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PI3KCA Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP8016b

Specification

PI3KCA Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P42336

PI3KCA Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 5290

Other Names
Phosphatidylinositol 4, 5-bisphosphate 3-kinase catalytic subunit alpha isoform, PI3-kinase subunit alpha, PI3K-alpha, PI3Kalpha, PtdIns-3-kinase subunit alpha, Phosphatidylinositol 4, 5-bisphosphate 3-kinase 110 kDa catalytic subunit alpha, PtdIns-3-kinase subunit p110-alpha, p110alpha, Phosphoinositide-3-kinase catalytic alpha polypeptide, Serine/threonine protein kinase PIK3CA, PIK3CA

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8016b was selected from the C-term region of human PI3KCA . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

PI3KCA Antibody (C-term) Blocking Peptide - Protein Information

Name PIK3CA

Function
Phosphoinositide-3-kinase (PI3K) phosphorylates phosphatidylinositol (PI) and its phosphorylated derivatives at position 3 of the inositol ring to produce 3-phosphoinositides (PubMed:15135396, PubMed:23936502, PubMed:28676499). Uses ATP and PtdIns(4,5)P2 (phosphatidylinositol 4,5-bisphosphate) to generate phosphatidylinositol 3,4,5-trisphosphate (PIP3) (PubMed:15135396, PubMed:<a href="http://www.uniprot.org/citations/28676499"

target="_blank">28676499). PIP3 plays a key role by recruiting PH domain- containing proteins to the membrane, including AKT1 and PDPK1, activating signaling cascades involved in cell growth, survival, proliferation, motility and morphology. Participates in cellular signaling in response to various growth factors. Involved in the activation of AKT1 upon stimulation by receptor tyrosine kinases ligands such as EGF, insulin, IGF1, VEGFA and PDGF. Involved in signaling via insulin-receptor substrate (IRS) proteins. Essential in endothelial cell migration during vascular development through VEGFA signaling, possibly by regulating RhoA activity. Required for lymphatic vasculature development, possibly by binding to RAS and by activation by EGF and FGF2, but not by PDGF. Regulates invadopodia formation through the PDPK1-AKT1 pathway. Participates in cardiomyogenesis in embryonic stem cells through a AKT1 pathway. Participates in vasculogenesis in embryonic stem cells through PDK1 and protein kinase C pathway. In addition to its lipid kinase activity, it displays a serine-protein kinase activity that results in the autophosphorylation of the p85alpha regulatory subunit as well as phosphorylation of other proteins such as 4EBP1, H-Ras, the IL-3 beta c receptor and possibly others (PubMed:23936502, PubMed:28676499). Plays a role in the positive regulation of phagocytosis and pinocytosis (By similarity).

PI3KCA Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PI3KCA Antibody (C-term) Blocking Peptide - Images

PI3KCA Antibody (C-term) Blocking Peptide - Background

Phosphatidylinositol 3-kinase is composed of an 85 kDa regulatory subunit and a 110 kDa catalytic subunit. This protein represents the catalytic subunit, which uses ATP to phosphorylate PtdIns, PtdIns4P and PtdIns(4,5)P2. PI3KCA has been found to be oncogenic and has been implicated in cervical cancers.

PI3KCA Antibody (C-term) Blocking Peptide - References

Ballou, L.M., et al., J. Biol. Chem. 278(26):23472-23479 (2003).Singh, B., et al., Genes Dev. 16(8):984-993 (2002).Shayesteh, L., et al., Nat. Genet. 21(1):99-102 (1999).Volinia, S., et al., Genomics 24(3):472-477 (1994).Hiles, I.D., et al., Cell 70(3):419-429 (1992).