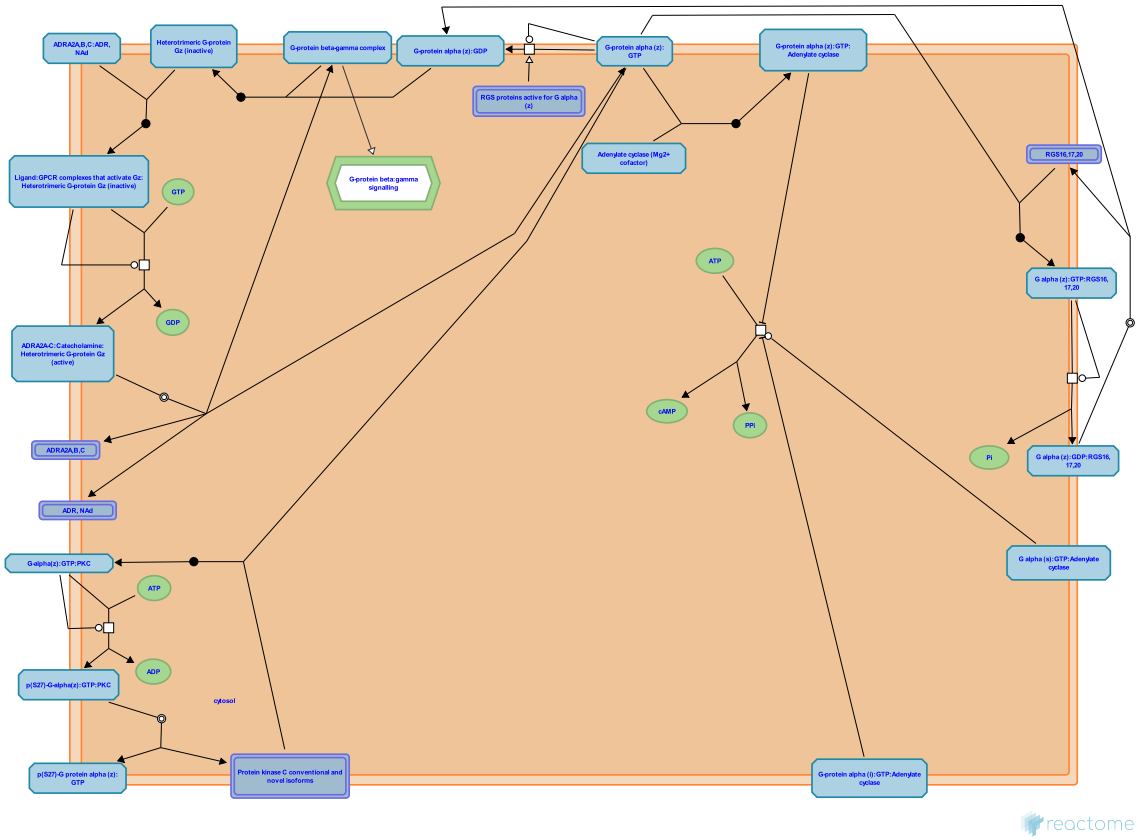
G alpha (z) signalling events



Akkerman, JW., D'Eustachio, P., Jupe, S., Siderovski, D., Varusai, TM.

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

30/01/2023

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. ↗

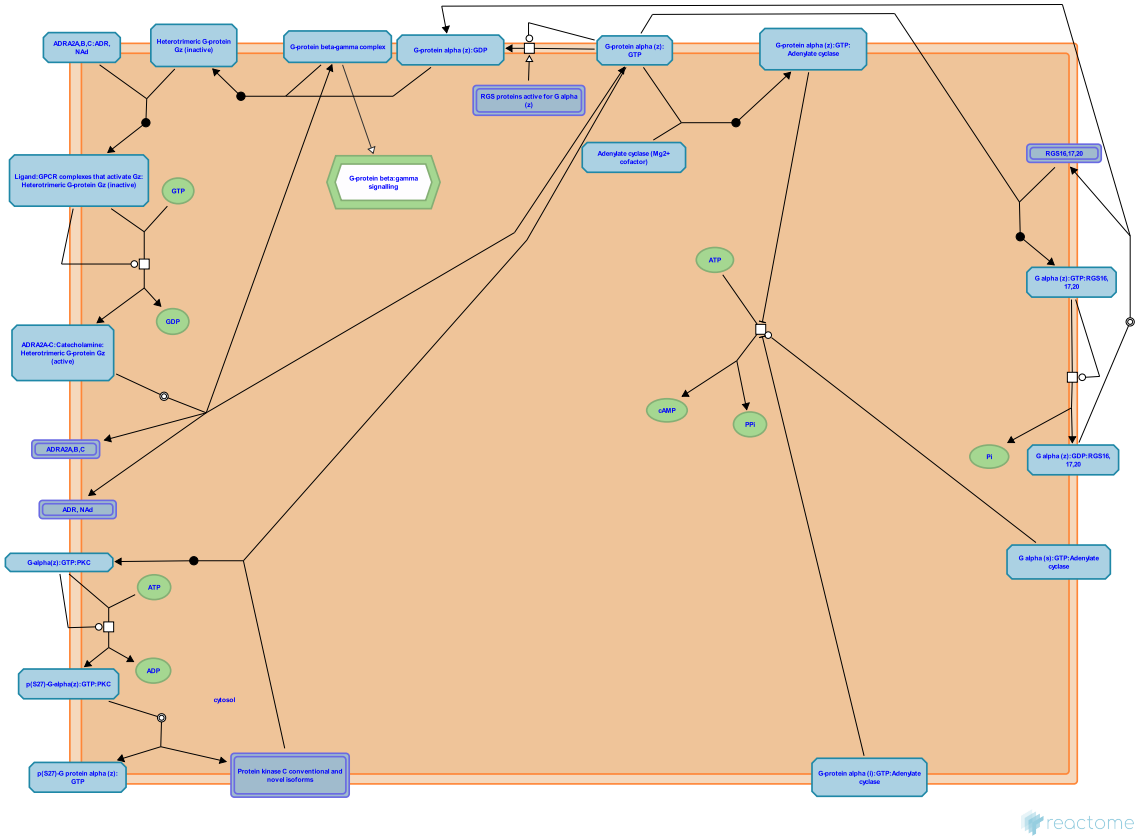
Fabregat, A., Jupe, S., Matthews, L., Sidiropoulos, K., Gillespie, M., Garapati, P. et al. (2018). The Reactome Pathway Knowledgebase. *Nucleic Acids Res, 46*, D649-D655. ↗

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. ↗

G alpha (z) signalling events ↗

Stable identifier: R-HSA-418597

Compartments: plasma membrane



The heterotrimeric G protein G alpha (z), is a member of the G (i) family. Unlike other G alpha (i) family members it lacks an ADP ribosylation site cysteine four residues from the carboxyl terminus and is thus pertussis toxin-insensitive. It inhibits adenylyl cyclase types I, V and VI (Wong Y H et al. 1992). G alpha (z) interacts with the Rap1 GTPase activating protein (Rap1GAP) to attenuate Rap1 signaling. Like all G-proteins G alpha (z) has an intrinsic GTPase activity, but this activity tends to be lower for the pertussis toxin insensitive G-proteins, most strikingly so for G alpha (z), whose kcat value for GTP hydrolysis is 200-fold lower than those of G alpha (s) or G alpha (i) (Grazziano et al. 1989). G alpha (z) knockout mice have disrupted platelet aggregation at physiological concentrations of epinephrine and responses to several neuroactive drugs are altered (Yang et al. 2000). Regulator of G-protein Signalling (RGS) proteins can regulate the activity of G alpha (z) (Soundararajan M et al. 2008).

Literature references

Manning, DR., Poncz, M., Gagnon, AW., Brass, LF., Gewirtz, A., Catani, L. (1991). Identification of Gz alpha as a pertussis toxin-insensitive G protein in human platelets and megakaryocytes. *Blood, 78*, 1247-53. ↗

Ho, MK., Wong, YH. (2001). G(z) signaling: emerging divergence from G(i) signaling. *Oncogene, 20*, 1615-25. ↗

Editions

2009-04-24	Authored	Jupe, S.
2009-06-03	Reviewed	Akkerman, JW.
2009-09-09	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

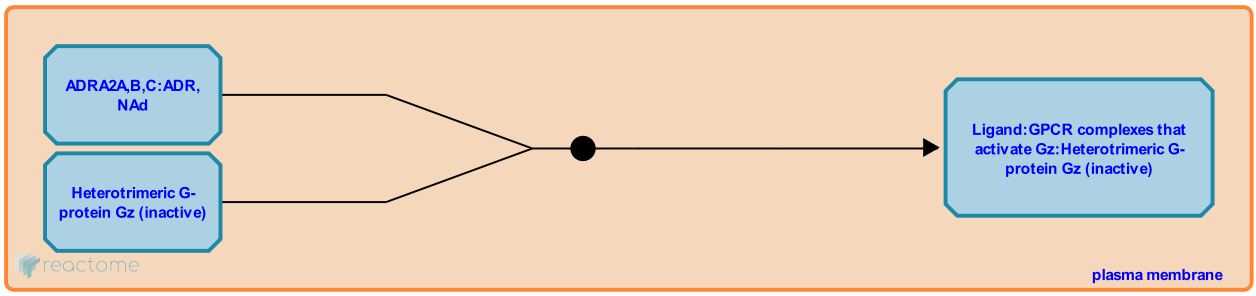
Liganded Gz-activating GPCRs bind inactive heterotrimeric G-protein Gz ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-749446

Type: binding

Compartments: plasma membrane



Gz is predominantly expressed in the nervous system and platelets. Gz interacts with receptors for many neurotransmitters and neuropeptides, including the adenosine A1, alpha2-adrenergic, dopamine D2, 5-HT1A, muscarinic M2, substance P, and all types of opioid receptors. In addition, Gz is capable of transducing signals from receptors such as the C5a and formyl peptide receptors. All these receptors can also signal via Gi. (Ho & Wong 2001).

Followed by: Liganded Gz-activating GPCR acts as a GEF for Gz

Literature references

Blendy, JA., Lucki, I., Manning, D., Dalvi, A., Yang, J., Poncz, M. et al. (2000). Loss of signaling through the G protein, Gz, results in abnormal platelet activation and altered responses to psychoactive drugs. *Proc Natl Acad Sci U S A, 97*, 9984-9. ↗

Ho, MK., Wong, YH. (2001). G(z) signaling: emerging divergence from G(i) signaling. *Oncogene, 20*, 1615-25. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2010-05-26	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

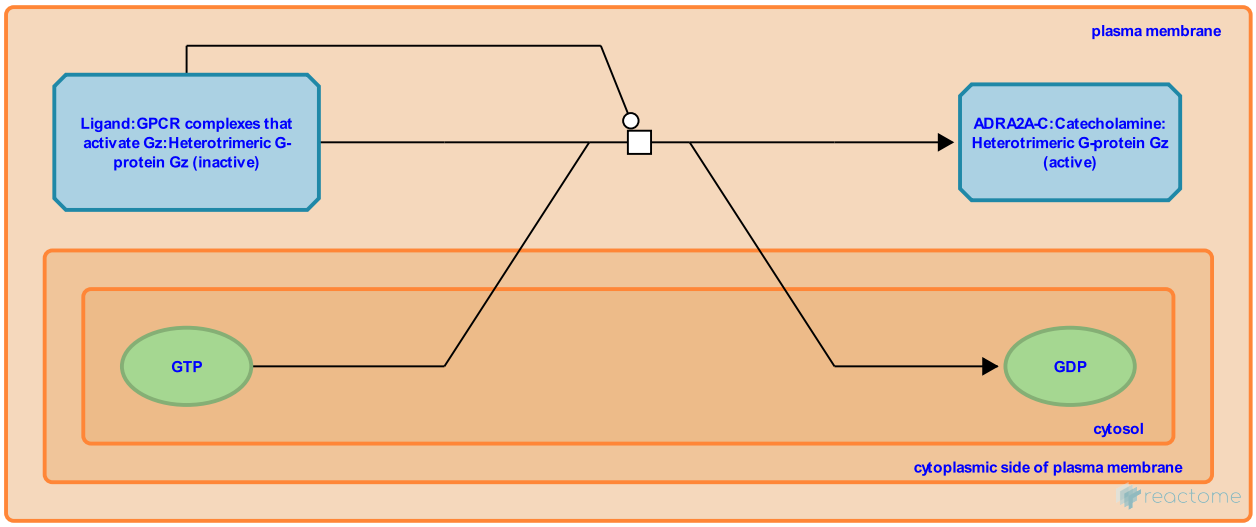
Liganded Gz-activating GPCR acts as a GEF for Gz ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-749453

Type: transition

Compartments: plasma membrane, cytosol



The liganded receptor undergoes a conformational change, generating a signal that is propagated in a manner that is not completely understood to the the G-protein. This stimulates the exchange of GDP for GTP in the G-protein alpha subunit, activating the G-protein.

This event is negatively regulated by some Activators of G protein signaling (AGS) proteins, a class of proteins identified in yeast functional screens for proteins able to activate G protein signaling in the absence of a G protein–coupled receptor (GPCR) (Cismowski et al. 1999, Takesono et al. 1999). AGS proteins contain G protein regulatory (GPR) motifs (also referred to as the GoLoco motif) that bind and stabilize the Galpha subunit in its GDP-bound conformation (Mochizuki et al. 1996, Peterson et al. 2000, Cao et al. 2004, Blumer & Lanier 2014). Some RGS proteins similarly bind to Galpha preventing the exchange of GDP for GTP (Soundararajan et al. 2008).

Preceded by: Liganded Gz-activating GPCRs bind inactive heterotrimeric G-protein Gz

Followed by: The Ligand:GPCR:Gz complex dissociates

Literature references

Ho, MK., Wong, YH. (2001). G(z) signaling: emerging divergence from G(i) signaling. *Oncogene, 20*, 1615-25. ↗

Editions

2009-02-26	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2010-05-26	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

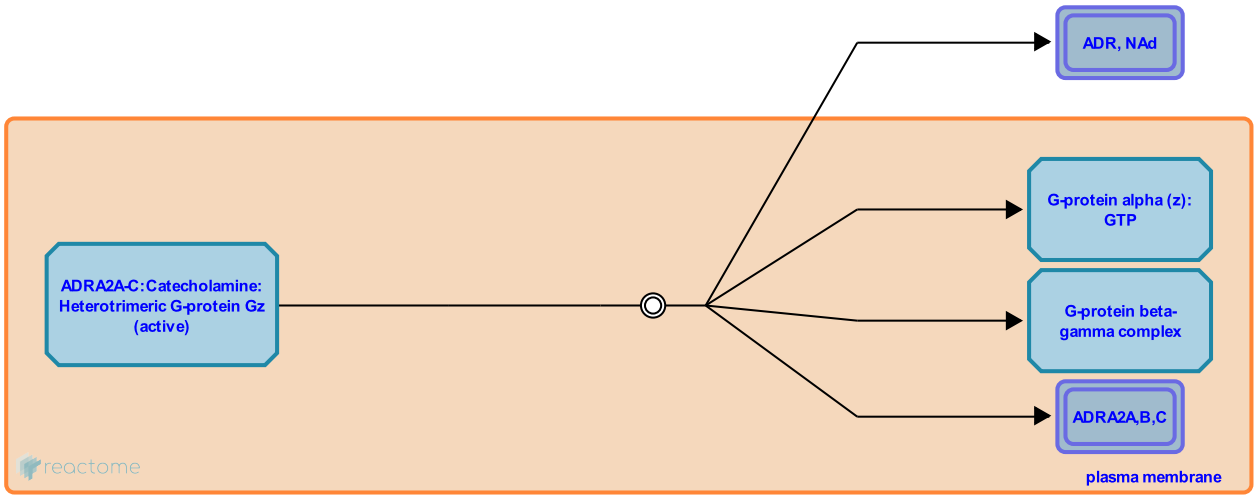
The Ligand:GPCR:Gz complex dissociates ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-751024

Type: dissociation

Compartments: plasma membrane, extracellular region



The classical view of G-protein signalling is that the G-protein alpha subunit dissociates from the beta:gamma dimer. Activated G alpha (z) and the beta:gamma dimer then participate in separate signaling cascades. Although G protein dissociation has been contested (e.g. Bassi et al. 1996), recent in vivo experiments have demonstrated that dissociation does occur, though possibly not to completion (Lambert 2008).

Preceded by: Liganded Gz-activating GPCR acts as a GEF for Gz

Followed by: Active G alpha (z) binds RGS proteins, PKC binds active G alpha (z), G alpha (z) auto-inactivates by hydrolysing GTP to GDP, G alpha (z) inhibits adenylate cyclase

Literature references

Lambert, NA. (2008). Dissociation of heterotrimeric g proteins in cells. *Sci Signal, 1*, re5. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2010-05-26	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

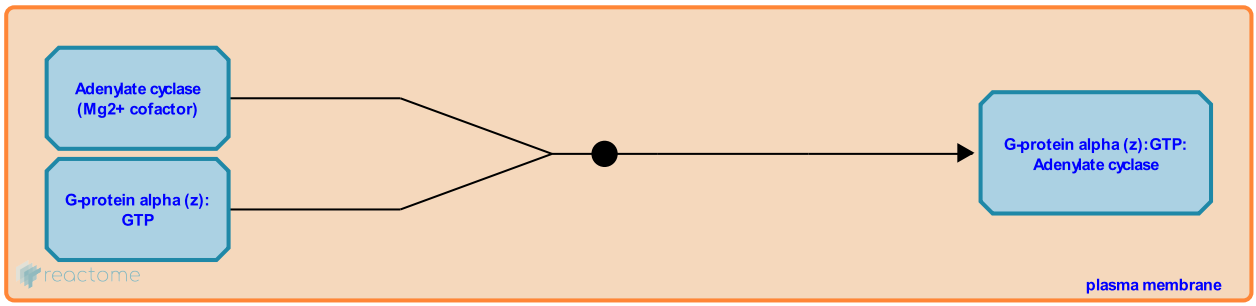
G alpha (z) inhibits adenylate cyclase ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-392064

Type: binding

Compartments: plasma membrane



G-proteins in the Gi class inhibit adenylate cyclase activity, decreasing the production of cAMP from ATP, which has many consequences but classically results in decreased activity of Protein Kinase A (PKA). cAMP also activates the cyclic nucleotide-gated ion channels, a process that is particularly important in olfactory cells. Experimental data for this reaction was obtained in vitro using rat G alpha (i) and dog Adenylate Cyclase.

Preceded by: The Ligand:GPCR:Gz complex dissociates

Followed by: Adenylate cyclase converts ATP to 3',5'-cyclic AMP (cAMP) and pyrophosphate

Literature references

Conklin, BR., Wong, YH., Bourne, HR. (1992). Gz-mediated hormonal inhibition of cyclic AMP accumulation. *Science, 255*, 339-42. ↗

Chiu, TT., Chan, JS., Wong, YH. (1995). Activation of type II adenylyl cyclase by the cloned mu-opioid receptor: coupling to multiple G proteins. *J Neurochem, 65*, 2682-9. ↗

Editions

2009-02-26	Authored	Jupe, S.
2009-06-03	Reviewed	Akkerman, JW.
2009-09-09	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

Adenylate cyclase converts ATP to 3',5'-cyclic AMP (cAMP) and pyrophosphate ↗

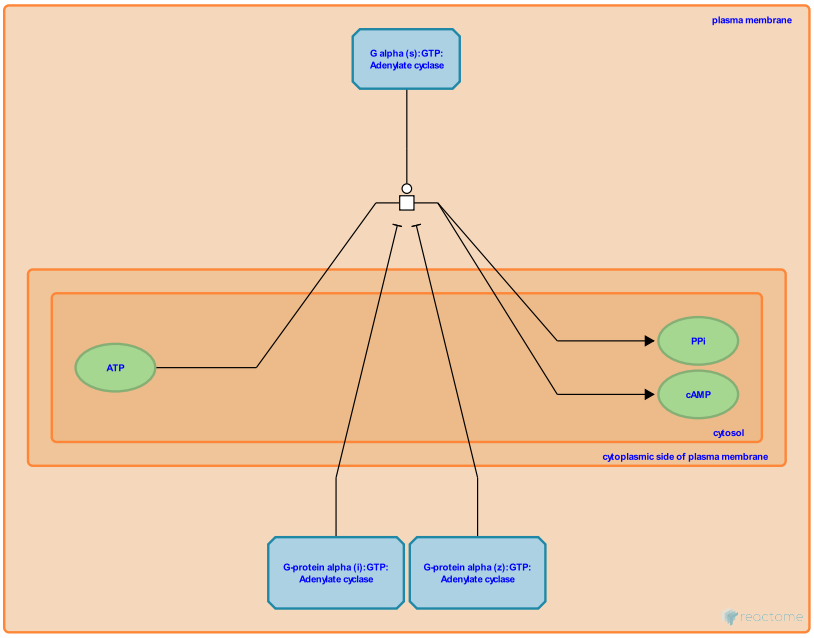
Location: G alpha (z) signalling events

Stable identifier: R-HSA-392129

Type: transition

Compartments: plasma membrane, cytosol

Inferred from: Adenylate cyclase converts ATP to 3',5'-cyclic AMP (cAMP) and pyrophosphate (Canis familiaris)



The activation of adenylyl (adenylate) cyclase (AC) results in the production of adenosine-3',5'-monophosphate i.e. cyclic AMP. Humans have 9 genes encoding membrane-associated AC and one encoding a soluble AC. Two of the classes of heterotrimeric G-proteins are named according to their effect on AC; G(s) stimulates all membrane-bound ACs (the s in G(s) denotes AC stimulatory); the G(i) class inhibits some AC isoforms, particularly 5 and 6. Beta-gamma subunits of heterotrimeric G-proteins can also regulate AC. Ca2+/Calmodulin activates some AC isoforms (1, 8 and 3) but is inhibitory to others (5 and 6).

Preceded by: G alpha (z) inhibits adenylate cyclase

Literature references

SUTHERLAND, EW., RALL, TW. (1958). Fractionation and characterization of a cyclic adenine ribonucleotide formed by tissue particles. *J Biol Chem, 232*, 1077-91. ↗

Editions

2009-05-22	Authored	Jupe, S.
2009-06-03	Reviewed	Akkerman, JW.
2009-09-09	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

PKC phosphorylates G alpha (z) ↗

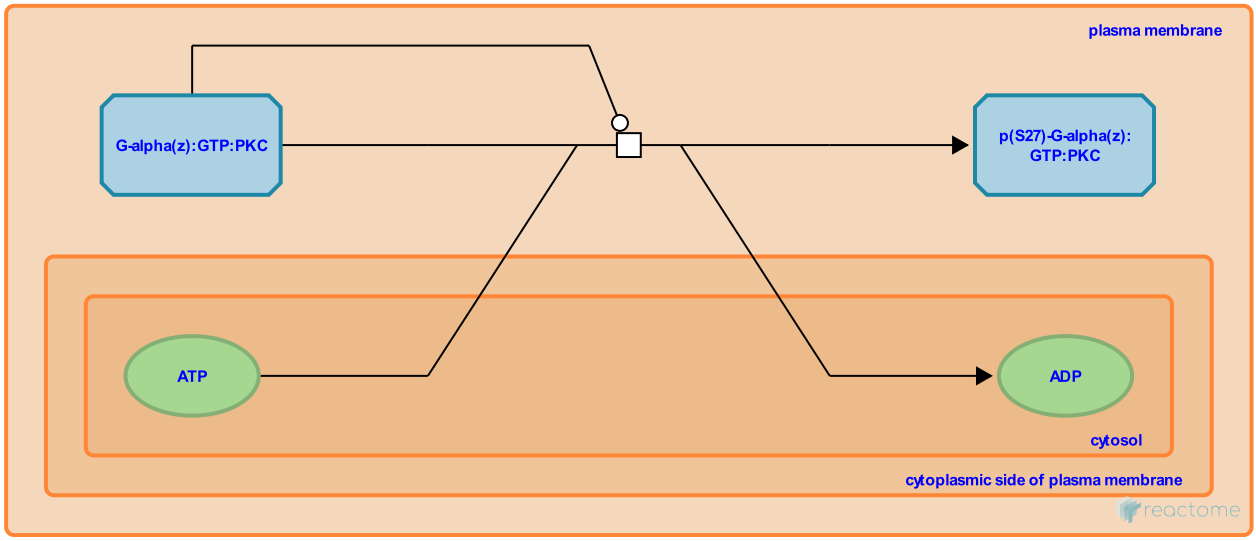
Location: G alpha (z) signalling events

Stable identifier: R-HSA-751040

Type: transition

Compartments: plasma membrane, cytosol

Inferred from: PKC (cow) phosphorylates G alpha z (rat) (Bos taurus)



G alpha z (Lounsbury et al. 1991) and G alpha 12 (Kozasa & Gilman, 1996) are excellent in vitro substrates for all three subtypes of protein kinase C (PKC). Activation of PKC in intact platelets by agents such as thrombin, thromboxane A2 (TXA2) analogues and phorbol esters leads to rapid and near-stoichiometric phosphorylation of G alpha z (Carlson et al. 1989). The primary phosphorylation site is Ser-27 (Lounsbury et al. 1993). This phosphorylation blocks the interaction of G alpha z with Gbeta:gamma suggesting that it is a regulatory mechanism for attenuating signalling by preventing subunit reassociation.

Preceded by: PKC binds active G alpha (z)

Followed by: G-alpha(z):PKC dissociates to give phosphorylated G alpha (z)

Literature references

Lounsbury, KM., Manning, DR., Casey, PJ., Brass, LF. (1991). Phosphorylation of Gz in human platelets. Selectivity and site of modification. *J Biol Chem, 266*, 22051-6. ↗

Casey, PJ., Fields, TA. (1995). Phosphorylation of Gz alpha by protein kinase C blocks interaction with the beta gamma complex. *J. Biol. Chem., 270*, 23119-25. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2010-05-24	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

G alpha (z) auto-inactivates by hydrolysing GTP to GDP ↗

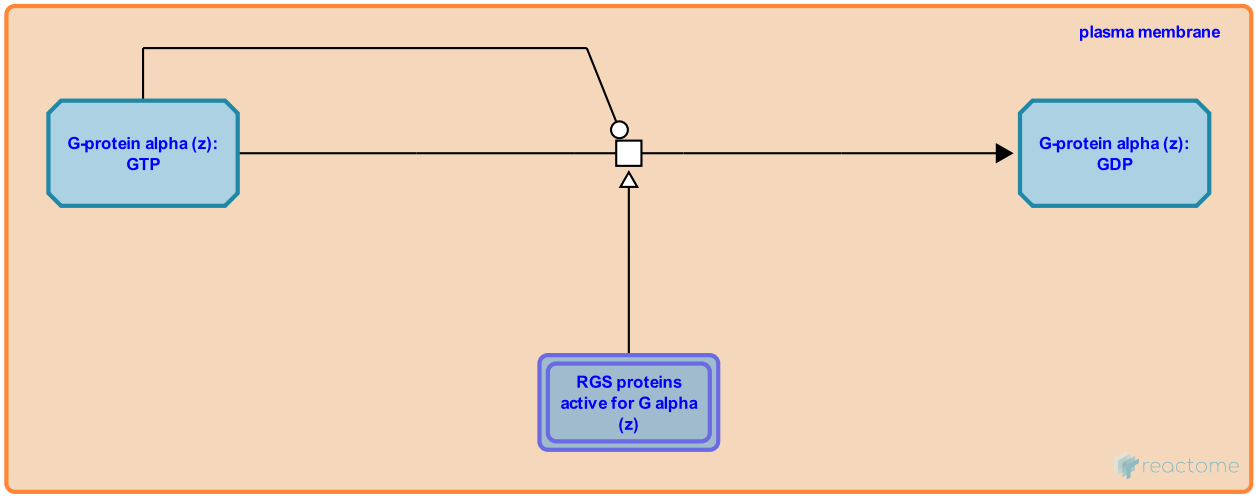
Location: G alpha (z) signalling events

Stable identifier: R-HSA-392133

Type: transition

Compartments: plasma membrane

Inferred from: G alpha (i)1 auto-inactivates by hydrolysing GTP to GDP (Rattus norvegicus)



When a ligand activates a G protein-coupled receptor, it induces a conformational change in the receptor (a change in shape) that allows the receptor to function as a guanine nucleotide exchange factor (GEF), stimulating the exchange of GDP for GTP on the G alpha subunit. In the traditional view of heterotrimeric protein activation, this exchange triggers the dissociation of the now active G alpha subunit from the beta:gamma dimer, initiating downstream signalling events. The G alpha subunit has intrinsic GTPase activity and will eventually hydrolyze the attached GTP to GDP, allowing reassociation with G beta:gamma. Additional GTPase-activating proteins (GAPs) stimulate the GTPase activity of G alpha, leading to more rapid termination of the transduced signal. In some cases the downstream effector may have GAP activity, helping to deactivate the pathway. This is the case for phospholipase C beta, which possesses GAP activity within its C-terminal region (Kleuss et al. 1994).

Preceded by: The Ligand:GPCR:Gz complex dissociates

Followed by: Inactive G alpha (z) reassociates with G beta:gamma

Literature references

Kleuss, C., Gilman, AG., Sprang, SR., Raw, AS., Lee, E. (1994). Mechanism of GTP hydrolysis by G-protein alpha subunits. *Proc Natl Acad Sci U S A, 91*, 9828-31. ↗

Editions

2009-02-27	Authored	Jupe, S.
2009-06-03	Reviewed	Akkerman, JW.
2009-09-09	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

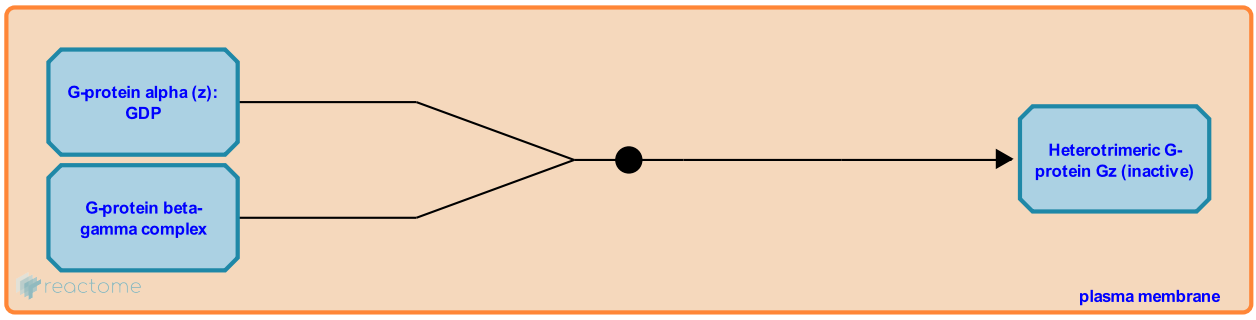
Inactive G alpha (z) reassociates with G beta:gamma ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-750988

Type: binding

Compartments: plasma membrane



The classical model of G-protein signaling suggests that the G-protein dissociates upon GPCR activation. The active G alpha (z) subunit then participates in signaling, until its intrinsic GTPase activity degrades the bound GTP to GDP. The inactive G alpha (z):GDP complex has much higher affinity for the G beta:gamma complex and consequently reassociates.

Preceded by: G alpha (z) auto-inactivates by hydrolysing GTP to GDP

Literature references

Oldham, WM., Hamm, HE. (2006). Structural basis of function in heterotrimeric G proteins. *Q Rev Biophys, 39*, 117-66 . ↗

Siderovski, DP., Willard, FS. (2005). The GAPs, GEFs, and GDIs of heterotrimeric G-protein alpha subunits. *Int J Biol Sci, 1*, 51-66. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2010-05-26	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

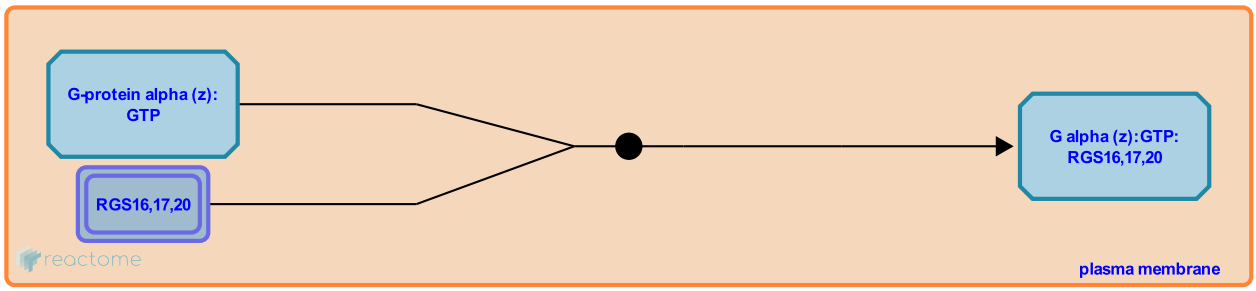
Active G alpha (z) binds RGS proteins ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-8981892

Type: binding

Compartments: plasma membrane, cytosol



G Protein Coupled Receptors (GPCR) sense extracellular signals and activate different Guanine nucleotide binding proteins (G proteins). Upon activation, GPCRs can replace the GDP with GTP in the alpha subunit of G proteins. GTP binding modifies the conformation of G alpha proteins and activates them. The Regulator of G protein Signalling (RGS) are GTPase Accelerating Proteins (GAPs) that can directly inhibit the G alpha subunit activity. There are at least 25 different types of RGS proteins known. RGS16, RGS17 and RGS20 can bind and stabilize the transition state of Guanine nucleotide binding protein G(z) subunit alpha (GNAZ) along its path to GTP hydrolysis. Subsequently, this leads to GTP hydrolysis and inactivation of GNAZ, terminating downstream signalling (Neubig & Siderovski 2002, Kach et al. 2012, Goto K et al. 2017). GNAZ inhibits adenylyl cyclase and interacts with Rap1GAP to attenuate Rap1 signaling.

Preceded by: The Ligand:GPCR:Gz complex dissociates

Followed by: G alpha (z) in G alpha (z):RGS complex is inactivated

Literature references

Neubig, RR., Siderovski, DP. (2002). Regulators of G-protein signalling as new central nervous system drug targets. *Nat Rev Drug Discov, 1*, 187-97. ↗

Wang, T., Doi, M., Murai, I., Goto, K., Kunisue, S., Okamura, H. (2017). G-protein-coupled receptor signaling through Gpr176, Gz, and RGS16 tunes time in the center of the circadian clock [Review]. *Endocr. J., 64*, 571-579. ↗

Kimple, AJ., Dowler, EF., Ball, LJ., Hutsell, SQ., Willard, FS., Soundararajan, M. et al. (2008). Structural diversity in the RGS domain and its interaction with heterotrimeric G protein alpha-subunits. *Proc. Natl. Acad. Sci. U.S.A., 105* , 6457-62. ↗

Editions

2017-07-27	Authored, Edited	Varusai, TM.
2018-09-03	Reviewed	Siderovski, D.

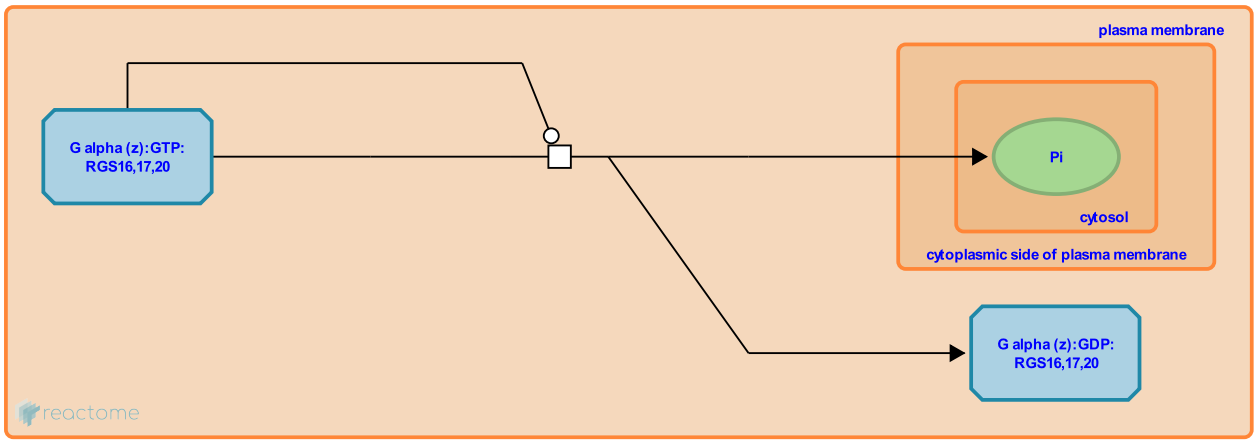
G alpha (z) in G alpha (z):RGS complex is inactivated ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-8982021

Type: transition

Compartments: plasma membrane



G Protein Coupled Receptors (GPCR) sense extracellular signals and activate different Guanine nucleotide binding proteins (G proteins). Upon activation, GPCRs can replace the GDP with GTP in the alpha subunit of G proteins. GTP binding modifies the conformation of G alpha proteins and activates them. The Regulator of G protein Signalling (RGS) are GTPase Accelerating Proteins (GAPs) that can directly inhibit the G alpha subunit activity. There are at least 25 different types of RGS proteins known. RGS16, RGS17 and RGS20 can bind and stabilize the transition state of Guanine nucleotide binding protein G(z) subunit alpha (GNAZ). Following this, RGS exerts its GAP activity on GNAZ and facilitates the hydrolyses GTP to GDP. This inactivates GNAZ and terminates downstream signalling (Neubig & Siderovski 2002, Kach et al. 2012, Goto K et al. 2017). GNAZ inhibits adenylyl cyclase and interacts with Rap1GAP to attenuate Rap1 signaling.

Preceded by: Active G alpha (z) binds RGS proteins

Followed by: G alpha (z):RGS complex dissociates to give inactive G alpha (z)

Literature references

Neubig, RR., Siderovski, DP. (2002). Regulators of G-protein signalling as new central nervous system drug targets. *Nat Rev Drug Discov, 1*, 187-97. ↗

Wang, T., Doi, M., Murai, I., Goto, K., Kunisue, S., Okamura, H. (2017). G-protein-coupled receptor signaling through Gpr176, Gz, and RGS16 tunes time in the center of the circadian clock [Review]. *Endocr. J., 64*, 571-579. ↗

Kimple, AJ., Dowler, EF., Ball, LJ., Hutsell, SQ., Willard, FS., Soundararajan, M. et al. (2008). Structural diversity in the RGS domain and its interaction with heterotrimeric G protein alpha-subunits. *Proc. Natl. Acad. Sci. U.S.A., 105* , 6457-62. ↗

Editions

2017-07-27	Authored, Edited	Varusai, TM.
2018-09-03	Reviewed	Siderovski, D.

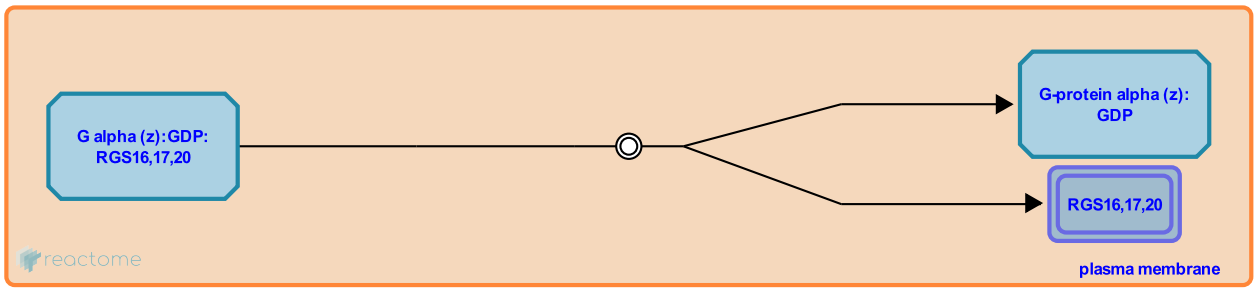
G alpha (z):RGS complex dissociates to give inactive G alpha (z) ↗

Location: G alpha (z) signalling events

Stable identifier: R-HSA-8982018

Type: dissociation

Compartments: plasma membrane, cytosol



G Protein Coupled Receptors (GPCR) sense extracellular signals and activate different Guanine nucleotide binding proteins (G proteins). Upon activation, GPCRs can replace the GDP with GTP in the alpha subunit of G proteins. GTP binding modifies the conformation of G alpha proteins and activates them. The Regulator of G protein Signalling (RGS) are GTPase Accelerating Proteins (GAPs) that can directly inhibit the G alpha subunit activity. There are at least 25 different types of RGS proteins known. RGS16, RGS17 and RGS20 can bind and stabilize the transition state of Guanine nucleotide binding protein G(z) subunit alpha (GNAZ). Subsequently, RGS proteins in the complex facilitate the hydrolysis of GNAZ:GTP to GNAZ:GDP. Following this, the complex dissociates releasing inactive GNAZ ((Neubig & Siderovski 2002, Kach et al. 2012, Goto K et al. 2017). GNAZ inhibits adenylyl cyclase and interacts with Rap1GAP to attenuate Rap1 signaling.

Preceded by: G alpha (z) in G alpha (z):RGS complex is inactivated

Literature references

Neubig, RR., Siderovski, DP. (2002). Regulators of G-protein signalling as new central nervous system drug targets. *Nat Rev Drug Discov, 1*, 187-97. ↗

Wang, T., Doi, M., Murai, I., Goto, K., Kunisue, S., Okamura, H. (2017). G-protein-coupled receptor signaling through Gpr176, Gz, and RGS16 tunes time in the center of the circadian clock [Review]. *Endocr. J., 64*, 571-579. ↗

Kimple, AJ., Dowler, EF., Ball, LJ., Hutsell, SQ., Willard, FS., Soundararajan, M. et al. (2008). Structural diversity in the RGS domain and its interaction with heterotrimeric G protein alpha-subunits. *Proc. Natl. Acad. Sci. U.S.A., 105* , 6457-62. ↗

Editions

2017-07-27	Authored, Edited	Varusai, TM.
2018-09-03	Reviewed	Siderovski, D.

PKC binds active G alpha (z) ↗

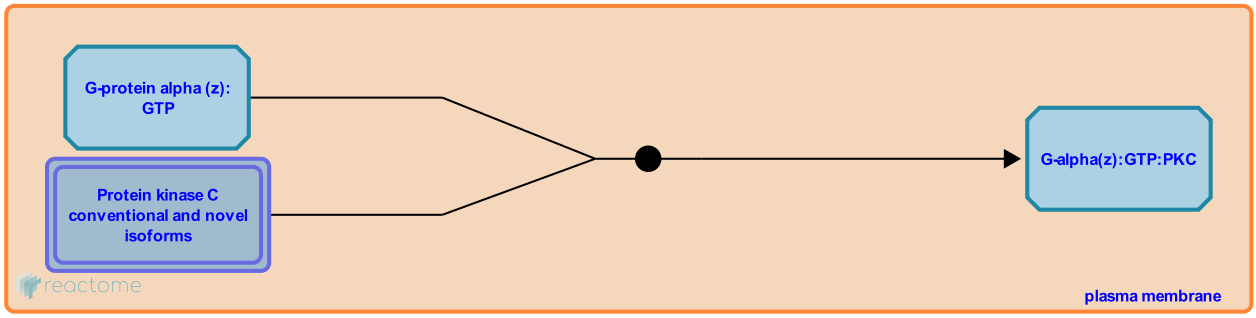
Location: G alpha (z) signalling events

Stable identifier: R-HSA-8982703

Type: binding

Compartments: plasma membrane

Inferred from: PKC (cow) binds G alpha z (rat) (Bos taurus)



G alpha z (Lounsbury et al. 1991) and G alpha 12 (Kozasa & Gilman, 1996) are excellent in vitro substrates for all three subtypes of protein kinase C (PKC). Activation of PKC in intact platelets by agents such as thrombin, thromboxane A2 (TXA2) analogues and phorbol esters leads to rapid and near-stoichiometric phosphorylation of G alpha z (Carlson et al. 1989). PKC can bind to G alpha z and facilitate its phosphorylation at Ser-27 (Lounsbury et al. 1993). This phosphorylation blocks the interaction of G alpha z with Gbeta:gamma suggesting that it is a regulatory mechanism for attenuating signalling by preventing subunit reassociation.

Preceded by: The Ligand:GPCR:Gz complex dissociates

Followed by: PKC phosphorylates G alpha (z)

Literature references

Lounsbury, KM., Manning, DR., Casey, PJ., Brass, LF. (1991). Phosphorylation of Gz in human platelets. Selectivity and site of modification. *J Biol Chem, 266*, 22051-6. ↗

Casey, PJ., Fields, TA. (1995). Phosphorylation of Gz alpha by protein kinase C blocks interaction with the beta gamma complex. *J. Biol. Chem., 270*, 23119-25. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2017-05-11	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

G-alpha(z):PKC dissociates to give phosphorylated G alpha (z) ↗

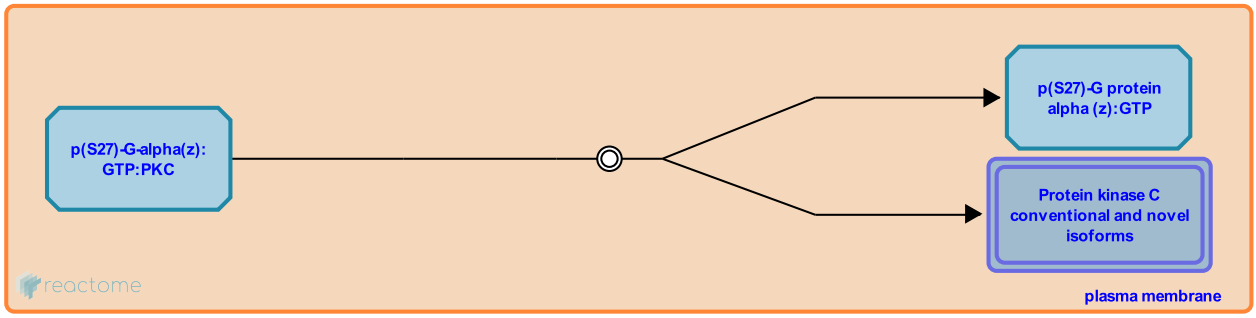
Location: G alpha (z) signalling events

Stable identifier: R-HSA-8982709

Type: dissociation

Compartments: plasma membrane

Inferred from: Gz(rat):PKC(cow) dissociates to give phosphorylated G alpha (z) (Bos taurus)



G alpha z (Lounsbury et al. 1991) and G alpha 12 (Kozasa & Gilman, 1996) are excellent in vitro substrates for all three subtypes of protein kinase C (PKC). Activation of PKC in intact platelets by agents such as thrombin, thromboxane A2 (TXA2) analogues and phorbol esters leads to rapid and near-stoichiometric phosphorylation of G alpha z (Carlson et al. 1989). PKC can bind to G alpha z and facilitate phosphorylation at Ser-27 (Lounsbury et al. 1993). Subsequently, phosphorylated G alpha z dissociates from the complex. This phosphorylation blocks the interaction of G alpha z with Gbeta:gamma suggesting that it is a regulatory mechanism for attenuating signalling by preventing subunit reassociation.

Preceded by: PKC phosphorylates G alpha (z)

Literature references

Lounsbury, KM., Manning, DR., Casey, PJ., Brass, LF. (1991). Phosphorylation of Gz in human platelets. Selectivity and site of modification. *J Biol Chem, 266*, 22051-6. ↗

Casey, PJ., Fields, TA. (1995). Phosphorylation of Gz alpha by protein kinase C blocks interaction with the beta gamma complex. *J. Biol. Chem., 270*, 23119-25. ↗

Editions

2010-05-18	Authored	Jupe, S.
2010-05-22	Reviewed	D'Eustachio, P.
2017-05-11	Edited	Jupe, S.
2017-07-10	Revised	Varusai, TM.

Table of Contents