TBC1D14 binds RAB11 and ULK1

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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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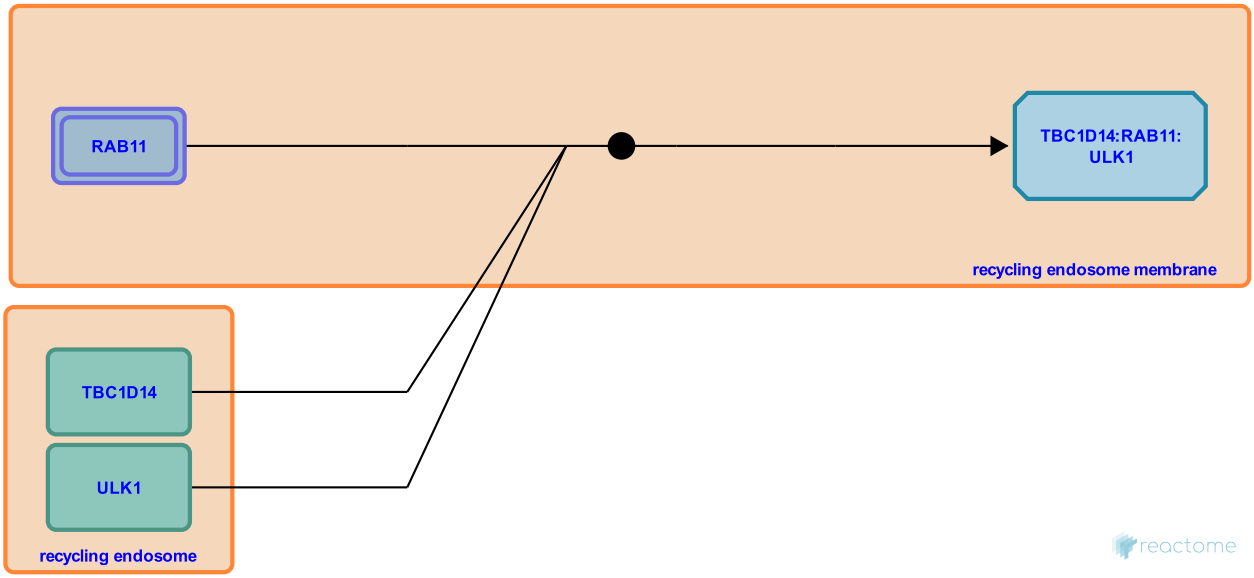
Stable identifier: R-HSA-8854759

Type: binding

Compartments: recycling endosome membrane



TBC (Tre2/Bub2/Cdc16) domain family, member 14 (TBC1D14) is a putative Rab GTPase activating protein (GAP) member that binds to activated Rab11 and regulates starvation-induced autophagy. TBC1D14 does not have the GAP activity and may function as Rab11 effector. TBC1D14 also colocalizes and interacts with the autophagy kinase ULK1. Overexpression of TBC1D14 causes tubulation of recycling endosomes (REs), accumulation of the ULK1 complex and Rab11 on REs, and inhibition of vesicular transport from the RE (Longatti et al. 2012).

Literature references

Longatti, A., Lamb, CA., Razi, M., Yoshimura, S., Barr, FA., Tooze, SA. (2012). TBC1D14 regulates autophagosome formation via Rab11- and ULK1-positive recycling endosomes. *J. Cell Biol., 197*, 659-75.

Editions

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