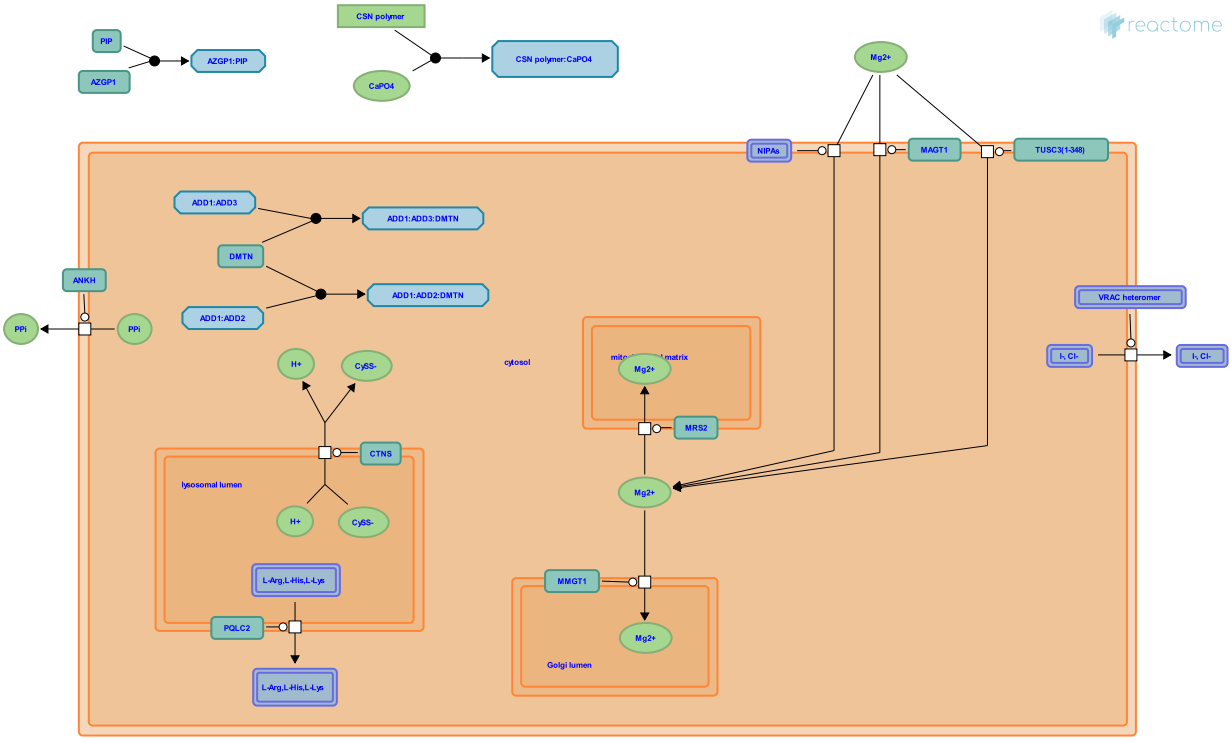
Miscellaneous transport and binding events



D'Eustachio, P., Jassal, B.

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

18/04/2024

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

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. ↗

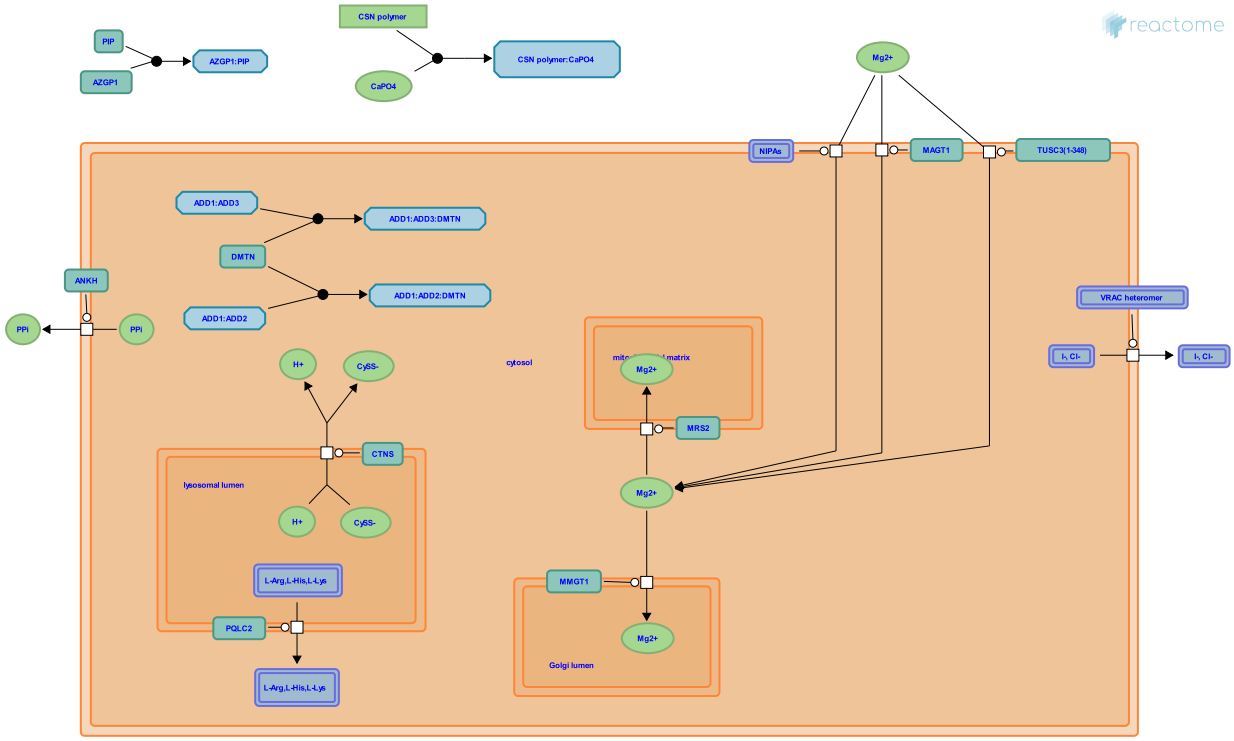
Sidiropoulos, K., Viteri, G., Sevilla, C., Jupe, S., Webber, M., Orlic-Milacic, M. et al. (2017). Reactome enhanced pathway visualization. *Bioinformatics, 33*, 3461-3467. ↗

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. ↗

Miscellaneous transport and binding events ↗

Stable identifier: R-HSA-5223345



This section contains known transport and binding events that as of yet cannot be placed in exisiting pathways (Purves 2001, He et al. 2009, Rees et al. 2009).

Literature references

Purves, D. (2001). Chapter 4: Channels and Transporters, Neuroscience (2nd ed.).

Nebert, DW., He, L., Vasiliou, K. (2009). Analysis and update of the human solute carrier (SLC) gene superfamily. *Hum Genomics, 3*, 195-206. ↗

Lewinson, O., Johnson, E., Rees, DC. (2009). ABC transporters: the power to change. *Nat Rev Mol Cell Biol, 10*, 218-27. ↗

Editions

2014-01-06	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

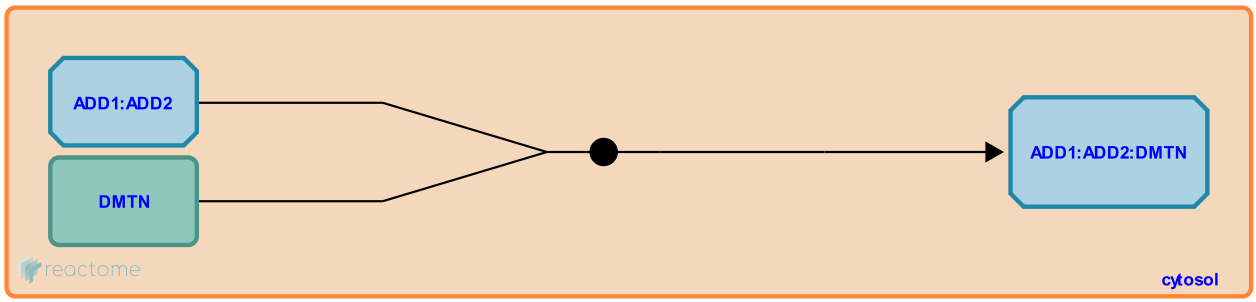
ADD1:ADD2 binds DMTN ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5226979

Type: binding

Compartments: cytosol



Alpha-adducin (ADD1 aka ADDA) (Joshi et al. 1991) is a ubiquitously expressed, membrane-cytoskeletal protein that can promote the assembly of the spectrin-actin network. It is functional in a heterodimeric form, in complex with either a beta (ADD2 aka ADDB) (Khan et al. 2008) or a gamma (ADD3 aka ADDL) subunit (Citterio et al. 2003). Either complex is able to bind dematin (DMTN) (Azim et al. 1995), a membrane-cytoskeletal protein that can induce F-actin bundles formation and stabilization. It can also bind the erythrocyte membrane glucose transporter 1 (SLC2A1 aka GLUT1), and hence stabilise the spectrin-actin network to the erythrocytic plasma membrane (Khan et al. 2008).

Literature references

Azim, AC., Beggs, AH., Knoll, JH., Chishti, AH. (1995). Isoform cloning, actin binding, and chromosomal localization of human erythroid dematin, a member of the villin superfamily. *J. Biol. Chem., 270*, 17407-13. ↗

Zeng, L., Khan, AA., Hanada, T., Mohseni, M., Chishti, AH., Jeong, JJ. et al. (2008). Dematin and adducin provide a novel link between the spectrin cytoskeleton and human erythrocyte membrane by directly interacting with glucose transporter-1. *J. Biol. Chem., 283*, 14600-9. ↗

Joshi, R., Otto, E., Gilligan, DM., McLaughlin, T., Bennett, V. (1991). Primary structure and domain organization of human alpha and beta adducin. *J. Cell Biol., 115*, 665-75. ↗

Bianchi, G., Catalano, M., Tizzoni, L., Citterio, L., Barlassina, C., Zerbini, G. (2003). Expression analysis of the human adducin gene family and evidence of ADD2 beta4 multiple splicing variants. *Biochem. Biophys. Res. Commun., 309*, 359-67. ↗

Editions

2014-01-09	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

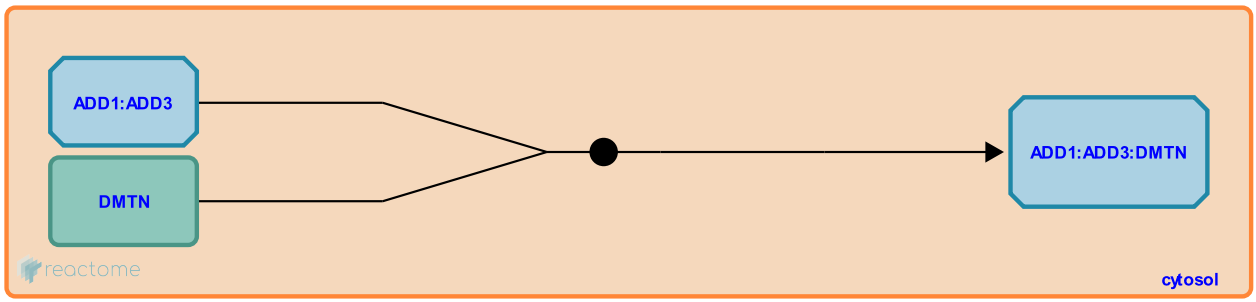
ADD1:ADD3 binds DMTN ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5226999

Type: binding

Compartments: cytosol



Alpha-adducin (ADD1 aka ADDA) (Joshi et al. 1991) is a ubiquitously expressed, membrane-cytoskeletal protein that can promote the assembly of the spectrin-actin network. It is functional in a heterodimeric form, in complex with either a beta (ADD2 aka ADDB) (Khan et al. 2008) or a gamma (ADD3 aka ADDL) subunit (Citterio et al. 2003). Either complex is able to bind dematin (DMTN) (Azim et al. 1995), a membrane-cytoskeletal protein that can induce F-actin bundles formation and stabilization. It can also bind the erythrocyte membrane glucose transporter 1 (SLC2A1 aka GLUT1), and hence stabilise the spectrin-actin network to the erythrocytic plasma membrane (Khan et al. 2008).

Literature references

Azim, AC., Beggs, AH., Knoll, JH., Chishti, AH. (1995). Isoform cloning, actin binding, and chromosomal localization of human erythroid dematin, a member of the villin superfamily. *J. Biol. Chem., 270*, 17407-13. ↗

Zeng, L., Khan, AA., Hanada, T., Mohseni, M., Chishti, AH., Jeong, JJ. et al. (2008). Dematin and adducin provide a novel link between the spectrin cytoskeleton and human erythrocyte membrane by directly interacting with glucose transporter-1. *J. Biol. Chem., 283*, 14600-9. ↗

Joshi, R., Otto, E., Gilligan, DM., McLaughlin, T., Bennett, V. (1991). Primary structure and domain organization of human alpha and beta adducin. *J. Cell Biol., 115*, 665-75. ↗

Bianchi, G., Catalano, M., Tizzoni, L., Citterio, L., Barlassina, C., Zerbini, G. (2003). Expression analysis of the human adducin gene family and evidence of ADD2 beta4 multiple splicing variants. *Biochem. Biophys. Res. Commun., 309*, 359-67. ↗

Editions

2014-01-09	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

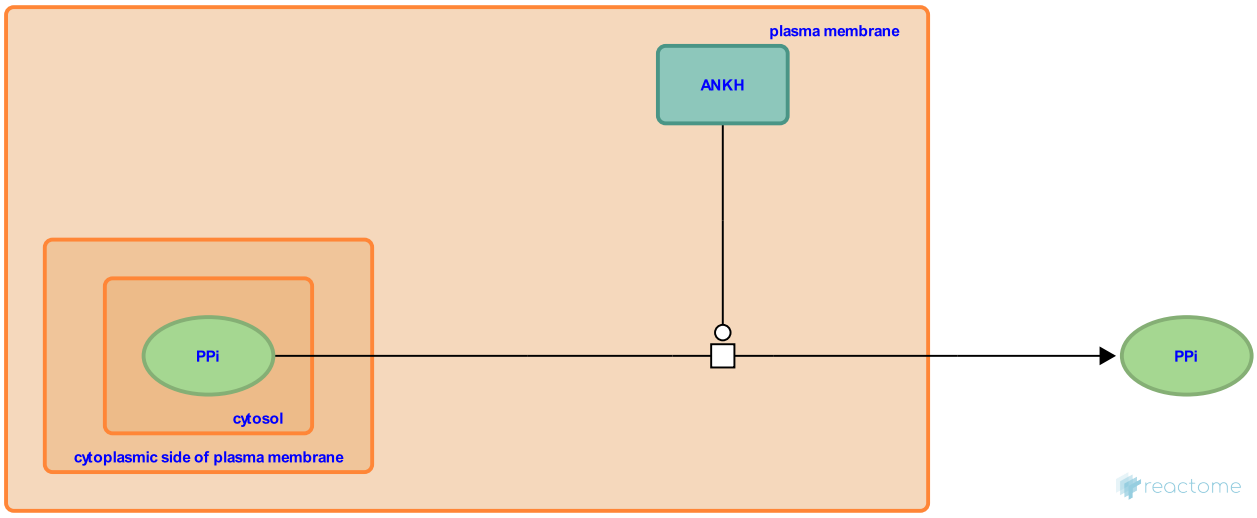
ANKH transports PPi from cytosol to extracellular region ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5226964

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Progressive ankylosis protein homolog (ANKH) is a putative transmembrane pyrophosphate (PPi) transport channel protein found in osteoblasts of various bones. It mediates the transport of cytosolic PPi to the extracellular matrix. Abnormal transport of PPi is implicated in familial calcium pyrophosphate dihydrate deposition (CPPD) disease. There are two forms of CPPD disease: CCAL1 and CCAL2. Defects in ANKH can cause chondrocalcinosis (CCAL2; MIM:118600), a chronic condition in which PPi crystals deposit in the joint fluid, cartilage, and periarticular tissues and there is calcium deposition in articular cartilage (Pendleton et al. 2002, Williams et al. 2002, Williams et al. 2003). Defects in ANKH can also cause craniometaphyseal dysplasia, autosomal dominant (CMDD; MIM:123000), an osteochondrodysplasia characterised by progressive thickening and increased mineral density of craniofacial bones and abnormal modelling of metaphyses in long bones (Nurnberg et al. 2001, Reichenberger et al. 2001).

Literature references

Chandler, D., Mundlos, S., Harrop, K., Nürnberg, P., Westermann, F., Goldblatt, J. et al. (2001). Heterozygous mutations in ANKH, the human ortholog of the mouse progressive ankylosis gene, result in craniometaphyseal dysplasia. *Nat. Genet., 28*, 37-41. ↗

Williams, CJ., Doherty, M., McCarty, DJ., Peariso, S., Reginato, AJ., Pendleton, A. et al. (2003). Mutations in the amino terminus of ANKH in two US families with calcium pyrophosphate dihydrate crystal deposition disease. *Arthritis Rheum., 48*, 2627-31. ↗

Beighton, P., Grange, DK., Sommer, A., Sellars, S., Raposo do Amaral, CM., Hamersma, H. et al. (2001). Autosomal dominant craniometaphyseal dysplasia is caused by mutations in the transmembrane protein ANK. *Am. J. Hum. Genet., 68*, 1321-6. ↗

Russell, RG., Williams, CJ., Cuthbertson, J., Timms, A., Brown, MA., Wordsworth, BP. et al. (2002). Autosomal dominant familial calcium pyrophosphate dihydrate deposition disease is caused by mutation in the transmembrane protein ANKH. *Am. J. Hum. Genet., 71*, 985-91. ↗

Netter, P., Ho, AM., Hughes, A., Kingsley, DM., Dixey, J., Loeuille, D. et al. (2002). Mutations in ANKH cause chondrocalcinosis. *Am. J. Hum. Genet., 71*, 933-40. ↗

Editions

2014-01-09	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

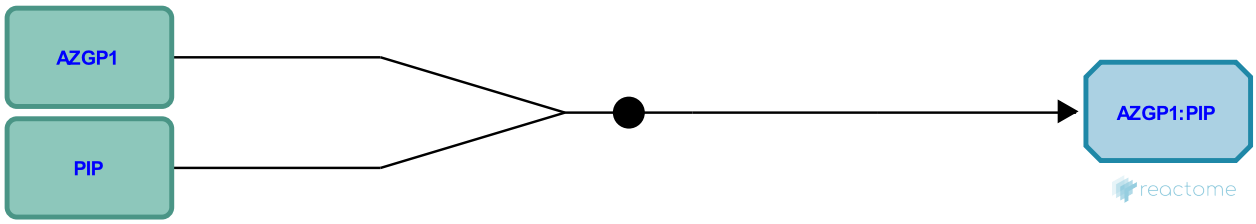
AZGP1 binds PIP ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5252072

Type: binding

Compartments: extracellular region



Zinc-alpha-2-glycoprotein (AZGP1) (Sanchez et al. 1997), a 41 kDa protein secreted in many bodily fluids, is thought to stimulate lipolysis and be the cause of the excessive fat loss seen in cancer cachexia (Russell et al. 2004). AZGP1 is able to bind prolactin-inducible protein (PIP) (Myal et al. 1991), another secreted protein overexpressed in certain breast cancers (Hassan et al. 2008, Debily et al. 2009).

Literature references

Bjorkman, PJ., Sánchez, LM., López-Otin, C. (1997). Biochemical characterization and crystalization of human Zn-alpha2-glycoprotein, a soluble class I major histocompatibility complex homolog. *Proc. Natl. Acad. Sci. U.S.A., 94*, 4626-30. ↗

Imbeaud, S., Mariage-Samson, R., Debily, MA., Eveno, E., Camarca, A., Marhomy, SE. et al. (2009). A functional and regulatory network associated with PIP expression in human breast cancer. *PLoS ONE, 4*, e4696. ↗

Iwasiow, B., Shiu, RP., Myal, Y., Wong, P., Robinson, DB., Tsuyuki, D. (1991). The prolactin-inducible protein (PIP/GCDFP-15) gene: cloning, structure and regulation. *Mol. Cell. Endocrinol., 80*, 165-75. ↗

Singh, TP., Bilgrami, S., Kumar, V., Yadav, S., Hassan, MI., Kaur, P. et al. (2008). Crystal structure of the novel complex formed between zinc alpha2-glycoprotein (ZAG) and prolactin-inducible protein (PIP) from human seminal plasma. *J. Mol. Biol., 384*, 663-72. ↗

Russell, ST., Tisdale, MJ., Zimmerman, TP., Domin, BA. (2004). Induction of lipolysis in vitro and loss of body fat in vivo by zinc-alpha2-glycoprotein. *Biochim. Biophys. Acta, 1636*, 59-68. ↗

Editions

2014-02-05	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

MMGT1 transports Mg2+ from cytosol to Golgi lumen ↗

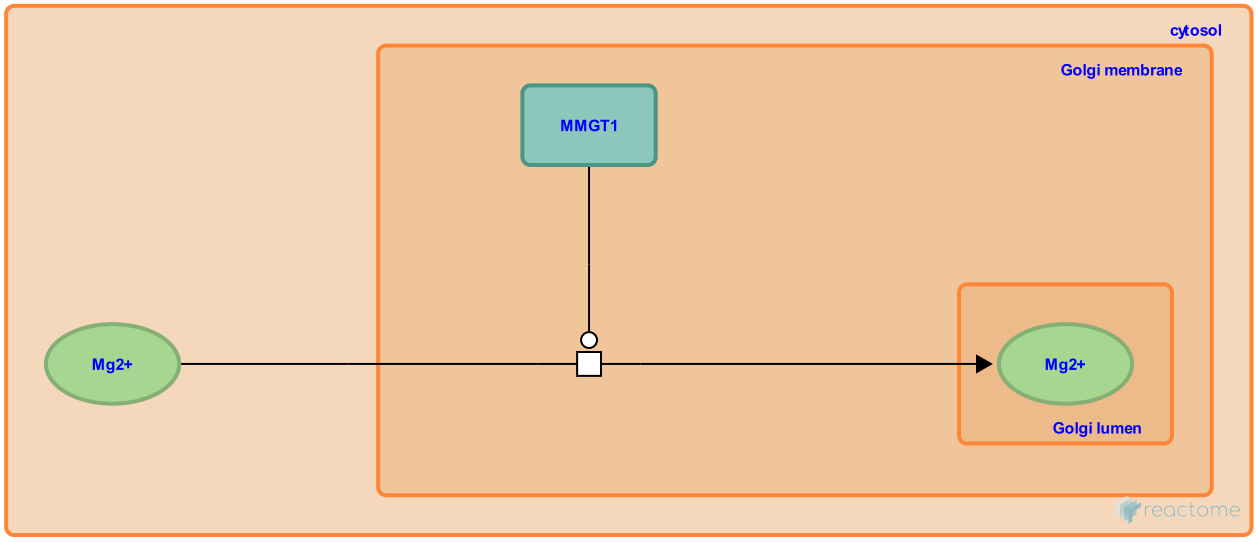
Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5336454

Type: transition

Compartments: Golgi membrane, Golgi lumen, cytosol

Inferred from: Mmgt1,2 transport Mg2+ from cytosol to Golgi lumen (Mus musculus)



Magnesium (Mg2+) is required for the catalytic activity of numerous metalloenzymes within a variety of subcellular organelles. Membrane magnesium transporter 1 (MMGT1) mediates the uptake of Mg2+ across the Golgi membrane. The human MMGT1 function is inferred from mouse experiments using the orthologous Mmgt1 and 2 (Goytain & Quamme 2008). MMGT1 is also found on the ER membrane as a component of the ER membrane protein complex (EMC) which functions to degrade incorrectly folded or assembled proteins by a ubiquitin- and proteasome-dependent process known as ER-associated degradation (ERAD). MMGT1 is not implicated in this function of protein quality control (Christianson et al. 2011).

Literature references

Tyler, RE., Bennett, EJ., Christianson, JC., Kopito, RR., Greenblatt, EJ., Sowa, ME. et al. (2012). Defining human ERAD networks through an integrative mapping strategy. *Nat. Cell Biol., 14*, 93-105. ↗

Goytain, A., Quamme, GA. (2008). Identification and characterization of a novel family of membrane magnesium transporters, MMgT1 and MMgT2. *Am. J. Physiol., Cell Physiol., 294*, C495-502. ↗

Editions

2014-03-03	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

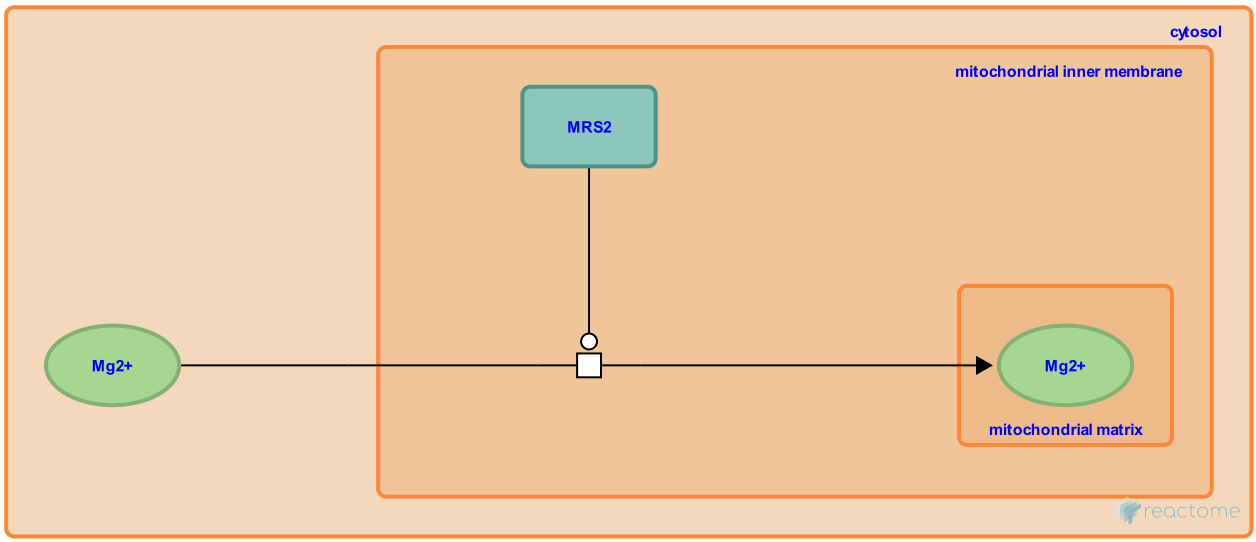
MRS2 transports Mg2+ from cytosol to mitochondrial matrix ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5336466

Type: transition

Compartments: mitochondrial inner membrane, mitochondrial matrix, cytosol



Magnesium (Mg2+) is required for the catalytic activity of numerous metalloenzymes within a variety of subcellular organelles. Magnesium transporter MRS2 homolog, mitochondrial (MRS2) mediates the influx of Mg2+ into the mitochondrial matrix (Zsurka et al. 2001). MRS2 is located on the inner mitochondrial membrane and its expression in yeast with a Mrs2-1 knock-out mutant partly restores mitochondrial magnesium concentrations that are otherwise much reduced (Zsurka et al. 2001).

Literature references

Schweyen, RJ., Gregán, J., Zsurka, G. (2001). The human mitochondrial Mrs2 protein functionally substitutes for its yeast homologue, a candidate magnesium transporter. *Genomics, 72*, 158-68. ↗

Editions

2014-03-03	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

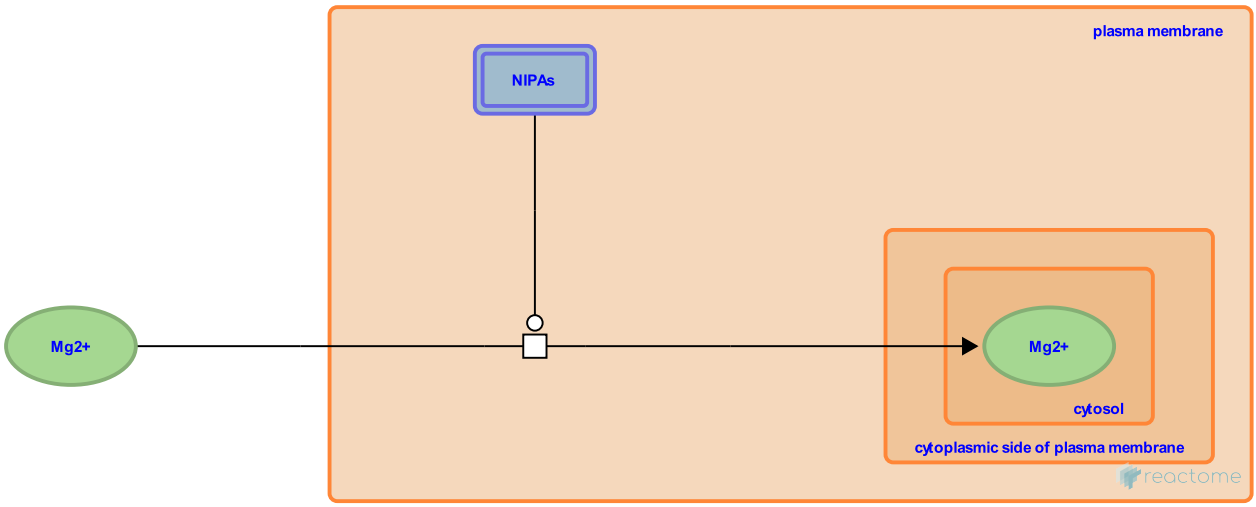
NIPAs transport Mg2+ from extracellular region to cytosol

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5336453

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Magnesium (Mg2+) is required for the catalytic activity of numerous metalloenzymes within a variety of subcellular organelles. Magnesium transporters NIPA1, 2, 3, 4 (NIPA1,2,3,4) and NIPA-like proteins 2 and 2 (NIPAL2 and 3) can act as Mg2+ transporters. They may also transport other divalent cations such as Fe2+, Mn2+ and Ba2+ but to a lesser extent than Mg2+. Human NIPA1 mediates Mg2+ uptake when expressed in Xenopus oocytes (Goytain et al. 2007). The other NIPA members are included as candidates based on NIPA1 function.

Literature references

Goytain, A., Hines, RM., Quamme, GA., El-Husseini, A. (2007). NIPA1(SPG6), the basis for autosomal dominant form of hereditary spastic paraplegia, encodes a functional Mg2+ transporter. *J. Biol. Chem., 282*, 8060-8.

Editions

2014-03-03	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

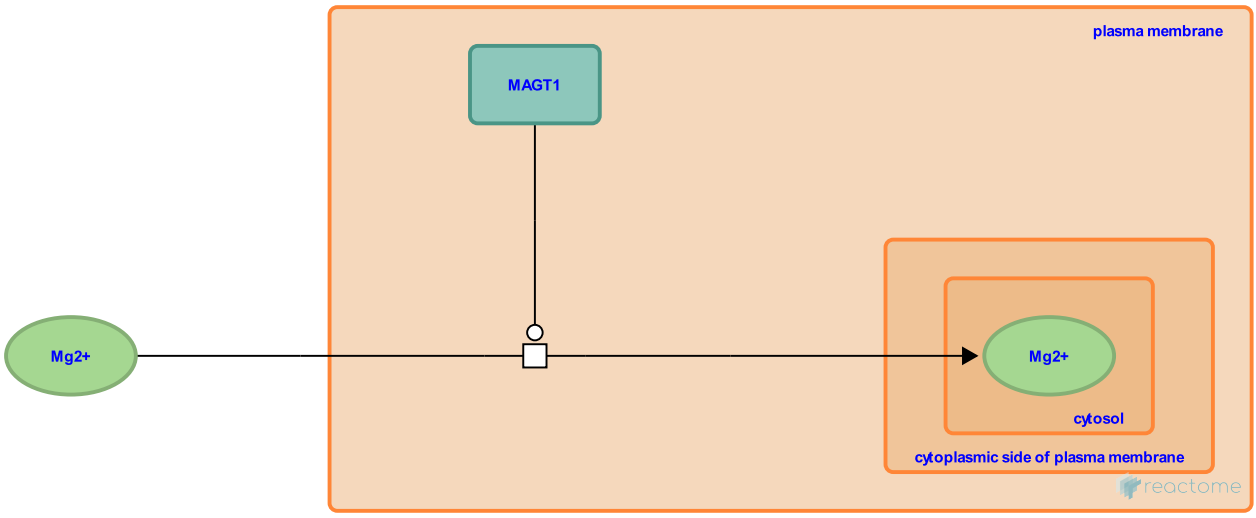
MAGT1 transports Mg2+ from extracellular region to cytosol ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5339538

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Magnesium (Mg2+) is required for the catalytic activity of numerous metalloenzymes within a variety of subcellular organelles. Magnesium transporter protein 1 (MAGT1) is ubiquitously expressed in all human tissues and is upregulated by low Mg2+ concentrations. It is an essential protein in Mg2+ uptake into cells (Zhou & Clapham 2009, Goytain & Quamme 2005).

Literature references

Zhou, H., Clapham, DE. (2009). Mammalian MagT1 and TUSC3 are required for cellular magnesium uptake and vertebrate embryonic development. *Proc. Natl. Acad. Sci. U.S.A., 106*, 15750-5. ↗

Quamme, GA., Goytain, A. (2005). Identification and characterization of a novel mammalian Mg2+ transporter with channel-like properties. *BMC Genomics, 6*, 48. ↗

Editions

2014-03-04	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

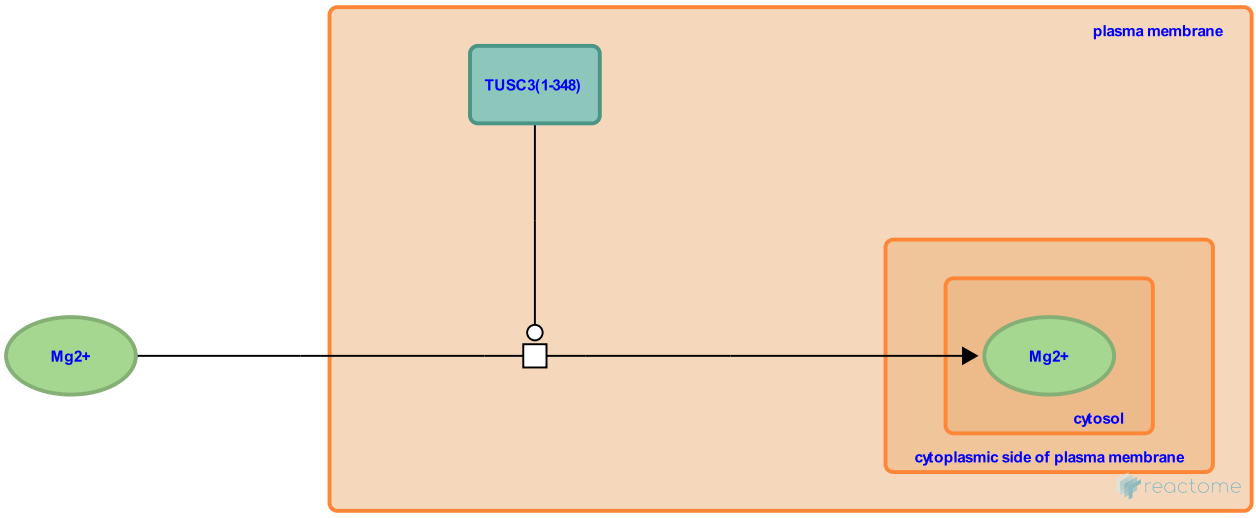
TUSC3 transports Mg2+ from extracellular region to cytosol ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5339528

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Magnesium (Mg2+) is required for the catalytic activity of numerous metalloenzymes within a variety of subcellular organelles. Tumor suppressor candidate 3 (TUSC3) is expressed in most non-lymphoid cells and tissues and is an essential protein in Mg2+ uptake into cells (Zhou & Clapham 2009).

Literature references

Zhou, H., Clapham, DE. (2009). Mammalian MagT1 and TUSC3 are required for cellular magnesium uptake and vertebrate embryonic development. *Proc. Natl. Acad. Sci. U.S.A., 106*, 15750-5. ↗

Editions

2014-03-04	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

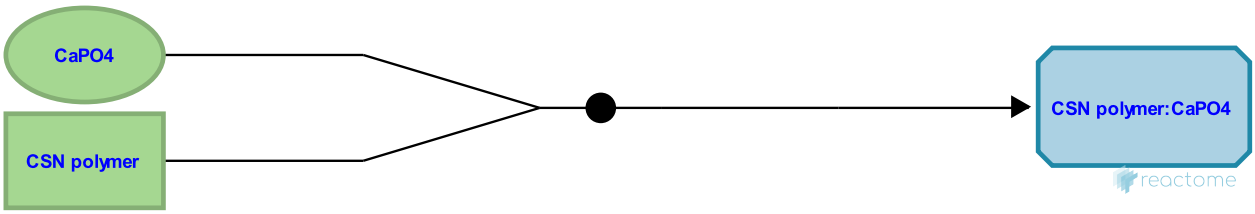
CSN polymer binds CaPO4 ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5340124

Type: binding

Compartments: extracellular region



In milk, caseins (CSNs) interact with calcium phosphate (CaPO4), forming large stable colloidal particles called micelles. These micelles make it possible to maintain a supersaturated CaPO4 concentration in milk, providing the newborn with sufficient calcium phosphate for the mineralisation of calcified tissues. Human alpha-S1-casein (CSN1S1) is able to bind CaPO4 in milk. CSN1S1 forms a disulfide cross-linked heteropolymer with kappa-casein (CSN3), another CSN that is thought to stabilise micelle formation and thus preventing casein precipitation in milk (Brignon et al. 1985, Rasmussen et al. 1995, Johnson et al. 1995).

Literature references

Johnsen, LB., Berglund, L., Rasmussen, LK., Petersen, TE. (1995). Characterization of three types of human alpha s1-casein mRNA transcripts. *Biochem. J., 309*, 237-42. ↗

Due, HA., Rasmussen, LK., Petersen, TE. (1995). Human alpha s1-casein: purification and characterization. *Comp. Biochem. Physiol. B, Biochem. Mol. Biol., 111*, 75-81. ↗

Ribadeau-Dumas, B., Brignon, G., Chtourou, A. (1985). Preparation and amino acid sequence of human kappa-casein . *FEBS Lett., 188*, 48-54. ↗

Editions

2014-03-10	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.

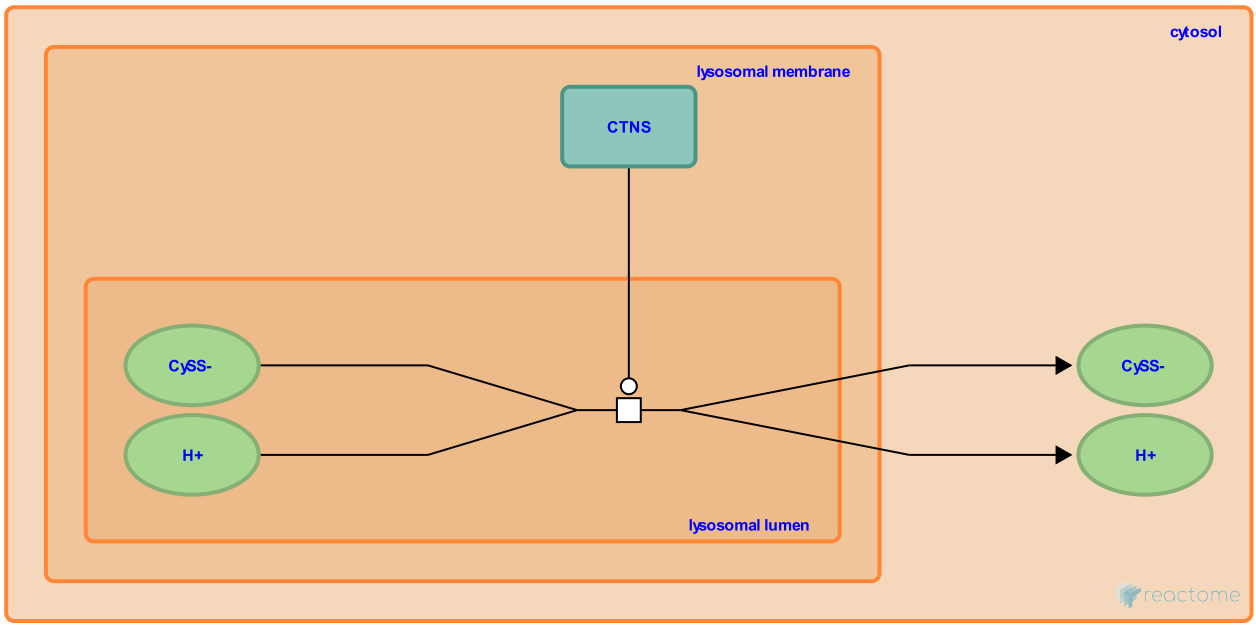
CTNS cotransports CySS-, H+ from lysosomal lumen to cytosol ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-5340130

Type: transition

Compartments: lysosomal lumen, cytosol, lysosomal membrane



Cystinosin (CTNS) is an integral lysosomal membrane protein which can transport L-cystine (CySS-, the oxidative product of two cysteine molecules linked via a disulfide bond) together with H+ out of lysosomes. CySS- is a component of hair, skin and nails. Defects in CTNS cause cystinosis, lysosomal storage-type diseases due to defective transport of CySS- across the lysosomal membrane (Town et al. 1998, Anikster et al. 1999; review Elmonem et al. 2016). Patients with cystinosis frequently exhibit blond hair and a fair complexion, suggesting an involvement in melanogenesis. Chiaverini et al. show CTNS is also localised to melanosomes. CTNS silencing led to a 75% reduction of melanin synthesis, caused by a degradation of tyrosinase (the enzyme responsible for melanin biosynthesis), thereby identifying a role for CTNS in melanogenesis (Chiaverini et al. 2012).

Literature references

Veys, KR., van den Heuvel, LP., Levtchenko, E., Soliman, NA., van Dyck, M., Elmonem, MA. (2016). Cystinosis: a review. *Orphanet J Rare Dis, 11*, 47. ↗

Anikster, Y., Shotelersuk, V., Gahl, WA. (1999). CTNS mutations in patients with cystinosis. *Hum. Mutat., 14*, 454-8. ↗

Attard, M., Bates, GP., Whitmore, SA., Cherqui, S., Callen, DF., van't Hoff, W. et al. (1998). A novel gene encoding an integral membrane protein is mutated in nephropathic cystinosis. *Nat. Genet., 18*, 319-24. ↗

Picardo, M., Flori, E., Berard, E., Antignac, C., Cochat, P., Fontas, E. et al. (2012). Cystinosin is a melanosomal protein that regulates melanin synthesis. *FASEB J., 26*, 3779-89. ↗

Editions

2014-03-10	Authored, Edited	Jassal, B.
2015-02-11	Reviewed	D'Eustachio, P.
2016-07-29	Revised	Jassal, B.

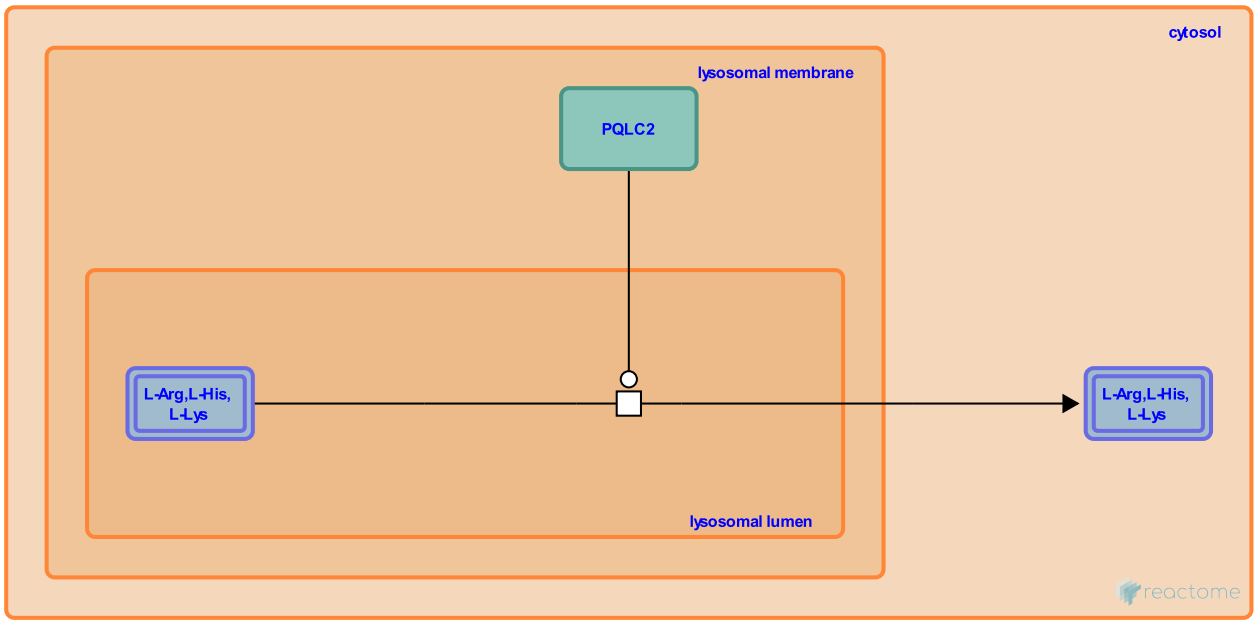
PQLC2 transports L-Arg,L-His,L-Lys from lysosomal lumen to cytosol ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-8932851

Type: transition

Compartments: lysosomal lumen, cytosol, lysosomal membrane



Lysosomal amino acid transporter 1 homolog (PQLC2) is a lysosomal membrane-associated protein that mediates the efflux of the cationic amino acids L-Arg, L-His and L-Lys from the lysosomal lumen to the cytosol, contributing to their homeostasis in cells. PQLC2 belongs to a family of heptahelical membrane proteins, together with the founding member cystinosin, the lysosomal cystine exporter defective in cystinosis. The family are characterised by a duplicated motif termed the PQ loop (Jezegou et al. 2012).

Literature references

Kieffer-Jaquinod, S., Llinares, E., Andre, B., Chadefaux-Vekemans, B., Anne, C., Sagné, C. et al. (2012). Heptahelical protein PQLC2 is a lysosomal cationic amino acid exporter underlying the action of cysteamine in cystinosis therapy. *Proc. Natl. Acad. Sci. U.S.A., 109*, E3434-43. ↗

Editions

2016-07-29	Authored, Edited	Jassal, B.
2016-08-12	Reviewed	D'Eustachio, P.

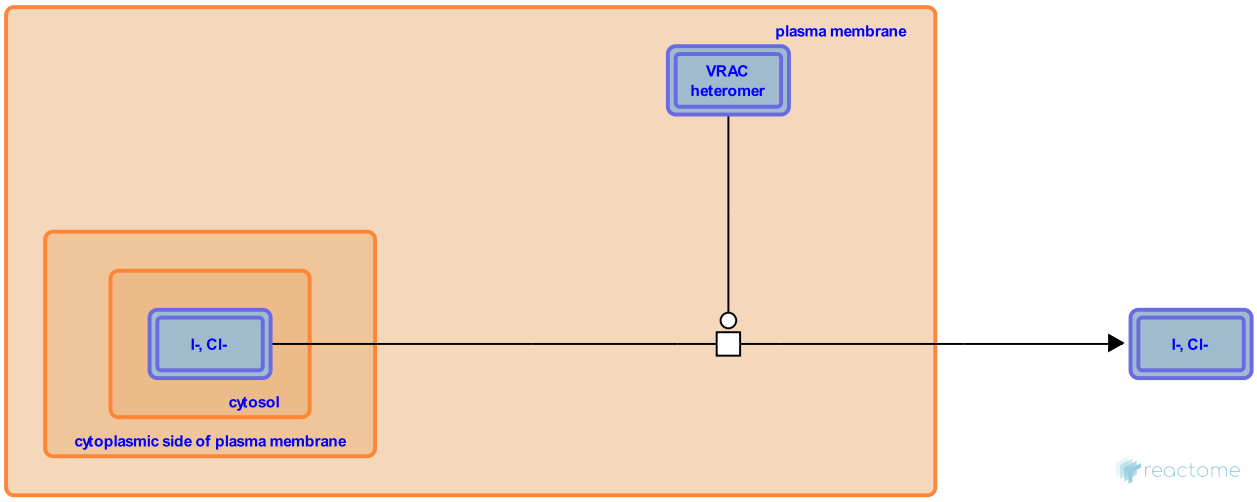
VRAC heteromer transports I-, Cl- from cytosol to extracellular region ↗

Location: Miscellaneous transport and binding events

Stable identifier: R-HSA-8941543

Type: transition

Compartments: plasma membrane, extracellular region, cytosol



Maintaining a constant cell volume in response to extracellular or intracellular osmotic changes is critical for cellular homeostasis. The volume-regulated anion channel (VRAC), localised on the plasma membrane, plays a key role in this process. VRAC is proposed to be a heterohexamer composed of an essential subunit, the volume-regulated anion channel subunit LRRC8A (SWELL1) (Qiu et al. 2014, Voss et al. 2014) and any one of four other LRRC8s; LRRC8B, LRRC8C, LRRC8D and LRRC8E (Syeda et al. 2016, Mongin 2016). The resulting diverse hexameric channels that can form are thought to produce diverse physiological roles for VRAC (Jentsch et al. 2016). VRAC mediates the so-called swelling-induced Cl- current (ICl, swell) which is primarily carried by Cl- but can also be other ions and small organic osmolytes. Indeed, VRAC has highest affinity for I- followed by Cl-. It counters cell swelling by causing a regulatory volume decrease (RVD) through ion and osmolyte efflux followed by release of osmotically-obligated water.

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Editions

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