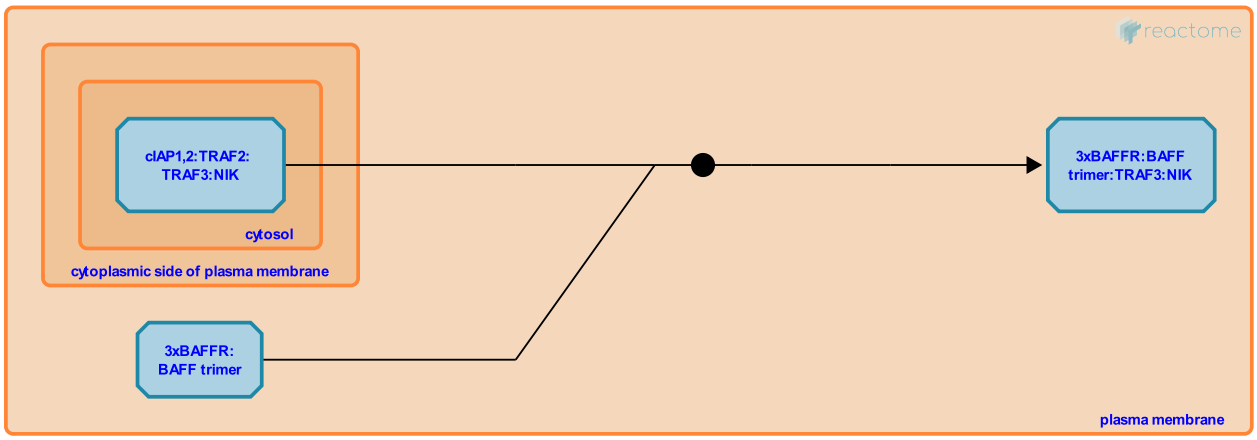
TRAF3 binds BAFFR:BAFF ↗

Stable identifier: R-HSA-5676596

Type: binding

Compartments: plasma membrane, cytosol



In the absence of BAFFR (TNFSF13C) ligation to BAFF (TNFSF13B) ligand, NFkB-inducing kinase (NIK) forms a complex with TNF receptor associated factor 3 (TRAF3) and TRAF2 which exists in a preassembled complex with cellular Inhibitor of apoptosis 1 (cIAP1) and cIAP2 in the cytosol. cIAP1/2 targets NIK for degradation by ubiquitylation, there by inhibiting non-canonical NFkB pathway (Vallabhapurapu et al. 2008, Zarnegar et al. 2008). Upon BAFF trimer binding to BAFFR, TRAF3 but not TRAF2 is recruited to the receptor via a 'PVPAT' binding site. This unique feature of BAFFR to recruit TRAF3 instead of TRAF2 is primarily due to its possession of an atypical TRAF-binding sequence (Morrison et al 2005). Following recruitment to BAFFR, TRAF3 undergoes proteasomal degradation, a process which requires TRAF2 and cIAP1/2.

Literature references

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Editions

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