IKKB phosphorylates SNAP23

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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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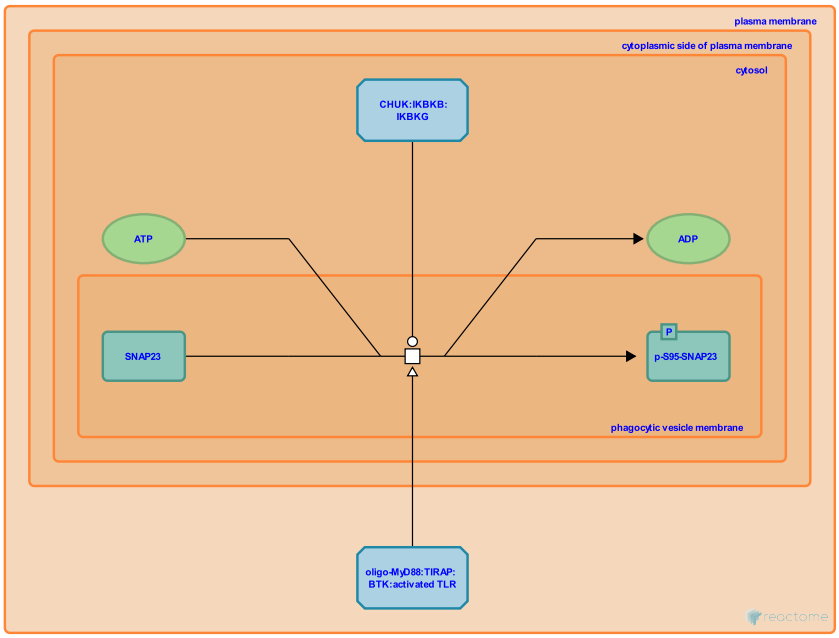
Stable identifier: R-HSA-8863895

Type: transition

Compartments: phagocytic vesicle membrane, cytosol



A major reserve of MHC-I in dendritic cells reside within the endocytic recycling compartments (ERC). MHC-I trafficking to the ERC is regulated by the activity of Rab11a and subsequent trafficking from ERC to phagosomes is controlled by TLR-MyD88-IKK2-dependent phosphorylation of phagosomal SNAP23. Toll-like receptor (TLR) signalling regulate cross-presentation as they regulate phagocytosis and phagolysosomal fusion (Nair et al. 2011). MHC-I bearing ERC are enriched with R-SNAREs like RAB11a, VAMP3, and VAMP8. These SNARE molecules can interact with Q-SNARE SNAP23 present on phagosomes and this mediates membrane fusion. This interaction of SNAP23 with R-SNAREs require phosphorylation of SNAP23 (on Ser-95) by IKK2, and IKK2 is activated by TLR signalling. SNAP23 phosphorylation may increase SNAP23 binding to SNAREs. It may also regulate platelet and mast cell secretion (Karim et al. 2013, Suziki & Verma 2008).

Literature references

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Editions

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