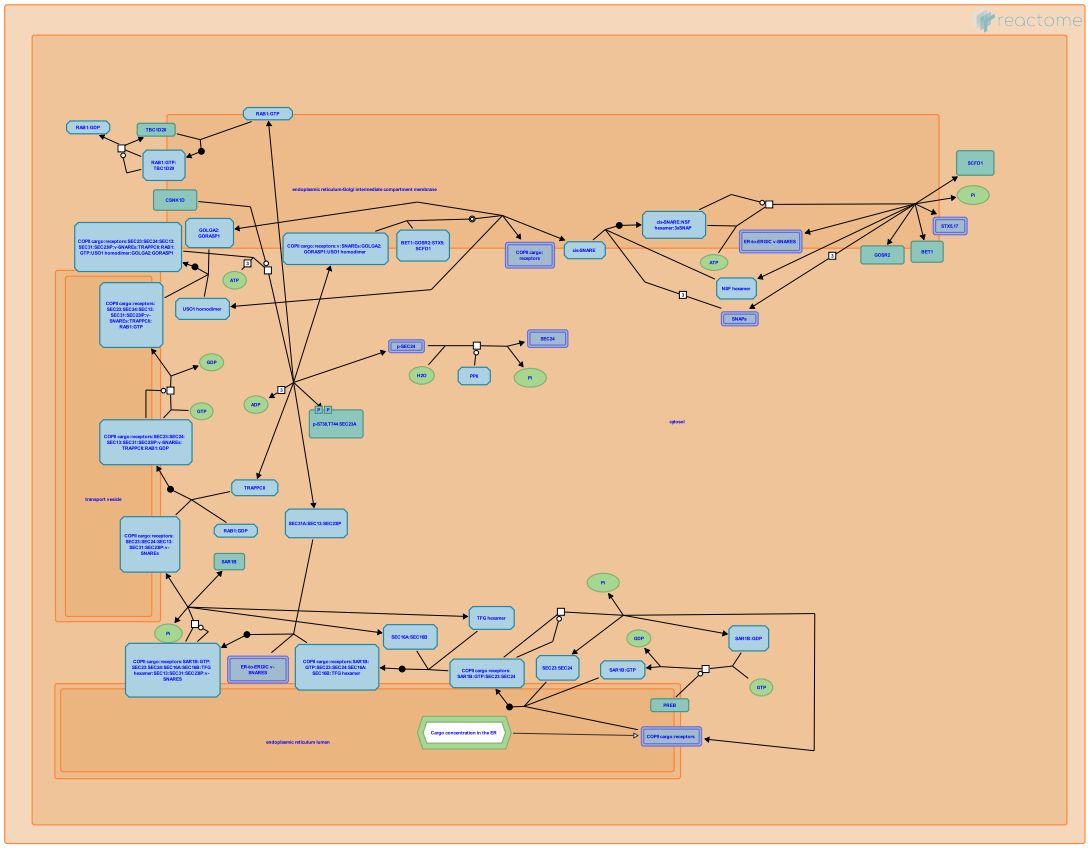
COPII-mediated vesicle transport



Gagneux, P., Gillespie, ME., Jassal, B., Rothfels, K.

European Bioinformatics Institute, New York University Langone Medical Center, Ontario Institute for Cancer Research, Oregon Health and Science University.

This is just an excerpt of a full-length report for this pathway. To access the complete report, please download it at the Reactome Textbook.

03/05/2024

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.
The development of Reactome is supported by grants from the US National Institutes of Health (P41 HG003751), University of Toronto (CFREF Medicine by Design), European Union (EU STRP, EMI-CD), and the European Molecular Biology Laboratory (EBI Industry program).

Literature references

Fabregat, A., Sidiropoulos, K., Viteri, G., Forner, O., Marin-Garcia, P., Arnau, V. et al. (2017). Reactome pathway analysis: a high-performance in-memory approach. *BMC bioinformatics, 18*, 142. ↗

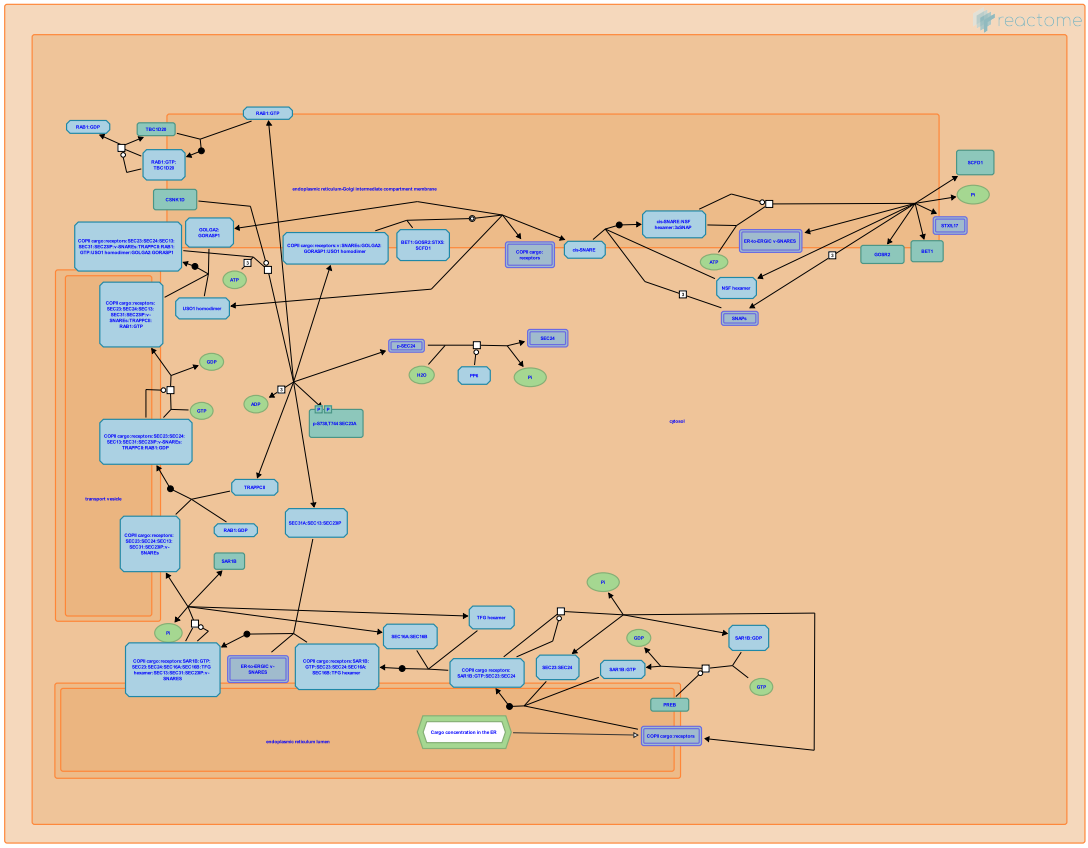
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

Fabregat, A., Korninger, F., Viteri, G., Sidiropoulos, K., Marin-Garcia, P., Ping, P. et al. (2018). Reactome graph database: Efficient access to complex pathway data. *PLoS computational biology, 14*, e1005968. ↗

COPII-mediated vesicle transport ↗

Stable identifier: R-HSA-204005



COPII components (known as Sec13p, Sec23p, Sec24p, Sec31p, and Sar1p in yeast) traffic cargo from the endoplasmic reticulum to the ER-Golgi intermediate compartment (ERGIC). COPII-coated vesicles were originally discovered in the yeast Saccharomyces cerevisiae using genetic approaches coupled with a cell-free assay. The mammalian counterpart of this pathway is represented here. Newly synthesized proteins destined for secretion are sorted into COPII-coated vesicles at specialized regions of the ER. These vesicles leave the ER, become uncoated and subsequently fuse with the ERGIC membrane.

Literature references

Nakano, A., Sato, K. (2007). Mechanisms of COPII vesicle formation and protein sorting. *FEBS Lett, 581*, 2076-82. ↗

Kirchhausen, Tomas. (2000). Three ways to make a vesicle. *Nat Rev Mol Cell Biol, 1*, 187-98. ↗

Editions

2007-07-14	Authored	Gillespie, ME.
2007-11-22	Edited	Gillespie, ME.
2010-11-18	Reviewed	Gagneux, P.
2015-04-18	Revised	Rothfels, K.
2015-08-18	Revised	Gillespie, ME.

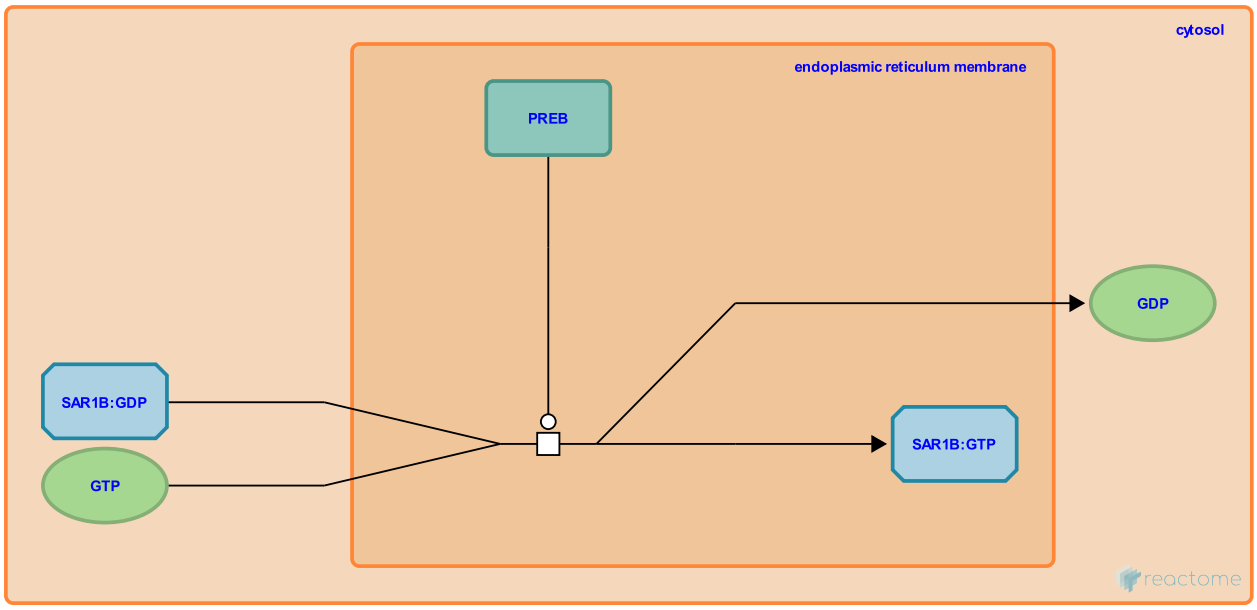
SAR1 Activation And Membrane Binding ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-203977

Type: transition

Compartments: endoplasmic reticulum membrane, cytosol



SAR1-GDP is recruited to the ER membrane by the transmembrane GEF (Guanine nucleotide exchange factor) PREB (also known as SEC12), where it is converted to SAR1-GTP. PREB itself is recruited to ER exit sites through interaction with MIA2 (also known as CTAGE5).

Preceded by: Loss of SAR1B GTPase

Followed by: Inner coat assembly and cargo binding

Literature references

Paccaud, JP., Lin-Marq, N., Stephens, DJ., Pagano, A., Pepperkok, R. (2000). COPI-coated ER-to-Golgi transport complexes segregate from COPII in close proximity to ER exit sites. *J Cell Sci, 113*, 2177-85. ↗

Saito, K., Yamashiro, K., Tanabe, T., Shimazu, N., Katada, T., Kontani, K. (2014). Concentration of Sec12 at ER exit sites via interaction with cTAGE5 is required for collagen export. *J. Cell Biol., 206*, 751-62. ↗

Glick, BS., Hammond, AT. (2000). Dynamics of transitional endoplasmic reticulum sites in vertebrate cells. *Mol Biol Cell, 11*, 3013-30. ↗

Editions

2007-07-14	Authored	Gillespie, ME.
2010-11-10	Edited	Jassal, B.
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2015-08-18	Revised	Gillespie, ME.

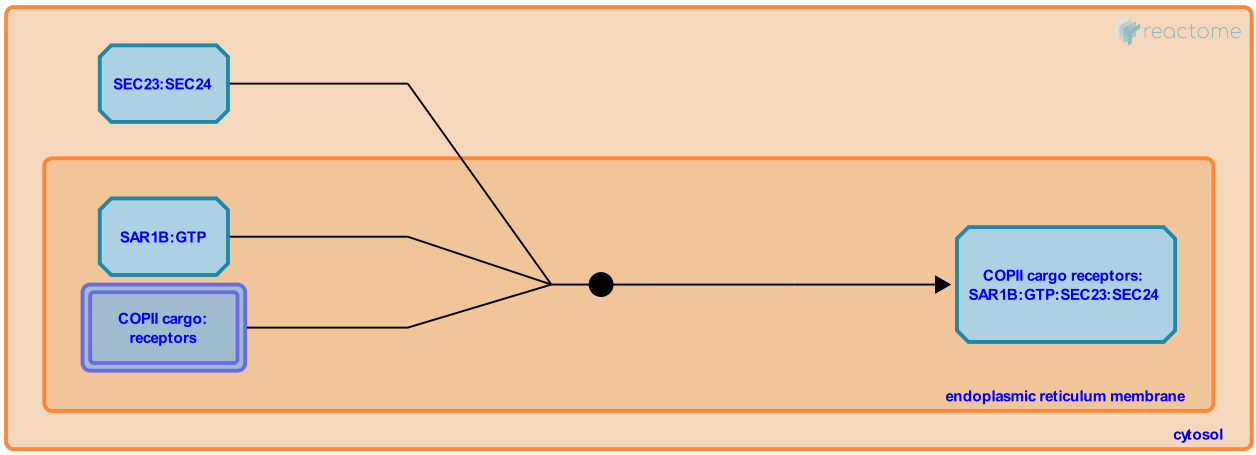
Inner coat assembly and cargo binding ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694522

Type: binding

Compartments: endoplasmic reticulum membrane



SAR1:GTP recruits the cytoplasmic SEC23:SEC24 complex. SEC24, and to a lesser extent SEC23, also mediate interaction with COPII cargo, concentrating it into the emerging vesicle. Transmembrane cargo may interact directly with the inner coat proteins, often in an isoform specific manner; alternately, some transmembrane proteins and all soluble cargo interact first with a cargo receptor of the p24, LMAN or ERV families.

Preceded by: SAR1 Activation And Membrane Binding

Followed by: SEC16 complex binds SAR1B:GTP:SEC23:SEC24, Loss of SAR1B GTPase

Literature references

Paccaud, JP., Lin-Marq, N., Stephens, DJ., Pagano, A., Pepperkok, R. (2000). COPI-coated ER-to-Golgi transport complexes segregate from COPII in close proximity to ER exit sites. *J Cell Sci, 113*, 2177-85. ↗

Glick, BS., Hammond, AT. (2000). Dynamics of transitional endoplasmic reticulum sites in vertebrate cells. *Mol Biol Cell, 11*, 3013-30. ↗

Editions

2010-11-18	Reviewed	Gagneux, P.
2015-03-28	Authored, Edited	Rothfels, K.
2015-08-18	Reviewed	Gillespie, ME.

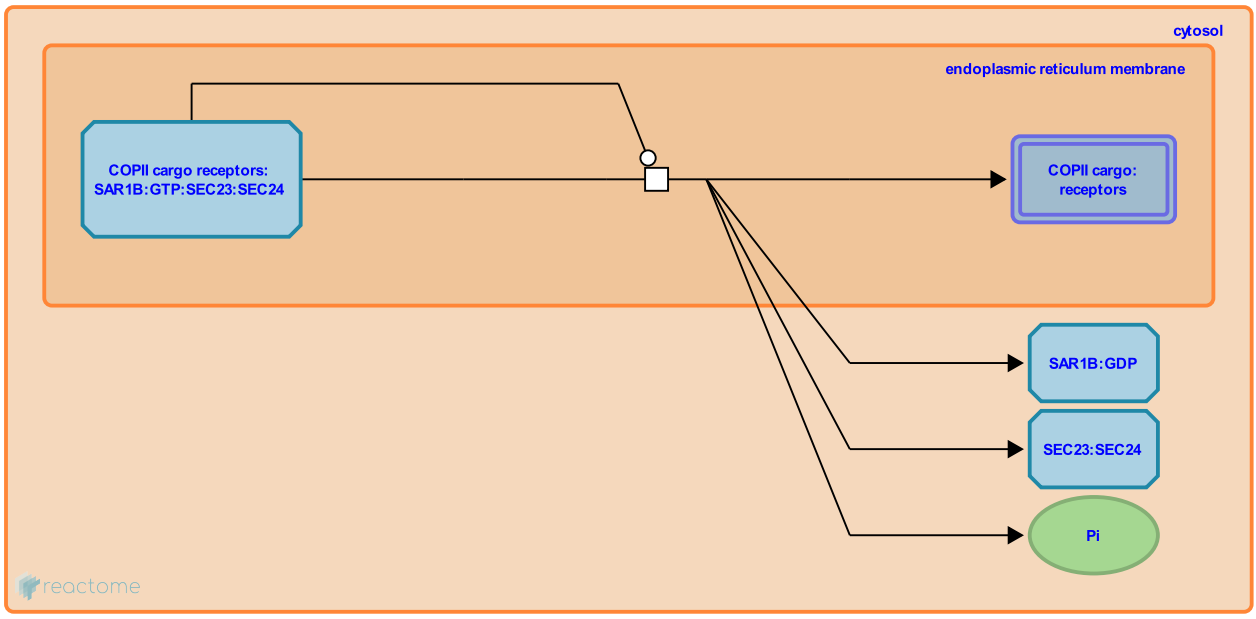
Loss of SAR1B GTPase ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694527

Type: transition

Compartments: endoplasmic reticulum membrane, cytosol



Sar1p-GTP hydrolysis is increased 15-30-fold by Sec23p. Sar1p-GDP is released as a result of this hydrolysis and used in further vesicle sculpting cycles. Sar1p-GTP hydrolysis occurs at two critical points during the cycle, first (as represented here) as a proofreading step, insuring that the cargo is loaded. Later in the cycle Sar1p-GTP hydrolysis triggers the uncoating of the budded vesicle.

Preceded by: Inner coat assembly and cargo binding

Followed by: SAR1 Activation And Membrane Binding

Literature references

Paccaud, JP., Lin-Marq, N., Stephens, DJ., Pagano, A., Pepperkok, R. (2000). COPI-coated ER-to-Golgi transport complexes segregate from COPII in close proximity to ER exit sites. *J Cell Sci, 113*, 2177-85. ↗

Glick, BS., Hammond, AT. (2000). Dynamics of transitional endoplasmic reticulum sites in vertebrate cells. *Mol Biol Cell, 11*, 3013-30. ↗

Nakano, A., Sato, K. (2007). Mechanisms of COPII vesicle formation and protein sorting. *FEBS Lett, 581*, 2076-82. ↗

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2007-07-14	Authored	Gillespie, ME.
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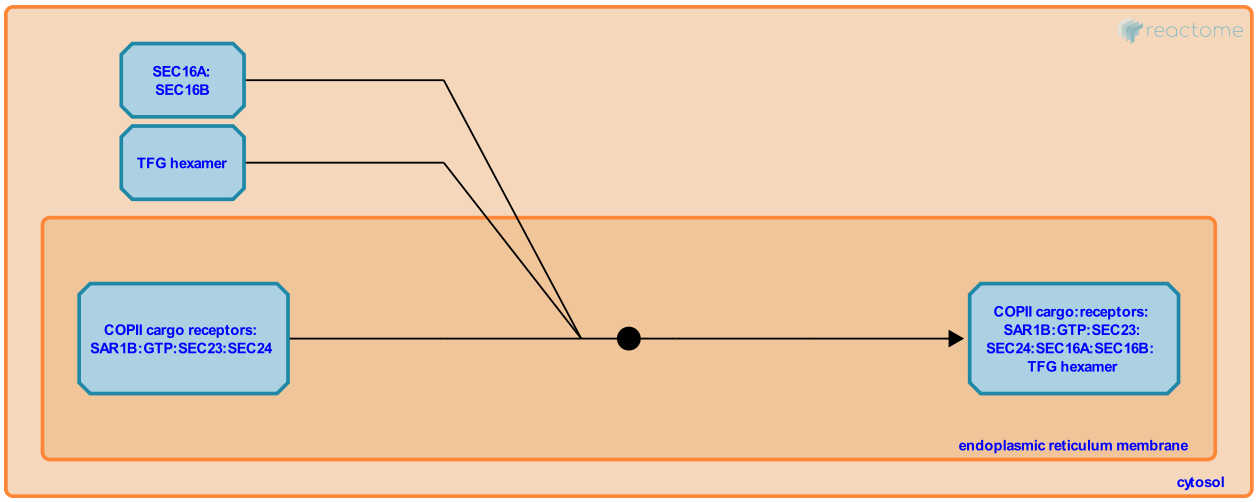
SEC16 complex binds SAR1B:GTP:SEC23:SEC24 ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694417

Type: binding

Compartments: endoplasmic reticulum membrane



The multimeric SEC16 complex marks sites of ER exit (ERES) and helps to localize nascent COPII coat complexes through interaction with SEC23 and SAR1 (Battacharyya et al, 2007; Watson et al, 2006; Hughes et al, 2009; Yorimitsu and Sato, 2012; reviewed in Sprangers and Rabouille, 2015). SEC16 may help to prevent premature dissociation of the COPII coats after activation of SEC13 GTPase (Supek et al, 2002; Kung et al, 2012). SEC16 functions with hexameric TFG1, which is recruited to the ERES through direct interaction with SEC16 and is required for cargo traffic out of the ER and for organization of the ER-to-ERGIC boundary (Witte et al, 2001; Beetz et al, 2012; Johnson et al, 2015).

Preceded by: Inner coat assembly and cargo binding

Followed by: SEC31:SEC13 and v-SNARE recruitment

Literature references

Pagant, S., Kung, LF., D'Arcangelo, JG., Hamamoto, S., Miller, EA., Schekman, R. et al. (2012). Sec24p and Sec16p cooperate to regulate the GTP cycle of the COPII coat. *EMBO J., 31*, 1014-27. ↗

Saxena, R., Schuh, AL., Altmüller, J., Beetz, C., Nürnberg, P., Audhya, A. et al. (2013). Inhibition of TFG function causes hereditary axon degeneration by impairing endoplasmic reticulum structure. *Proc. Natl. Acad. Sci. U.S.A., 110*, 5091-6. ↗

Rabouille, C., Sprangers, J. (2015). SEC16 in COPII coat dynamics at ER exit sites. *Biochem. Soc. Trans., 43*, 97-103. ↗

Audhya, A., Otegui, MS., Bhattacharya, N., Hanna, M., Schuh, AL., Wang, L. et al. (2015). TFG clusters COPII-coated transport carriers and promotes early secretory pathway organization. *EMBO J., 34*, 811-27. ↗

Madden, DT., Orci, L., Hamamoto, S., Supek, F., Schekman, R. (2002). Sec16p potentiates the action of COPII proteins to bud transport vesicles. *J. Cell Biol., 158*, 1029-38. ↗

Editions

2015-03-16	Authored	Rothfels, K.
2015-04-18	Edited	Rothfels, K.
2015-08-18	Reviewed	Gillespie, ME.

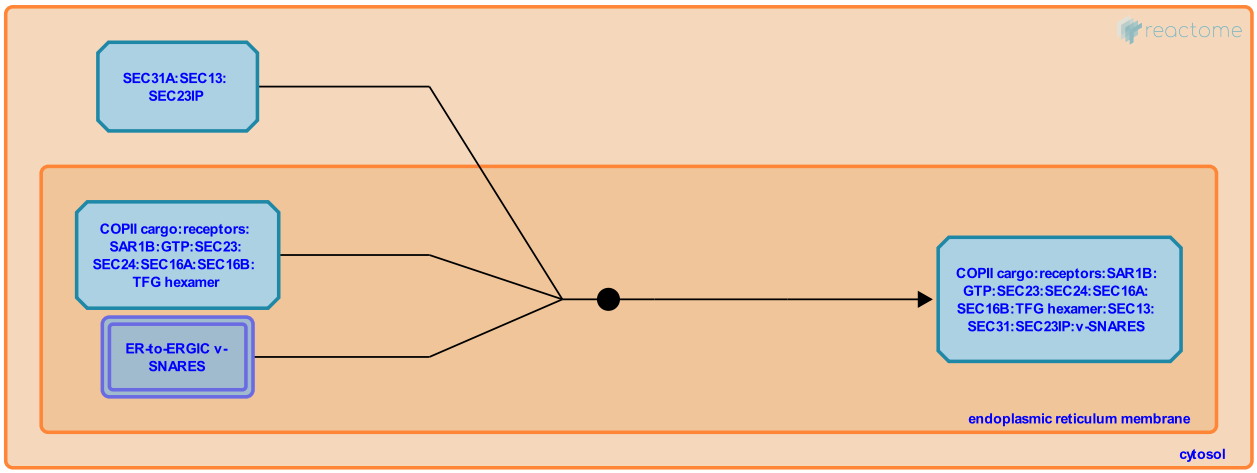
SEC31:SEC13 and v-SNARE recruitment ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-204008

Type: binding

Compartments: endoplasmic reticulum membrane, cytosol



The outer coat proteins SEC13 and SEC31A are thought to exist in a cytosolic heterohexamer with SEC23IP and are recruited to the ER through interaction with pre-bound SEC23-SEC24 complexes (Stephens et al, 2000; Hammond and Glick, 2000; Ong et al, 2010; reviewed in Szul and Sztul, 2011). v-SNAREs such as the SEC22 proteins are also incorporated into the emerging vesicle through interactions with components of the COPII coat (Xu et al, 2000; Mancias and Goldberg, 2008; Gordon et al, 2010; reviewed in Szul and Sztul, 2011; Lord et al, 2013).

Preceded by: SEC16 complex binds SAR1B:GTP:SEC23:SEC24

Followed by: Vesicle budding

Literature references

Sztul, E., Szul, T. (2011). COPII and COPI traffic at the ER-Golgi interface. *Physiology (Bethesda), 26*, 348-64. ↗

Paccaud, JP., Lin-Marq, N., Stephens, DJ., Pagano, A., Pepperkok, R. (2000). COPI-coated ER-to-Golgi transport complexes segregate from COPII in close proximity to ER exit sites. *J Cell Sci, 113*, 2177-85. ↗

Xu, D., Joglekar, AP., Williams, AL., Hay, JC. (2000). Subunit structure of a mammalian ER/Golgi SNARE complex. *J. Biol. Chem., 275*, 39631-9. ↗

Ferro-Novick, S., Miller, EA., Lord, C. (2013). The highly conserved COPII coat complex sorts cargo from the endoplasmic reticulum and targets it to the golgi. *Cold Spring Harb Perspect Biol, 5*. ↗

Hong, W., Loo, LS., Ong, YS., Tang, BL. (2010). p125A exists as part of the mammalian Sec13/Sec31 COPII subcomplex to facilitate ER-Golgi transport. *J. Cell Biol., 190*, 331-45. ↗

Editions

2007-07-14	Authored	Gillespie, ME.
2010-11-17	Edited	Jassal, B.
2010-11-18	Reviewed	Gagneux, P.
2015-03-16	Revised	Rothfels, K.
2015-08-18	Revised	Gillespie, ME.

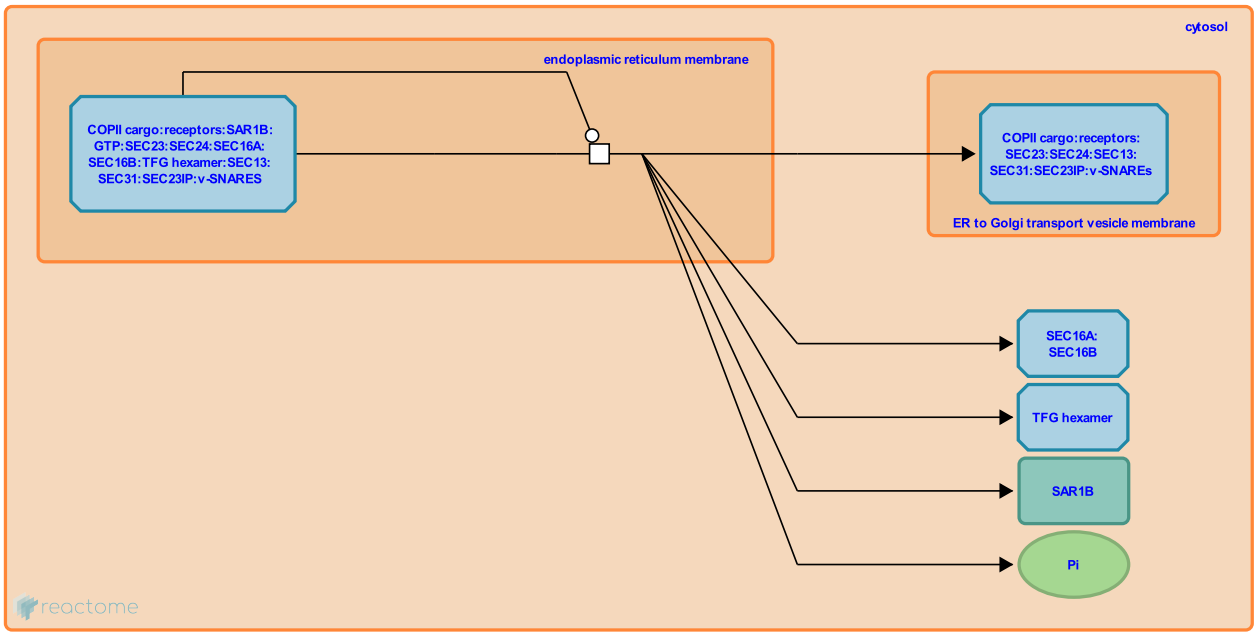
Vesicle budding

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-203973

Type: transition

Compartments: endoplasmic reticulum membrane, COPII-coated ER to Golgi transport vesicle



Once loaded the vesicles become fully sculpted, pinch off from the ER and bud into the cytosol. Budding depends on SAR1:GTPase activity and likely releases the SEC16 complex. SAR1:GTPase activity may result in partial uncoating of the emerging vesicle, allowing SEC23 to be exposed for subsequent TRAPPCII binding.

Preceded by: SEC31:SEC13 and v-SNARE recruitment

Followed by: COPII coat binds TRAPPCII and RAB1:GDP

Literature references

Paccaud, JP., Lin-Marq, N., Stephens, DJ., Pagano, A., Pepperkok, R. (2000). COPI-coated ER-to-Golgi transport complexes segregate from COPII in close proximity to ER exit sites. *J Cell Sci, 113*, 2177-85.

Glick, BS., Hammond, AT. (2000). Dynamics of transitional endoplasmic reticulum sites in vertebrate cells. *Mol Biol Cell, 11*, 3013-30.

Editions

2007-07-14	Authored	Gillespie, ME.
2010-11-18	Reviewed	Gagneux, P.
2010-11-18	Edited	Jassal, B.
2015-03-16	Revised	Rothfels, K.
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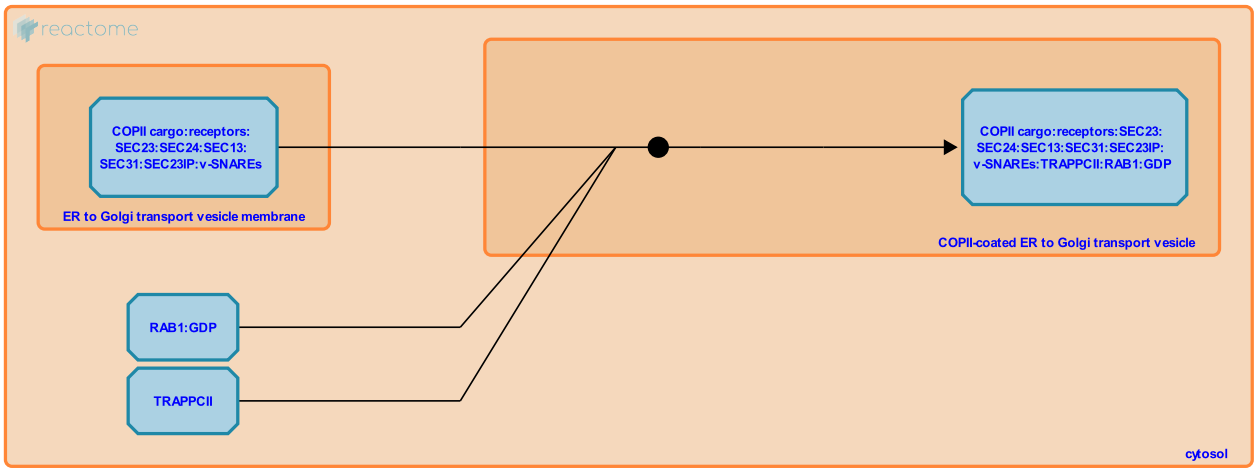
COPII coat binds TRAPPCII and RAB1:GDP ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694439

Type: binding

Compartments: COPII-coated ER to Golgi transport vesicle



TRAPPC is a multi-subunit tethering complex that facilitates ER-to-Golgi traffic (reviewed in Barrowman et al, 2010; Brunet and Sacher, 2014). TRAPPC is a guanine-nucleotide exchange factor for RAB1 and is recruited to ER-derived vesicles by virtue of an interaction between the TRAPPC component TRAPPC3 and the coat protein SEC23 (Cai et al, 2007; Lord et al, 2011).

Preceded by: Vesicle budding

Followed by: Nucleotide exchange on RAB1

Literature references

Fu, C., Ferro-Novick, S., Yu, S., Cai, H., Menon, S., Lazarova, D. et al. (2007). TRAPPI tethers COPII vesicles by binding the coat subunit Sec23. *Nature, 445*, 941-4. ↗

Sacher, M., Brunet, S. (2014). In sickness and in health: the role of TRAPP and associated proteins in disease. *Traffic, 15*, 803-18. ↗

Ghassemian, M., Ghosh, P., Bhandari, D., Ferro-Novick, S., Menon, S., Nycz, D. et al. (2011). Sequential interactions with Sec23 control the direction of vesicle traffic. *Nature, 473*, 181-6. ↗

Bhandari, D., Ferro-Novick, S., Barrowman, J., Reinisch, K. (2010). TRAPP complexes in membrane traffic: convergence through a common Rab. *Nat. Rev. Mol. Cell Biol., 11*, 759-63. ↗

Editions

2015-03-16	Authored	Rothfels, K.
2015-04-18	Edited	Rothfels, K.
2015-08-18	Reviewed	Gillespie, ME.

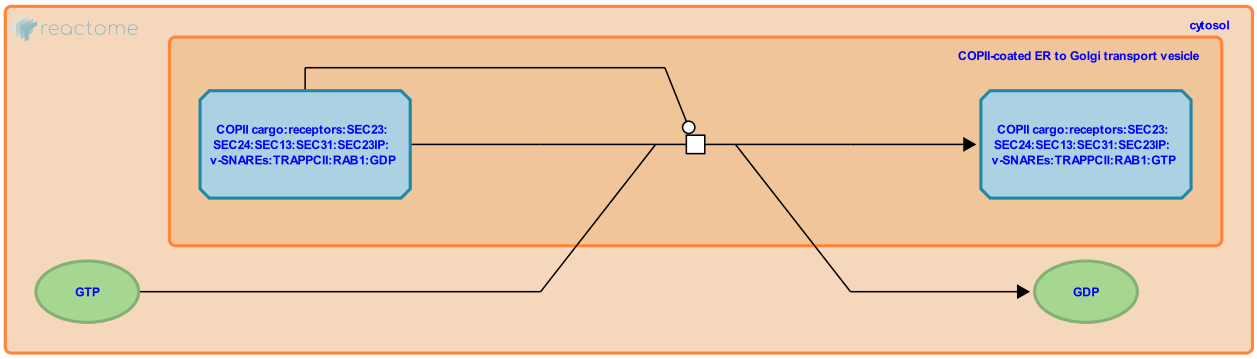
Nucleotide exchange on RAB1 ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694409

Type: transition

Compartments: COPII-coated ER to Golgi transport vesicle



The TRAPPC complex acts as a guanine-nucleotide exchange factor for RAB1, activating it (Yamasaki et al, 2009; reviewed in Brunet and Sacher, 2014).

Preceded by: COPII coat binds TRAPPCII and RAB1:GDP

Followed by: RAB1:GTP binds USO1 and GORASP1:GOLGA2

Literature references

Klumperman, J., Oorschot, V., Ferro-Novick, S., Yu, S., Yamasaki, A., Meerloo, T. et al. (2009). mTrs130 is a component of a mammalian TRAPPII complex, a Rab1 GEF that binds to COPI-coated vesicles. *Mol. Biol. Cell, 20*, 4205-15. ↗

Sacher, M., Brunet, S. (2014). In sickness and in health: the role of TRAPP and associated proteins in disease. *Traffic, 15*, 803-18. ↗

Editions

2015-03-16	Authored	Rothfels, K.
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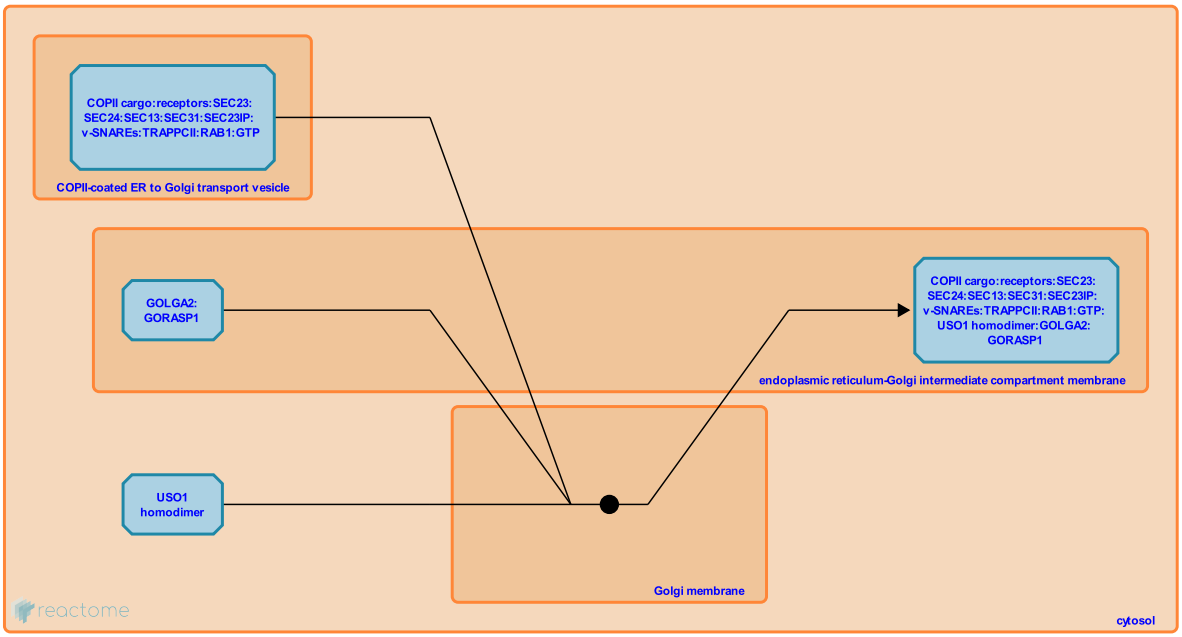
RAB1:GTP binds USO1 and GORASP1:GOLGA2

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694418

Type: binding

Compartments: Golgi membrane



Protein protein interactions involving activated RAB1:GTP help dock the ER-derived vesicle on the cis-Golgi membrane (reviewed in Lord et al, 2013) . Independently, RAB:GTP interacts with both the USO1 homodimer (a long coiled coil tethering factor also known as p115) and the GOLGA2 component of the GOLGA2:GORASP1 complex on the cis-Golgi membrane (Allan et al, 2000; Moyer et al, 2001; Weide et al, 2001). USO1 also contacts GOLGA2 directly (Nakamura et al, 1997; Seeman et al, 2000). t-SNARES such as STX5, GOSR1and GOSR2 also participate in this complex on the cis-ERGIC membrane (Brandon et al, 2006; Shorter et al, 2002; reviewed in Brandizzi and Barlowe, 2013).

Preceded by: Nucleotide exchange on RAB1

Followed by: CSNK1D phosphorylates SEC23

Literature references

Brandizzi, F., Barlowe, C. (2013). Organization of the ER-Golgi interface for membrane traffic control. *Nat. Rev. Mol. Cell Biol., 14*, 382-92.

Allan, BB., Balch, WE., Moyer, BD. (2001). Rab1 interaction with a GM130 effector complex regulates COPII vesicle cis--Golgi tethering. *Traffic, 2*, 268-76.

Warren, G., Levine, TP., Lowe, M., Rabouille, C., Nakamura, N. (1997). The vesicle docking protein p115 binds GM130, a cis-Golgi matrix protein, in a mitotically regulated manner. *Cell, 89*, 445-55.

Warren, G., Dirac-Svejstrup, AB., Beard, MB., Seemann, J., Shorter, J. (2002). Sequential tethering of Golgins and catalysis of SNAREpin assembly by the vesicle-tethering protein p115. *J. Cell Biol., 157*, 45-62.

Ferro-Novick, S., Miller, EA., Lord, C. (2013). The highly conserved COPII coat complex sorts cargo from the endoplasmic reticulum and targets it to the golgi. *Cold Spring Harb Perspect Biol, 5*.

Editions

2015-03-16	Authored	Rothfels, K.
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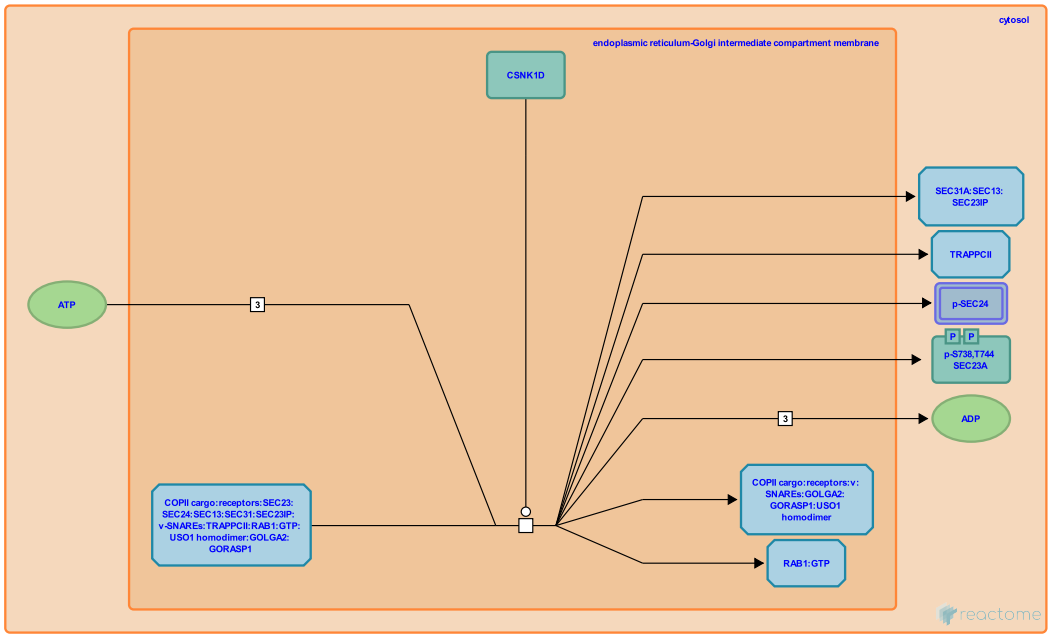
CSNK1D phosphorylates SEC23 ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694441

Type: transition

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



Coat release at the ERGIC membrane may be stimulated by CSNK1D-dependent phosphorylation of SEC23 and SEC24 (Lord et al, 2011).

Preceded by: RAB1:GTP binds USO1 and GORASP1:GOLGA2

Followed by: PP6 dephosphorylates SEC24, BET1:GOSR2:STX5 bind v-SNARES on tethered vesicle

Literature references

Ghassemian, M., Ghosh, P., Bhandari, D., Ferro-Novick, S., Menon, S., Nycz, D. et al. (2011). Sequential interactions with Sec23 control the direction of vesicle traffic. *Nature, 473*, 181-6. ↗

Editions

2015-03-16	Authored, Edited	Rothfels, K.
2015-08-18	Reviewed	Gillespie, ME.

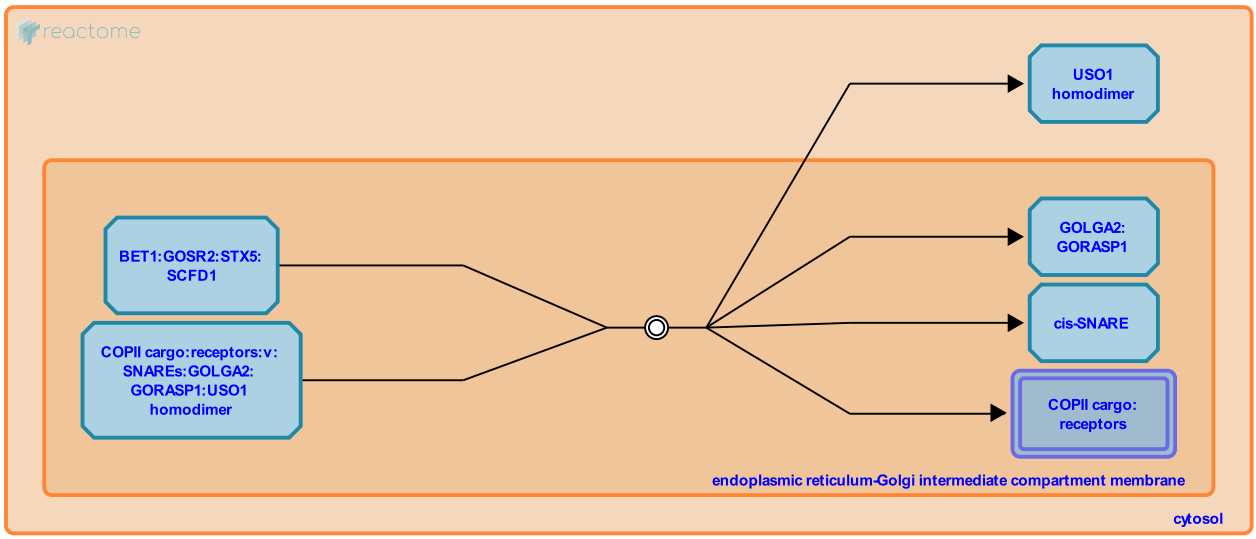
BET1:GOSR2:STX5 bind v-SNARES on tethered vesicle ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694446

Type: dissociation

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



After displacement of the COPII coat, a tetrameric SNAREpin forms between the v-SNARE and the three t-SNARE proteins (Hay et al, 1997; Hay et al, 1998; Hui et al, 1997; Bentley et al, 2006; reviewed in Hong and Lev 2014). This interaction is promoted by the tethering factor USO1 and by the STX5-interacting protein SCFD1 (Wang et al, 2014; Dascher and Balch, 1996; Cao and Barlowe 2000; Flanagan and Barlowe, 2006; reviewed in Lord et al, 2013). The zippering of the SNAREpin provides the mechanical force that drives membrane fusion, releasing cargo into the acceptor compartment (reviewed in Sudhof and Rothman, 2009).

Preceded by: CSNK1D phosphorylates SEC23

Followed by: NSF and SNAPs bind cis-SNARE complex

Literature references

Flanagan, JJ., Barlowe, C. (2006). Cysteine-disulfide cross-linking to monitor SNARE complex assembly during endoplasmic reticulum-Golgi transport. *J. Biol. Chem., 281*, 2281-8. ↗

Steegmaier, M., Klumperman, J., Oorschot, V., Kuo, CS., Scheller, RH., Hay, JC. (1998). Localization, dynamics, and protein interactions reveal distinct roles for ER and Golgi SNAREs. *J. Cell Biol., 141*, 1489-502. ↗

Hong, W., Lev, S. (2014). Tethering the assembly of SNARE complexes. *Trends Cell Biol., 24*, 35-43. ↗

Balch, WE., Dascher, C. (1996). Mammalian Sly1 regulates syntaxin 5 function in endoplasmic reticulum to Golgi transport. *J. Biol. Chem., 271*, 15866-9. ↗

Grabski, R., Wang, T., Sztul, E., Hay, JC. (2015). p115-SNARE interactions: a dynamic cycle of p115 binding monomeric SNARE motifs and releasing assembled bundles. *Traffic, 16*, 148-71. ↗

Editions

2015-03-30	Authored	Rothfels, K.
2015-04-18	Edited	Rothfels, K.
2015-08-18	Reviewed	Gillespie, ME.

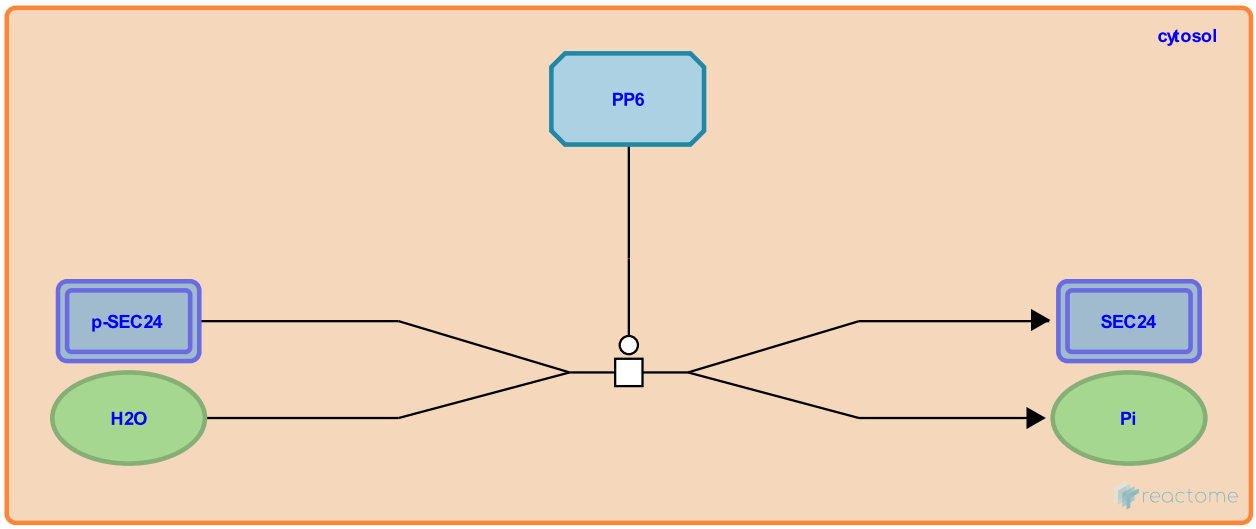
PP6 dephosphorylates SEC24 ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694421

Type: transition

Compartments: cytosol



PP6 dephosphorylates released coat proteins including SEC24, SEC31 and SEC23, allowing them to recycle for further rounds of vesicle budding from the ER (Lord et al, 2011; Bhandari et al, 2013).

Preceded by: CSNK1D phosphorylates SEC23

Literature references

Bhandari, D., Ferro-Novick, S., Zhang, J., Menon, S., Thorsen, K., Helm, JR. et al. (2013). Sit4p/PP6 regulates ER-to-Golgi traffic by controlling the dephosphorylation of COPII coat subunits. *Mol. Biol. Cell, 24*, 2727-38. ↗

Ghassemian, M., Ghosh, P., Bhandari, D., Ferro-Novick, S., Menon, S., Nycz, D. et al. (2011). Sequential interactions with Sec23 control the direction of vesicle traffic. *Nature, 473*, 181-6. ↗

Editions

2015-03-16	Authored	Rothfels, K.
2015-04-18	Edited	Rothfels, K.
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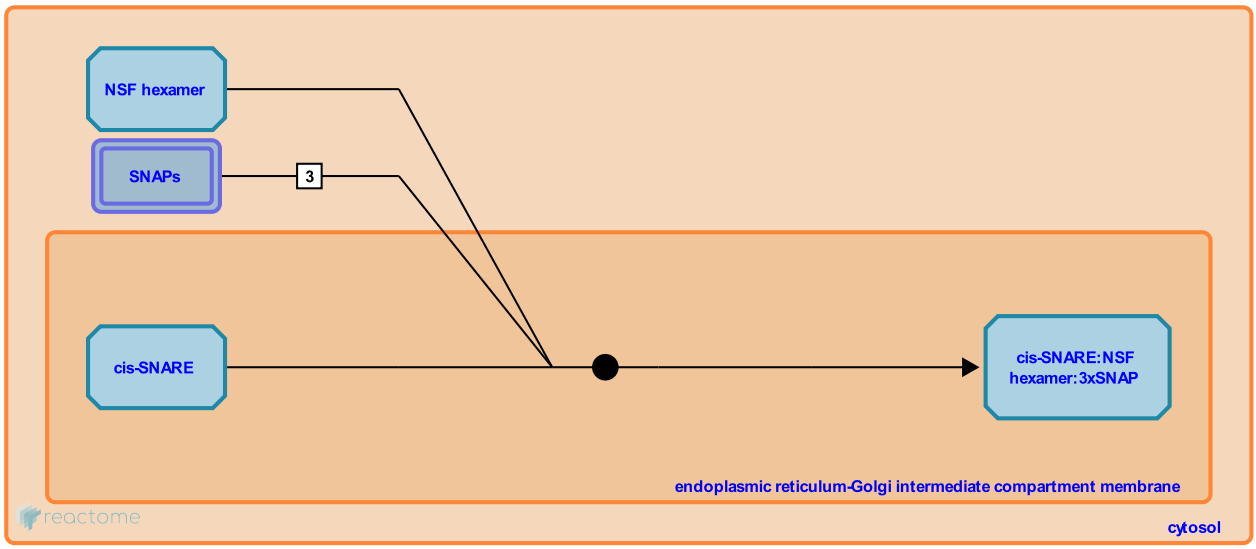
NSF and SNAPs bind cis-SNARE complex ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694423

Type: binding

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



After membrane fusion, the cis-SNARE complex is dissociated in an ATP-dependent fashion by the AAA protein NSF in conjunction with SNAP proteins (Mayer et al, 1996; Sollner et al, 1993; reviewed in Jahn and Scheller, 2006; Sudhof and Rothman, 2009).

Preceded by: BET1:GOSR2:STX5 bind v-SNARES on tethered vesicle

Followed by: NSF ATPase activity dissociates cis-SNARE

Literature references

Haas, A., Mayer, A., Wickner, W. (1996). Sec18p (NSF)-driven release of Sec17p (alpha-SNAP) can precede docking and fusion of yeast vacuoles. *Cell, 85*, 83-94. ↗

Söllner, T., Whiteheart, SW., Scheller, RH., Bennett, MK., Rothman, JE. (1993). A protein assembly-disassembly pathway in vitro that may correspond to sequential steps of synaptic vesicle docking, activation, and fusion. *Cell, 75*, 409-18. ↗

Südhof, TC., Rothman, JE. (2009). Membrane fusion: grappling with SNARE and SM proteins. *Science, 323*, 474-7. ↗

Scheller, RH., Jahn, R. (2006). SNAREs--engines for membrane fusion. *Nat. Rev. Mol. Cell Biol., 7*, 631-43. ↗

Editions

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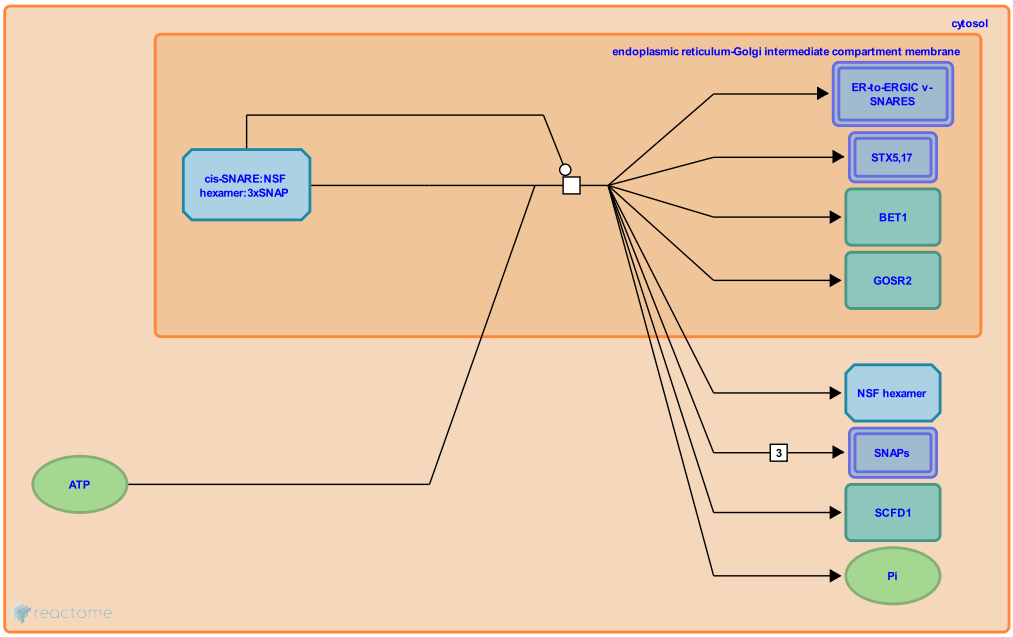
NSF ATPase activity dissociates cis-SNARE ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-5694425

Type: transition

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



NSF-dependent hydrolysis of ATP is required to disassociate the cis-SNARE complex, releasing the SNAREs for further rounds of membrane fusion (Mayer et al, 1996; Muller et al, 1999; Muller et al, 2002; Otto et al, 1997; Whiteheart et al, 2004; Yu et al, 1999; Zhao et al, 2012; Shah et al, 2015; reviewed in Sudhof and Rothman, 2009).

Preceded by: NSF and SNAPs bind cis-SNARE complex

Literature references

Haas, A., Mayer, A., Wickner, W. (1996). Sec18p (NSF)-driven release of Sec17p (alpha-SNAP) can precede docking and fusion of yeast vacuoles. *Cell, 85*, 83-94. ↗

Hanson, PI., Jahn, R., Otto, H. (1997). Assembly and disassembly of a ternary complex of synaptobrevin, syntaxin, and SNAP-25 in the membrane of synaptic vesicles. *Proc. Natl. Acad. Sci. U.S.A., 94*, 6197-201. ↗

Enos, MD., Colbert, KN., Herschlag, D., Shah, N., Weis, WI. (2015). Three ?SNAP and 10 ATP molecules are used in SNARE complex disassembly by N-ethylmaleimide-sensitive factor (NSF). *J. Biol. Chem., 290*, 2175-88. ↗

Yu, RC., Jahn, R., Brunger, AT. (1999). NSF N-terminal domain crystal structure: models of NSF function. *Mol. Cell, 4*, 97-107. ↗

Matveeva, EA., Whiteheart, SW. (2004). Multiple binding proteins suggest diverse functions for the N-ethylmaleimide sensitive factor. *J. Struct. Biol., 146*, 32-43. ↗

Editions

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2015-08-18	Reviewed	Gillespie, ME.

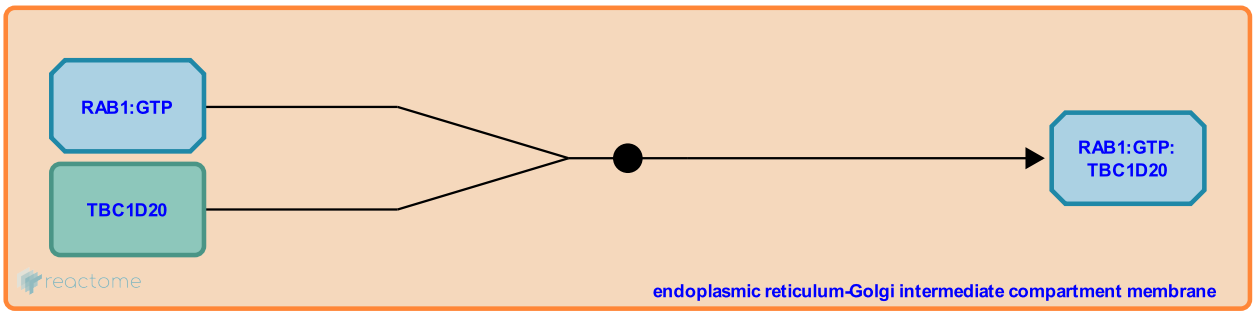
TBC1D20 binds RAB1:GTP ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-6814831

Type: binding

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



TBC1D20 was identified as a GTPase activating protein for RAB1 that stimulates basal GTP hydrolysis by more than 5 orders of magnitude (Haas et al, 2007; Fuchs et al, 2007).

Followed by: TBC1D20 stimulates GTPase activity of RAB1, resulting in hydrolysis of GTP

Literature references

Spooner, RA., Barr, FA., Lord, JM., Haas, AK., Yoshimura, S., Fuchs, E. (2007). Specific Rab GTPase-activating proteins define the Shiga toxin and epidermal growth factor uptake pathways. *J. Cell Biol., 177*, 1133-43. ↗

Barr, FA., Haas, AK., Stephens, DJ., Yoshimura, S., Fuchs, E., Preisinger, C. (2007). Analysis of GTPase-activating proteins: Rab1 and Rab43 are key Rabs required to maintain a functional Golgi complex in human cells. *J. Cell. Sci., 120*, 2997-3010. ↗

Editions

2015-11-25	Reviewed	Gillespie, ME.
2015-11-28	Authored, Edited	Rothfels, K.

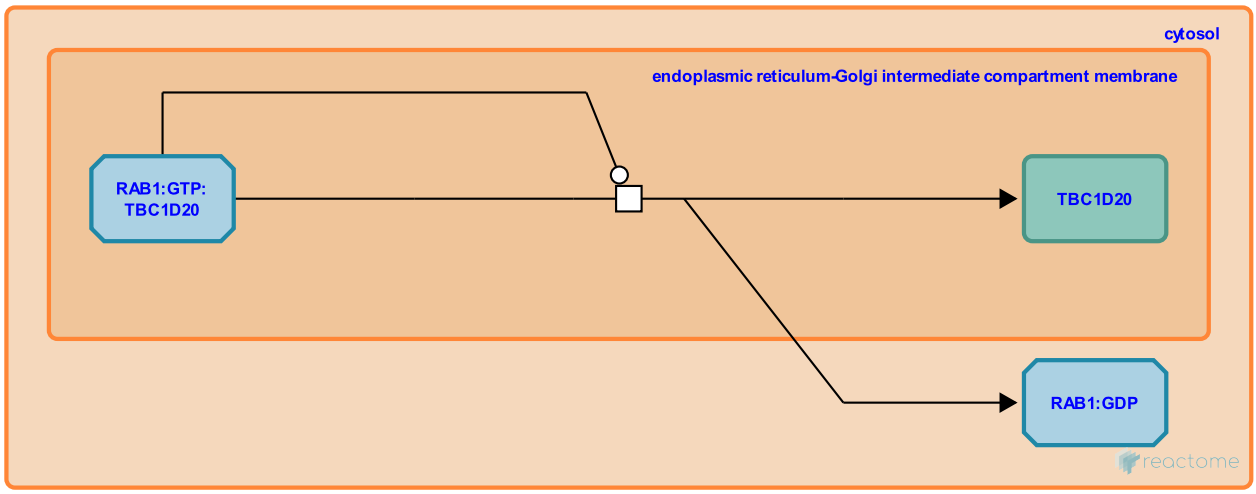
TBC1D20 stimulates GTPase activity of RAB1, resulting in hydrolysis of GTP ↗

Location: COPII-mediated vesicle transport

Stable identifier: R-HSA-6814833

Type: transition

Compartments: endoplasmic reticulum-Golgi intermediate compartment membrane



TBC1D20 was identified as a RAB1-specific GTPase activating protein (GAP) that stimulates RAB1-mediated GTP hydrolysis and plays roles in ER-to-Golgi trafficking. TBC1D20 is the only GAP that has been identified to block delivery of secretory cargo from the ER to the cell surface (Haas et al, 2007).

Preceded by: TBC1D20 binds RAB1:GTP

Literature references

Barr, FA., Haas, AK., Stephens, DJ., Yoshimura, S., Fuchs, E., Preisinger, C. (2007). Analysis of GTPase-activating proteins: Rab1 and Rab43 are key Rabs required to maintain a functional Golgi complex in human cells. *J. Cell. Sci., 120*, 2997-3010. ↗

Editions

2015-11-25	Reviewed	Gillespie, ME.
2015-11-28	Authored, Edited	Rothfels, K.

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