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PIK3C2A Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP11855b

Specification

PIK3C2A Antibody (C-term) Blocking peptide - Product Information

Primary Accession O00443

PIK3C2A Antibody (C-term) Blocking peptide - Additional Information

Gene ID 5286

Other Names
Phosphatidylinositol 4-phosphate 3-kinase C2 domain-containing subunit alpha, PI3K-C2-alpha, PtdIns-3-kinase C2 subunit alpha, Phosphoinositide 3-kinase-C2-alpha, PIK3C2A

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PIK3C2A Antibody (C-term) Blocking peptide - Protein Information

Name PIK3C2A

Function
Generates phosphatidylinositol 3-phosphate (PtdIns3P) and phosphatidylinositol 3,4-bisphosphate (PtdIns(3,4)P2) that act as second messengers. Has a role in several intracellular trafficking events. Functions in insulin signaling and secretion. Required for translocation of the glucose transporter SLC2A4/GLUT4 to the plasma membrane and glucose uptake in response to insulin-mediated RHOQ activation. Regulates insulin secretion through two different mechanisms: involved in glucose-induced insulin secretion downstream of insulin receptor in a pathway that involves AKT1 activation and TBC1D4/AS160 phosphorylation, and participates in the late step of insulin granule exocytosis probably in insulin granule fusion. Synthesizes PtdIns3P in response to insulin signaling. Functions in clathrin-coated endocytic vesicle formation and distribution. Regulates dynamin-independent endocytosis, probably by recruiting EEA1 to internalizing vesicles. In neurosecretory cells synthesizes PtdIns3P on large dense core vesicles. Participates in calcium induced contraction of vascular smooth muscle by regulating myosin light chain (MLC) phosphorylation through a mechanism involving Rho kinase-dependent phosphorylation of the MLCP-regulatory subunit MYPT1. May play a role in the EGF signaling cascade. May be involved in mitosis and UV-induced damage response. Required for maintenance of normal renal structure and function by supporting normal podocyte function. Involved in the regulation of ciliogenesis and trafficking of ciliary components (PubMed:<a href="http://www.uniprot.org/citations/31034465"

target="_blank">31034465).

Cellular Location
Cell membrane. Cytoplasmic vesicle, clathrin-coated vesicle. Nucleus Cytoplasm Golgi apparatus, trans-Golgi network. Note=Inserts preferentially into membranes containing PtdIns(4,5)P2 (PubMed:17038310). Associated with RNA-containing structures (PubMed:11606566)

Tissue Location
Expressed in columnar and transitional epithelia, mononuclear cells, smooth muscle cells, and endothelial cells lining capillaries and small venules (at protein level). Ubiquitously expressed, with highest levels in heart, placenta and ovary, and lowest levels in the kidney. Detected at low levels in islets of Langerhans from type 2 diabetes mellitus individuals

PIK3C2A Antibody (C-term) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

PIK3C2A Antibody (C-term) Blocking peptide - Images

PIK3C2A Antibody (C-term) Blocking peptide - Background

Serine/threonine protein kinase required for postnatal development, possibly by regulating the homeostasis of cerebral spinal fluid or ciliary function. Controls the activity of the transcriptional regulators GLI1, GLI2 and GLI3 by opposing the effect of SUFU and promoting their nuclear localization. GLI2 requires an additional function of STK36 to become transcriptionally active, but the enzyme does not need to possess an active kinase catalytic site for this to occur.

PIK3C2A Antibody (C-term) Blocking peptide - References

Ewing, R.M., et al. Mol. Syst. Biol. 3, 89 (2007) :Osterlund, T., et al. BMC Genomics 5 (1), 49 (2004) :Nakayama, M., et al. Genome Res. 12(11):1773-1784(2002)Murone, M., et al. Nat. Cell Biol. 2(5):310-312(2000)Gold, M.O., et al. J. Virol. 72(5):4448-4453(1998)