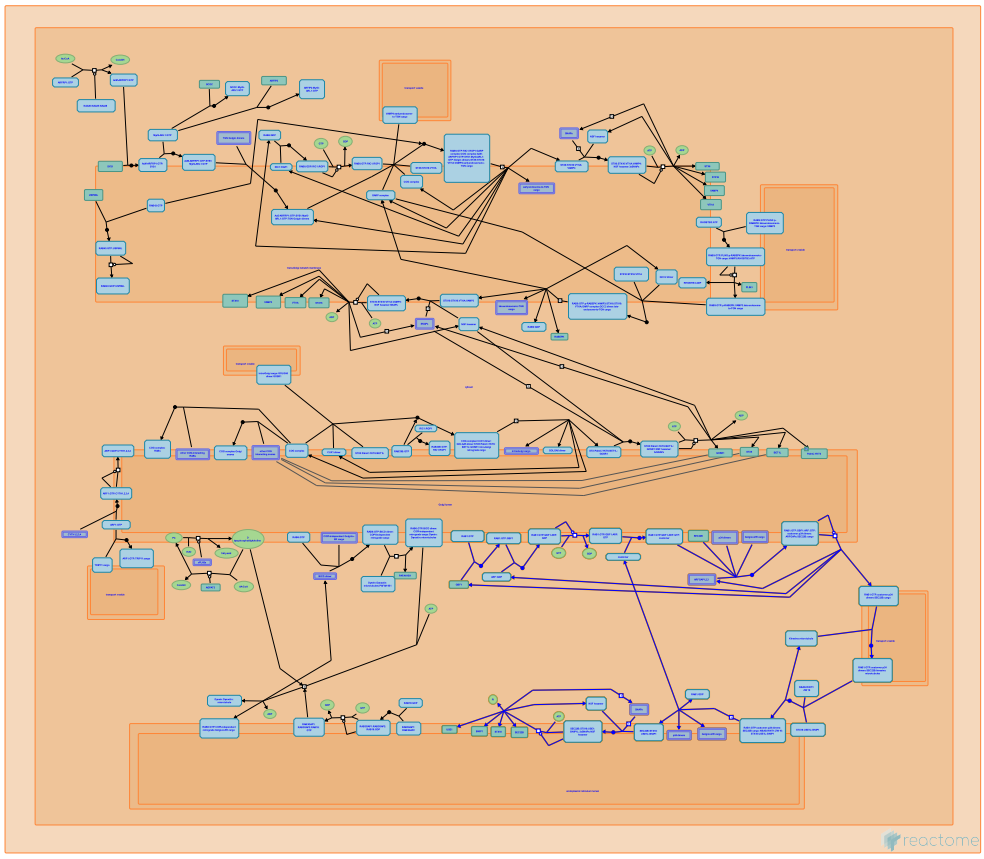
COPI-dependent Golgi-to-ER retrograde traffic



Gillespie, ME., Rothfels, K.

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27/04/2024

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

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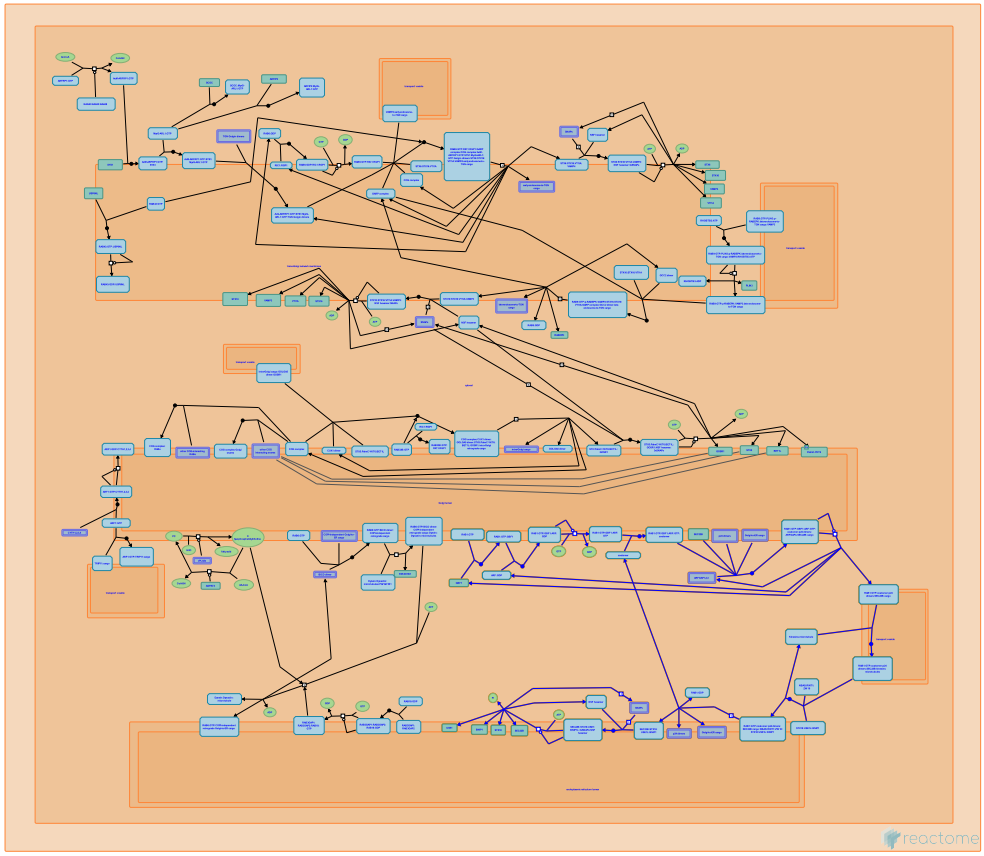
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

COPI-dependent Golgi-to-ER retrograde traffic ↗

Stable identifier: R-HSA-6811434



Retrograde traffic from the cis-Golgi to the ERGIC or the ER is mediated in part by microtubule-directed COPI-coated vesicles (Letourneur et al, 1994; Shima et al, 1999; Spang et al, 1998; reviewed in Lord et al, 2013; Spang et al, 2013). These assemble at the cis side of the Golgi in a GBF-dependent fashion and are tethered at the ER by the ER-specific SNAREs and by the conserved NRZ multisubunit tethering complex, known as DSL in yeast (reviewed in Tagaya et al, 2014; Hong and Lev, 2014). Typical cargo of these retrograde vesicles includes 'escaped' ER chaperone proteins, which are recycled back to the ER for reuse by virtue of their interaction with the Golgi localized KDEL receptors (reviewed in Capitani and Sallese, 2009; Cancino et al, 2013).

Literature references

Luini, A., Cancino, J., Jung, JE. (2013). Regulation of Golgi signaling and trafficking by the KDEL receptor. *Histochem. Cell Biol., 140*, 395-405. ↗

Scales, SJ., Kreis, TE., Pepperkok, R., Shima, DT. (1999). Segregation of COPI-rich and anterograde-cargo-rich domains in endoplasmic-reticulum-to-Golgi transport complexes. *Curr. Biol., 9*, 821-4. ↗

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

Tagaya, M., Kimura, H., Inoue, H., Arasaki, K. (2014). Moonlighting functions of the NRZ (mammalian Dsl1) complex . *Front Cell Dev Biol, 2*, 25. ↗

Spang, A. (2013). Retrograde traffic from the Golgi to the endoplasmic reticulum. *Cold Spring Harb Perspect Biol, 5*. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

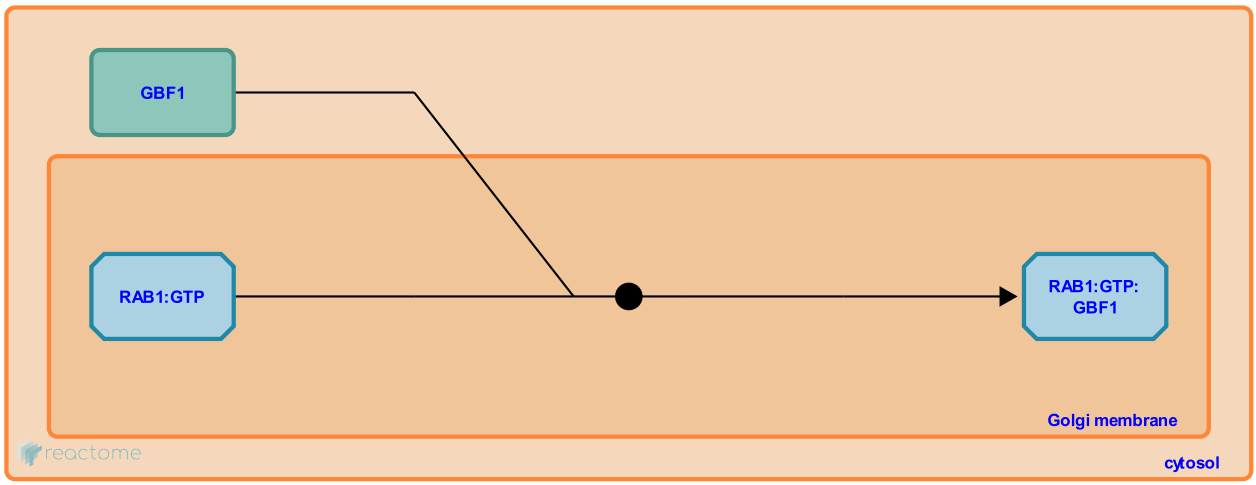
RAB:GTP recruits GBF1 to the Golgi membrane ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811415

Type: binding

Compartments: Golgi membrane



In its GTP-bound active state, RAB1 recruits the ARF GEF GBF1 to the Golgi (Monetta et al, 2007). GBF is the only ARF activator required for the formation of COPI coats, and therefore it has roles in the anterograde ERGIC-to-cis-Golgi as well as in COPI-mediated retrograd transport within the Golgi and back to the ERGIC and ER (Kawamoto et al, 2002; Szul et al, 2005; Zhao et al, 2006; Szul et al, 2007; reviewed in Szul and Sztul, 2011). GBR1 activates ARF1, 2, 3 and 5 which play overlapping roles in the secretory pathway (Volpicelli-Daley et al, 2005; Chun et al, 2008; reviewed in D'Souza-Schorey and Chavrier, 2006).

Followed by: GBF1 recruits ARF:GDP to the Golgi

Literature references

Yoshida, Y., Tamaki, H., Kawamoto, K., Yamashina, S., Nakayama, K., Torii, S. et al. (2002). GBF1, a guanine nucleotide exchange factor for ADP-ribosylation factors, is localized to the cis-Golgi and involved in membrane association of the COPI coat. *Traffic, 3*, 483-95. ↗

Presley, JF., Melançon, P., Chun, J., Dejgaard, SY., Shapovalova, Z. (2008). Characterization of class I and II ADP-ribosylation factors (Arfs) in live cells: GDP-bound class II Arfs associate with the ER-Golgi intermediate compartment independently of GBF1. *Mol. Biol. Cell, 19*, 3488-500. ↗

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

Slavin, I., Monetta, P., Romero, N., Alvarez, C. (2007). Rab1b interacts with GBF1 and modulates both ARF1 dynamics and COPI association. *Mol. Biol. Cell, 18*, 2400-10. ↗

Volpicelli-Daley, LA., Zhang, CJ., Li, Y., Kahn, RA. (2005). Isoform-selective effects of the depletion of ADP-ribosylation factors 1-5 on membrane traffic. *Mol. Biol. Cell, 16*, 4495-508. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

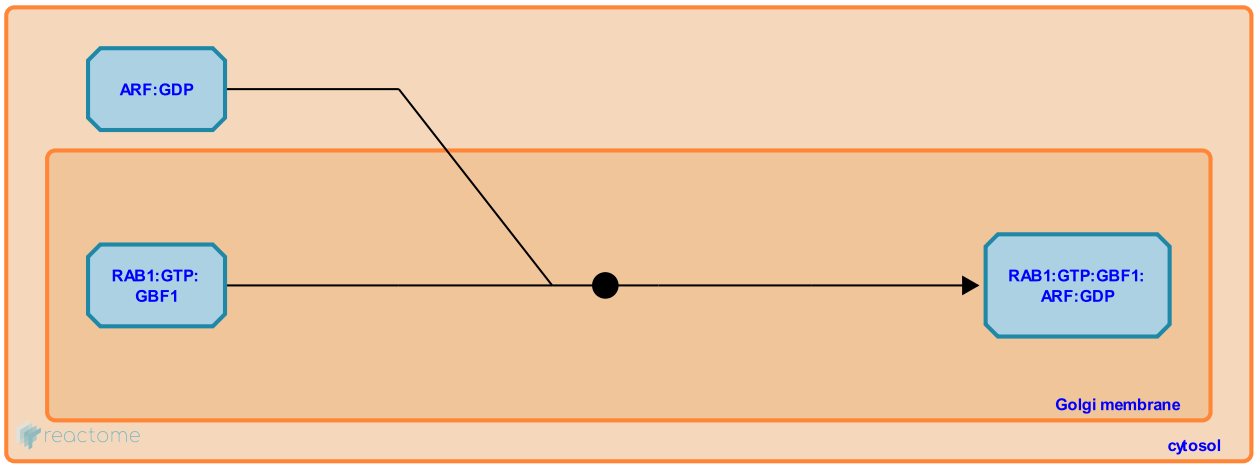
GBF1 recruits ARF:GDP to the Golgi ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811411

Type: binding

Compartments: Golgi membrane



GBF1 recruits inactive ARF:GDP complexes to the Golgi (Monetta et al, 2007). There are 5 known ARFs in the human cell. Class I members ARF1 and ARF 3 are expressed at high levels and broadly distributed through the secretory system, while Class II members ARF4 and 5 are expressed at lower levels. ARF6, the single Class III ARF, appears to function more specifically in endocytosis and actin dynamics (Chun et al, 2008; reviewed in D'Souza-Schorey and Chavrier, 2006; Szul and Sztul, 2011). GBF1 has been shown to activate ARF1, 4, and 5, but not ARF3, while single and pairwise knockdown of ARF1, 3, 4 and 5 suggests that no single ARF is responsible for any given step in the secretory pathway (Manolea et al, 2010; Volpicelli-Daley et al, 2005).

Preceded by: RAB:GTP recruits GBF1 to the Golgi membrane

Followed by: GBF1 stimulates nucleotide exchange on ARF

Literature references

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

Presley, JF., Melançon, P., Chun, J., Dejgaard, SY., Shapovalova, Z. (2008). Characterization of class I and II ADP-ribosylation factors (Arfs) in live cells: GDP-bound class II Arfs associate with the ER-Golgi intermediate compartment independently of GBF1. *Mol. Biol. Cell, 19*, 3488-500. ↗

Manolea, F., Chen, DW., Melançon, P., Summerfeldt, N., Clarke, I., Dacks, JB. et al. (2010). Arf3 is activated uniquely at the trans-Golgi network by brefeldin A-inhibited guanine nucleotide exchange factors. *Mol. Biol. Cell, 21*, 1836-49. ↗

Slavin, I., Monetta, P., Romero, N., Alvarez, C. (2007). Rab1b interacts with GBF1 and modulates both ARF1 dynamics and COPI association. *Mol. Biol. Cell, 18*, 2400-10. ↗

Volpicelli-Daley, LA., Zhang, CJ., Li, Y., Kahn, RA. (2005). Isoform-selective effects of the depletion of ADP-ribosylation factors 1-5 on membrane traffic. *Mol. Biol. Cell, 16*, 4495-508. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

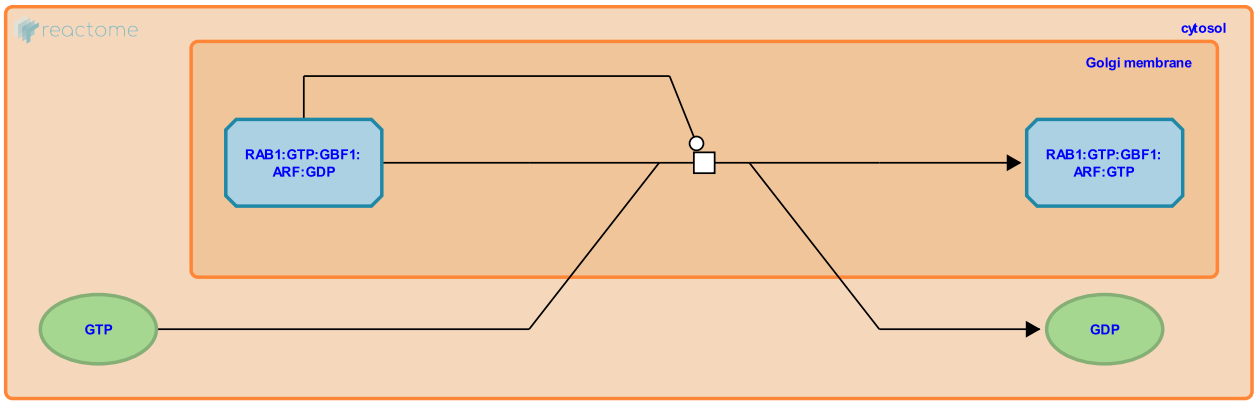
GBF1 stimulates nucleotide exchange on ARF ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811414

Type: transition

Compartments: Golgi membrane



GBF1 facilitates the exchange of GDP for GTP, activating ARF (Niu et al, 2005; Szul et al, 2005; Szul et al, 2007; Kawamoto et al, 2002; reviewed in Szul and Sztul, 2011).

Preceded by: GBF1 recruits ARF:GDP to the Golgi

Followed by: Active ARF recruits coatomer to the Golgi

Literature references

Yoshida, Y., Tamaki, H., Kawamoto, K., Yamashina, S., Nakayama, K., Torii, S. et al. (2002). GBF1, a guanine nucleotide exchange factor for ADP-ribosylation factors, is localized to the cis-Golgi and involved in membrane association of the COPI coat. *Traffic, 3*, 483-95. ↗

Jackson, CL., Lippincott-Schwartz, J., Pfeifer, AC., Niu, TK. (2005). Dynamics of GBF1, a Brefeldin A-sensitive Arf1 exchange factor at the Golgi. *Mol. Biol. Cell, 16*, 1213-22. ↗

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

Shestopal, S., Sztul, E., Szul, T., Alvarez, C., Brandon, E., Garcia-Mata, R. (2005). Dissection of membrane dynamics of the ARF-guanine nucleotide exchange factor GBF1. *Traffic, 6*, 374-85. ↗

Grabski, R., Shestopal, S., Morohashi, Y., Lyons, S., Sztul, E., Szul, T. et al. (2007). Dissecting the role of the ARF guanine nucleotide exchange factor GBF1 in Golgi biogenesis and protein trafficking. *J. Cell. Sci., 120*, 3929-40. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

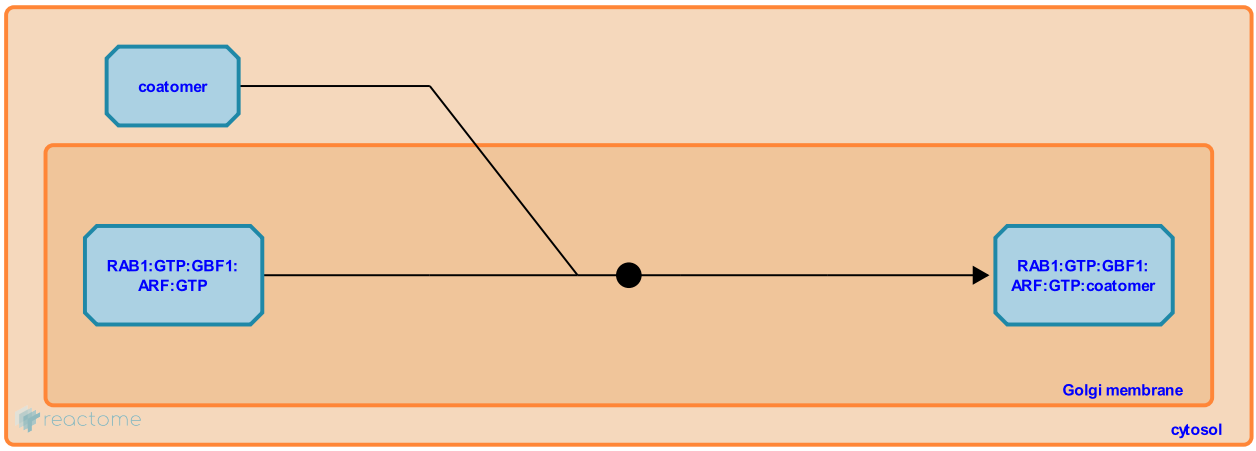
Active ARF recruits coatomer to the Golgi ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811412

Type: binding

Compartments: Golgi membrane



Activation of ARF is tightly correlated to recruitment of the COPI coat (Donaldson et al, 1991; Serafini et al, 1991; Donaldson et el, 1992; Palmer et al, 1993; reveiwed in Szul and Sztul, 2011). Studies in yeast and in mammalian cells support a direct interaction between the GTPase and components of the COPI coat (Zhao et al, 1997; Zhao et al, 1999; Zhao et al, 2006; Eugster et al, 2000; Sun et al, 2007; Yu et al, 2012; Harter and Wieland, 1998; Bethune et al, 2006; reviewed in Popoff et al, 2011). The COPI coat consists of 7 subunits arranged in 2 subcomplexes. The inner coat is made up of a tetrameric complex consisting of the beta, gamma, zeta and delta COPI subunits, while the outer coat is a trimer consisting of the alpha, beta prime and epsilon subunits (Eugster et al, 2000; Waters et al, 1991). Both of the zeta and gamma subunits have 2 isoforms with different subcellular locations, suggesting that different COPI coats may mediate different steps of the secretory pathway (Moelleken et al, 2007). Unlike the case for COPII or clathrin coats, all components of the COPI coat are recruited simultaneously as a preformed heptameric complex (Hara-Kuge et al, 1994).

Preceded by: GBF1 stimulates nucleotide exchange on ARF

Followed by: ARFGAP, cargo, vSNARES and p24 proteins bind COPI vesicles at Golgi

Literature references

Russell, RB., Moelleken, J., Meissner, I., Söllner, T., Betts, MJ., Brugger, B. et al. (2007). Differential localization of coatomer complex isoforms within the Golgi apparatus. *Proc. Natl. Acad. Sci. U.S.A., 104*, 4425-30. ↗

Zhao, L., Wieland, FT., Helms, JB., Brunner, J. (1999). GTP-dependent binding of ADP-ribosylation factor to coatomer in close proximity to the binding site for dilysine retrieval motifs and p23. *J. Biol. Chem., 274*, 14198-203 . ↗

Serafini, T., Brunner, M., Orci, L., Amherdt, M., Rothman, JE., Kahn, RA. (1991). ADP-ribosylation factor is a subunit of the coat of Golgi-derived COP-coated vesicles: a novel role for a GTP-binding protein. *Cell, 67*, 239-53. ↗

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Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

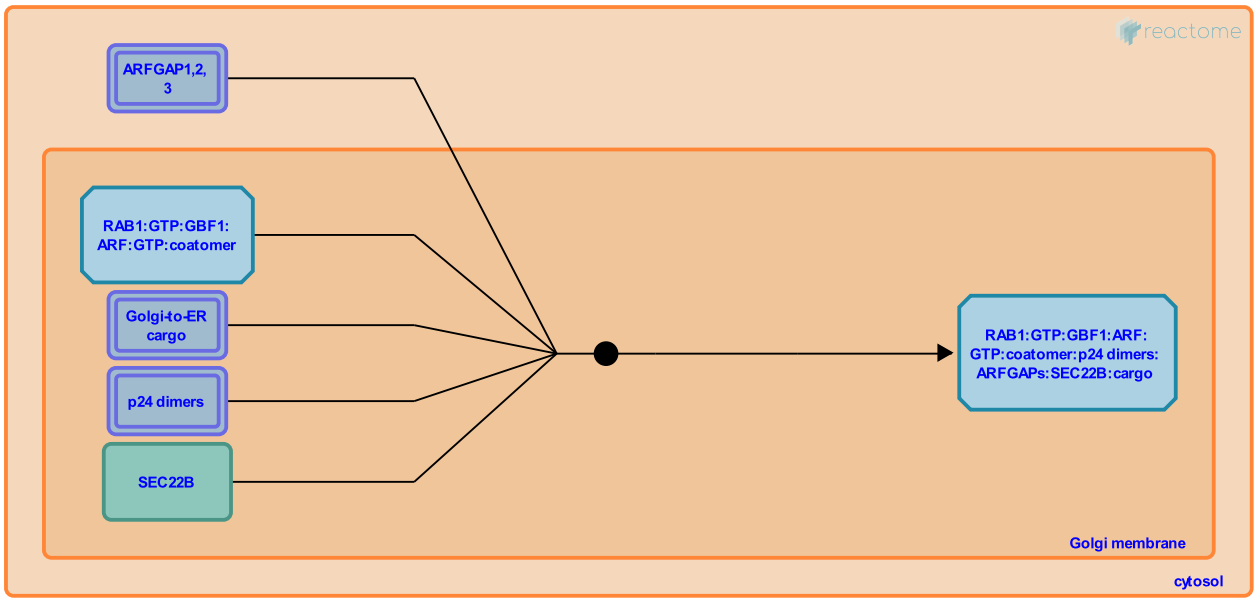
ARFGAP, cargo, vSNARES and p24 proteins bind COPI vesicles at Golgi ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811417

Type: binding

Compartments: Golgi membrane



Binding and polymerization of coatomer is coordinated with the incorporation of cargo proteins and Golgi-targeting snares, as well as with recruitment of ARFGAP proteins (Letourneur et al, 1994; Nagahama et al,1996; Bremser et al, 1999).
Typical cargo for COPI-mediated retrograde traffic includes the KDEL receptors, which bind and recycle ER-resident proteins, as well as other cycling proteins such as SURF4 that interacts with p24 proteins and contributes to Golgi maintenance (Cosson and Letourner, 1994; Ben-Tekaya et al, 2005; Majoul et al, 2001; Orci et al, 1997, Bremser et al, 1999; Presley et al, 1997; Mitrovic et al, 2008; reviewed in Beck et al, 2009).
Other protein components of the COPI vesicle include the p24 family of proteins, which serve diverse roles in the early secretory pathway (reviewed in Schuiki and Volchuk, 2012). Oligomeric p24 proteins interact with ADP-bound ARF and components of the COPI coat, contributing to coatomer recruitment and oligomerization (Gommel et al, 2001; Majoul et al, 2001; Bethune et al, 2006; Harter and Wieland, 1998; Langer et al, 2008; Reinhard et al, 1999). p24 proteins also act as cargo receptors for various proteins destined for packaging in COPI vesicles; these include GPI-anchored transmembrane proteins, WNT ligands and some G-protein coupled receptors, among others (Takida et al, 2008; Bonnon et al, 2010; Luo et al, 2011; Beuchling et al, 2011; Wang and Kazanietz, 2002; reviewed in Schuiki and Volchuk, 2012). p24 proteins also contribute to COPI coat disassembly by restricting ARF GTPase activity until cargo has been loaded (Goldberg, 2000; Lanoix et al, 2001).
ARFGAPs are recruited to the budding vesicle through direct interaction with active ARF, the cytoplasmic tails of cargo proteins and with components of the COPI coat (Goldberg, 2000; Majoul et al, 2001; Aoe et al, 1997; Kliouchnikov et al, 2009; Luo et al, 2009). Stimulation of ARF GTPase activity is coordinated with cargo recruitment to ensure that only cargo-loaded vesicles are produced (Goldberg, 2000; Luo et al, 2009).
Mammalian cells have 3 ARFGAPs that appear to be involved in COPI-mediated traffic, ARFGAP1,2 and 3 (Frigerio et al, 2007; Liu et al, 2001; Kahn et al, 2008). ARFGAP1 has a ALPS domain that recognizes membrane curvature and that is required for the GTPase stimulating activity of the protein, suggesting a mechanism for coordinating ARF1-mediated GTP hydrolysis with vesicle formation (Bigay et al, 2003; Mesmin et al, 2007). ARFGAP 2 and 3 do not contain this motif, and their activity is dependent upon interaction with coatomer (Weimar et al 2008; Kliouchnikov et al, 2009; Luo et al, 2009).

Preceded by: Active ARF recruits coatomer to the Golgi

Followed by: ARFGAPs stimulate ARF GTPase activity at the Golgi membrane

Literature references

Riezman, H., Hennecke, S., Emr, SD., Gaynor, EC., Letourneur, F., Cosson, P. et al. (1994). Coatomer is essential for retrieval of dilysine-tagged proteins to the endoplasmic reticulum. *Cell, 79*, 1199-207. ↗

Letourneur, F., Cosson, P. (1994). Coatomer interaction with di-lysine endoplasmic reticulum retention motifs. *Science, 263*, 1629-31. ↗

Weimer, C., Moelleken, J., Wieland, F., Brugger, B., Eckert, P., Beck, R. et al. (2008). Differential roles of ArfGAP1, ArfGAP2, and ArfGAP3 in COPI trafficking. *J. Cell Biol., 183*, 725-35. ↗

Wang, H., Kazanietz, MG. (2002). Chimaerins, novel non-protein kinase C phorbol ester receptors, associate with Tmp21-I (p23): evidence for a novel anchoring mechanism involving the chimaerin C1 domain. *J. Biol. Chem., 277* , 4541-50. ↗

Zapp, ML., Theibert, AB., Inoue, H., Satake, M., Premont, RT., Logsdon, JM. et al. (2008). Consensus nomenclature for the human ArfGAP domain-containing proteins. *J. Cell Biol., 182*, 1039-44. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

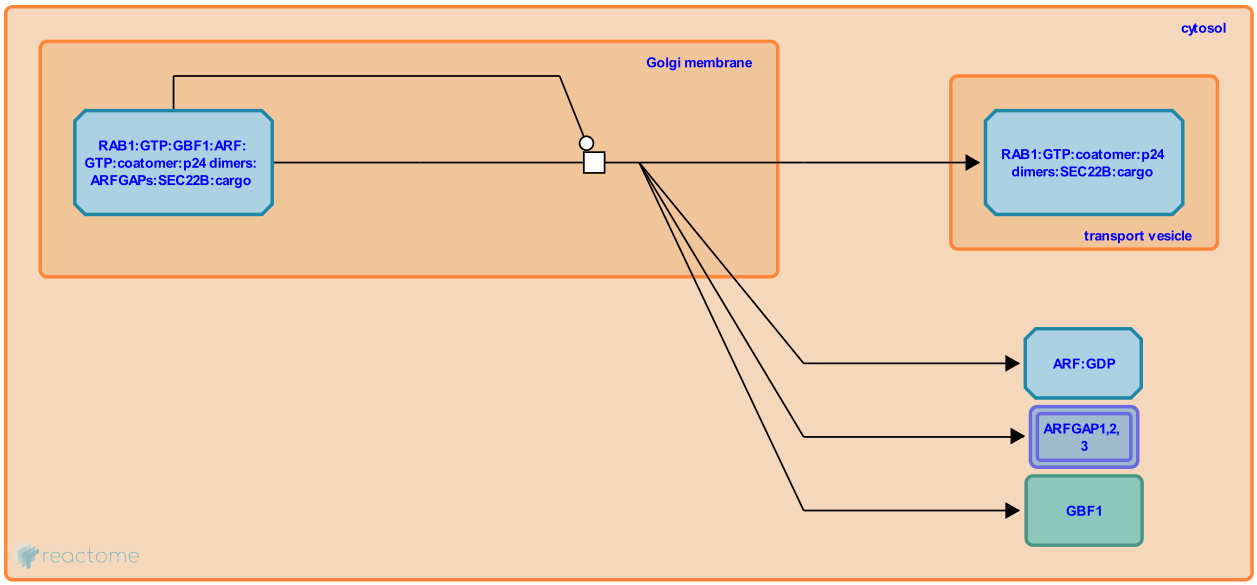
ARFGAPs stimulate ARF GTPase activity at the Golgi membrane ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811418

Type: transition

Compartments: Golgi membrane



The ARFGAP proteins stimulates ARF GTPase activity, promoting the release of the nascent COPI vesicle from the membrane and release of ARF:ADP (Tanigawa et al, 1993; reviewed in Beck et al, 2009; East and Kahn, 2011). Although this reaction shows their dissociation, it is not clear whether ARFGAPs persist on the COPI vesicle after GTP hydrolysis, nor is it known when GBF is released from the nascent COPI vesicle.

Preceded by: ARFGAP, cargo, vSNARES and p24 proteins bind COPI vesicles at Golgi

Followed by: Retrograde COPI vesicles bind kinesin and microtubules

Literature references

Rawet, M., Ravet, M., Wieland, FT., Beck, R., Cassel, D. (2009). The COPI system: molecular mechanisms and function. *FEBS Lett., 583*, 2701-9. ↗

Tanigawa, G., Amherdt, M., Rothman, JE., Orci, L., Ravazzola, M., Helms, JB. (1993). Hydrolysis of bound GTP by ARF protein triggers uncoating of Golgi-derived COP-coated vesicles. *J. Cell Biol., 123*, 1365-71. ↗

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Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

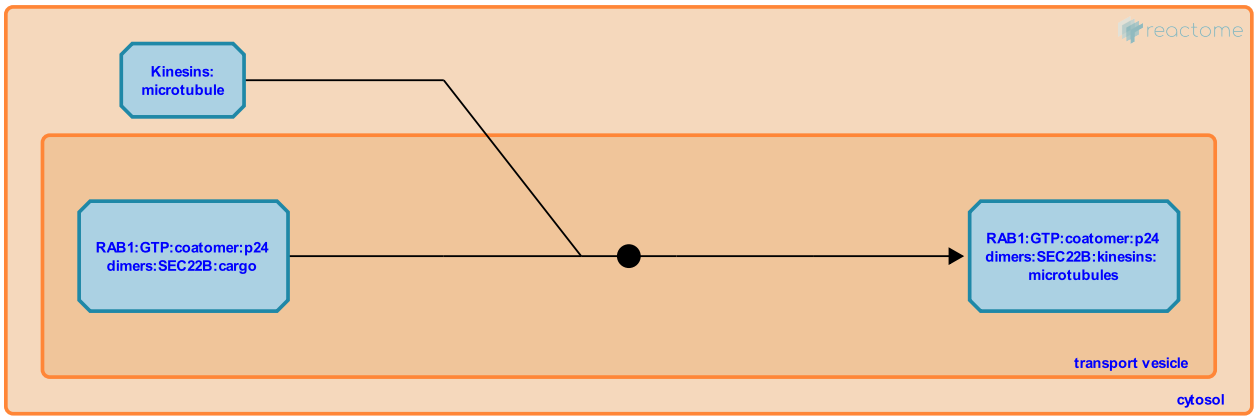
Retrograde COPI vesicles bind kinesin and microtubules ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811426

Type: binding

Compartments: transport vesicle



COPI-mediated retrograde traffic is dependent on microtubules and the plus-end motor kinsesin. Although it is not shown in this reaction, vesicle translocation along the microtubules by kinesin depends on ATP hydrolysis (Lippincott-Scwartz et al, 1995; Stauber et al, 2006; Tomas et al, 2010)

Preceded by: ARFGAPs stimulate ARF GTPase activity at the Golgi membrane

Followed by: Retrograde vesicle is tethered at the ER by the NRZ complex and t-SNAREs

Literature references

Bloom, GS., Lippincott-Schwartz, J., Cole, NB., Marotta, A., Conrad, PA. (1995). Kinesin is the motor for microtubule-mediated Golgi-to-ER membrane traffic. *J. Cell Biol., 128*, 293-306. ↗

Simpson, JC., Pepperkok, R., Stauber, T., Vernos, I. (2006). A role for kinesin-2 in COPI-dependent recycling between the ER and the Golgi complex. *Curr. Biol., 16*, 2245-51. ↗

Tomás, M., Martínez-Alonso, E., Ballesta, J., Martínez-Menárguez, JA. (2010). Regulation of ER-Golgi intermediate compartment tubulation and mobility by COPI coats, motor proteins and microtubules. *Traffic, 11*, 616-25. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

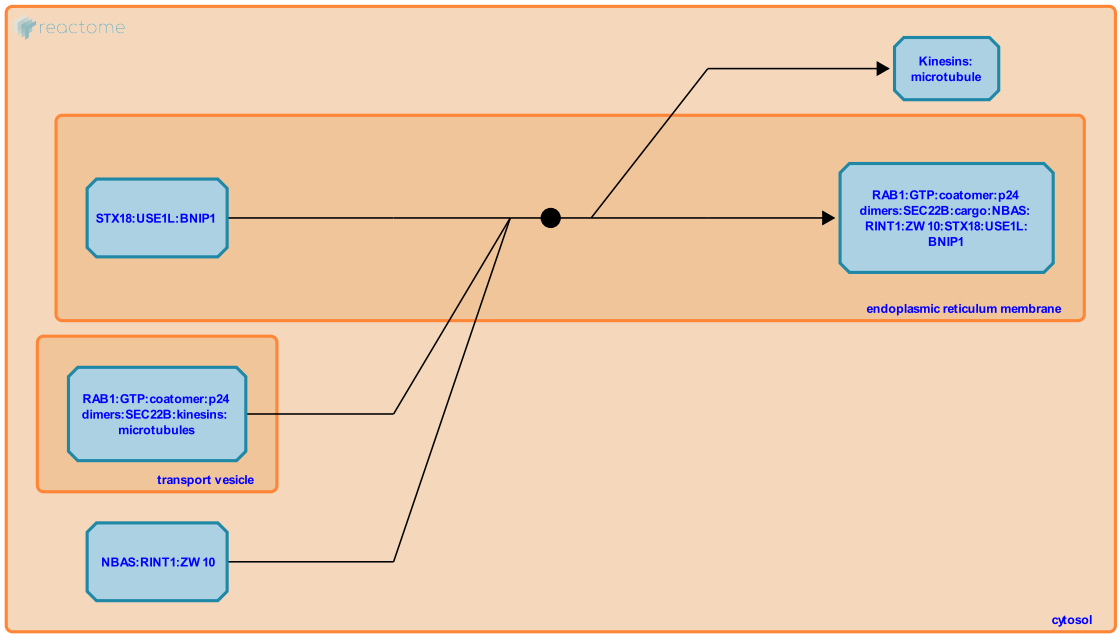
Retrograde vesicle is tethered at the ER by the NRZ complex and t-SNAREs ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811423

Type: binding

Compartments: endoplasmic reticulum membrane



Retrograde COPI vesicles destined for fusion with the ER are tethered to the ER membrane by interactions with the ER t-SNARE proteins and with the CATCHR ('complexes associated with tethering containing helical rods') complex NRZ (reviewed in Szul and Sztul, 2011; Tagaya et al, 2014). The trimeric NRZ complex, known as Dsl in yeast, is composed of NBAS, RINT1 and ZW10 and is recruited to the ER through association with the ER t-SNAREs USE1L, STX18 and BNIP1 (Hirose et al, 2004; Aoki et al, 2004; Nakajima et al, 2004; Arasaki et al, 2006; Ren et al, 2009; Civril et al, 2010; reviewed in Tagaya et al, 2014). Evidence in yeast suggests components of the Dsl complex also interact with the coatomer coat; these interactions contribute to vesicle fusion both by aiding in the recruitment of the vesicle to the ER membrane and also to the depolymerization of coatomer and thus vesicle uncoating Interactions (Andag et al, 2001; Andag et al, 2003; Reilly et al, 2001; Hsia and Hoelz, 2010; Meiringer et al, 2011; Zink et al, 2009). Note that although this pathway shows COPI vesicles from the Golgi being 'received' exclusively at the ER, vesicles are also tethered and fused at the ERGIC. The SNAREs and tethering complexes that mediate this fusion are not identified.

Preceded by: Retrograde COPI vesicles bind kinesin and microtubules

Followed by: COPI vesicle uncoating at the ER

Literature references

Tagaya, M., Hatsuzawa, K., Takio, K., Tohyama, M., Tani, K., Hirose, H. et al. (2004). Implication of ZW10 in membrane trafficking between the endoplasmic reticulum and Golgi. *EMBO J., 23*, 1267-78. ↗

Tagaya, M., Taniguchi, M., Nakajima, K., Tani, K., Hirose, H., Arasaki, K. et al. (2004). Involvement of BNIP1 in apoptosis and endoplasmic reticulum membrane fusion. *EMBO J., 23*, 3216-26. ↗

Schmitt, HD., Perz, A., Meiringer, CT., Auffarth, K., Ungermann, C., Barlowe, C. et al. (2011). The Dsl1 protein tethering complex is a resident endoplasmic reticulum complex, which interacts with five soluble NSF (N-ethylmaleimide-sensitive factor) attachment protein receptors (SNAREs): implications for fusion and fusion regulation. *J. Biol. Chem., 286*, 25039-46. ↗

Tagaya, M., Kimura, H., Inoue, H., Arasaki, K. (2014). Moonlighting functions of the NRZ (mammalian Dsl1) complex . *Front Cell Dev Biol, 2*, 25. ↗

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Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

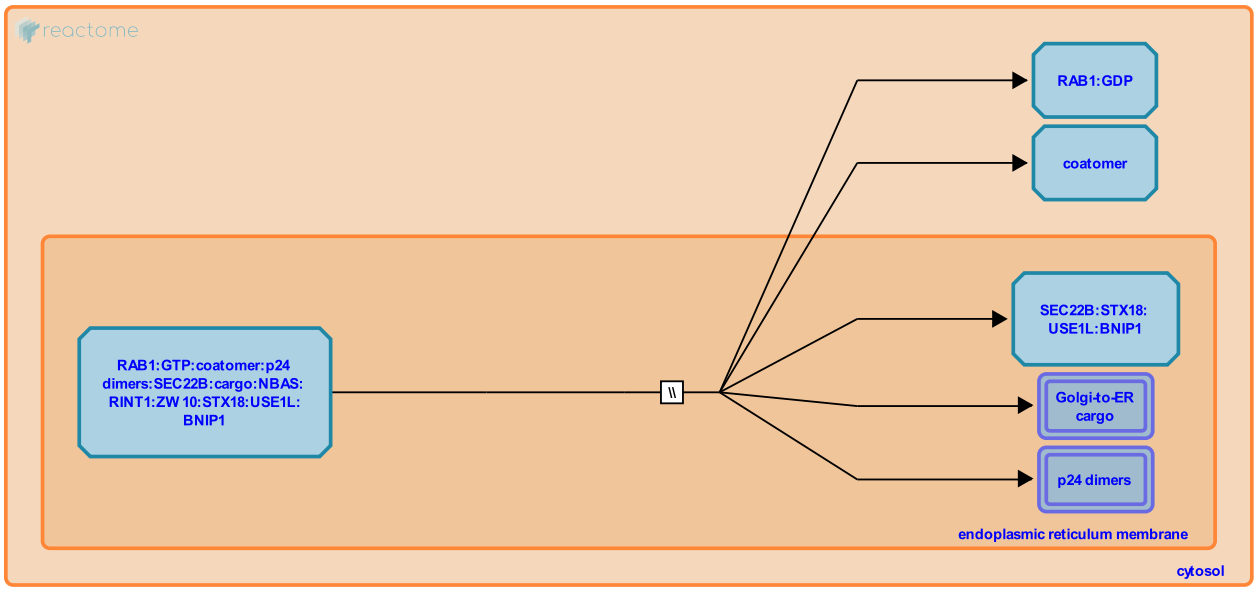
COPI vesicle uncoating at the ER ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811427

Type: omitted

Compartments: endoplasmic reticulum membrane



While the details of COPI vesicle uncoating are not fully established, interactions between components of the NRZ tethering complex and coatomer may contribute to coat depolymerization and release (Zink et al, 2009; Ren et al, 2009; Tripathi et al, 2009; reviewed in Barlowe and Fink, 2013).

Preceded by: Retrograde vesicle is tethered at the ER by the NRZ complex and t-SNAREs

Followed by: NSF and SNAPs bind cis-SNARE at the ER membrane

Literature references

Miller, EA., Barlowe, CK. (2013). Secretory protein biogenesis and traffic in the early secretory pathway. *Genetics, 193*, 383-410. ↗

Yip, CK., Ren, Y., Jeffrey, PD., Tripathi, A., Walz, T., Hughson, FM. et al. (2009). A structure-based mechanism for vesicle capture by the multisubunit tethering complex Dsl1. *Cell, 139*, 1119-29. ↗

Ren, Y., Jeffrey, PD., Tripathi, A., Hughson, FM. (2009). Structural characterization of Tip20p and Dsl1p, subunits of the Dsl1p vesicle tethering complex. *Nat. Struct. Mol. Biol., 16*, 114-23. ↗

Wenzel, D., Zink, S., Schmitt, HD., Wurm, CA. (2009). A link between ER tethering and COP-I vesicle uncoating. *Dev. Cell, 17*, 403-16. ↗

Editions

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

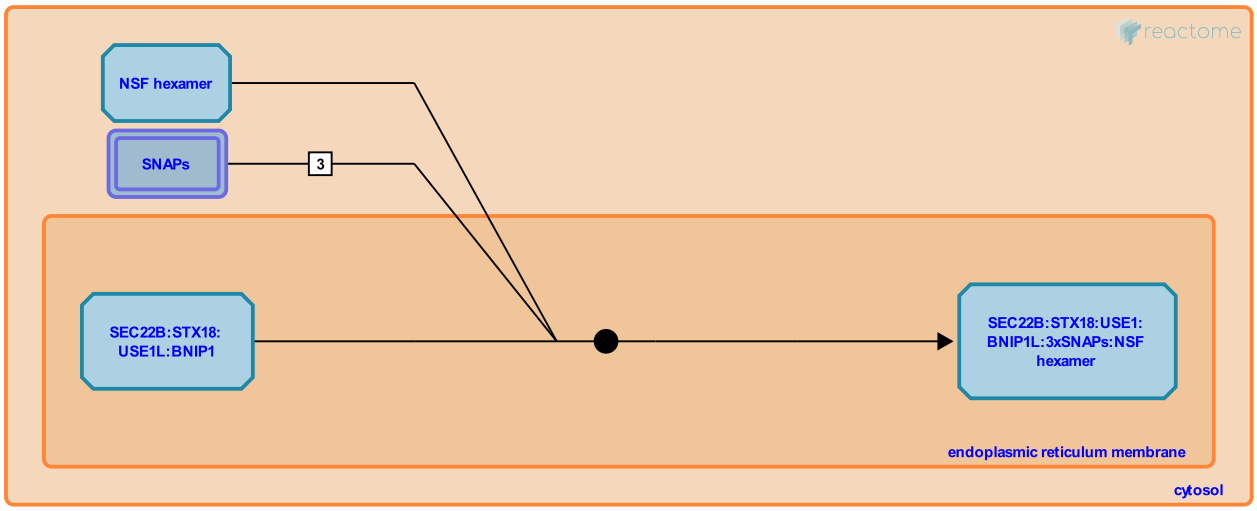
NSF and SNAPs bind cis-SNARE at the ER membrane ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811425

Type: binding

Compartments: endoplasmic reticulum membrane



After membrane fusion, the 4-membered cis-SNARE complex is dissociated in an ATP-dependent manner by SNAP and NSF (Mayer et al, 1996; Sollner et al, 1993; reviewed in Jahn and Scheller, 2006; Sudhof and Rothman, 2009).

Preceded by: COPI vesicle uncoating at the ER

Followed by: NSF ATPase activity dissociates cis-SNARE at the ER

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

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

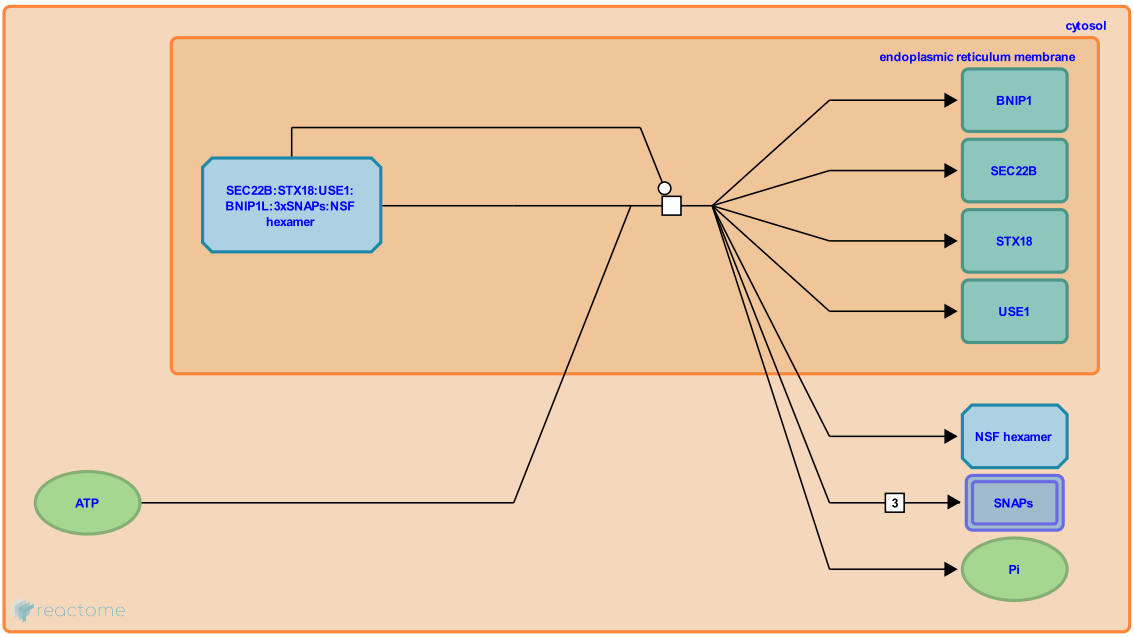
NSF ATPase activity dissociates cis-SNARE at the ER ↗

Location: COPI-dependent Golgi-to-ER retrograde traffic

Stable identifier: R-HSA-6811422

Type: transition

Compartments: endoplasmic reticulum 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 at the ER membrane

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. ↗

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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

2015-11-09	Authored, Edited	Rothfels, K.
2016-02-02	Reviewed	Gillespie, ME.

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