

PI3KCG Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8021B**Specification**

PI3KCG Antibody (C-term) - Product Information

Application	IHC-P, WB,E
Primary Accession	P48736
Other Accession	O02697 , O9JHG7 , NP_002640
Reactivity	Human
Predicted	Mouse, Pig
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	1074-1102

PI3KCG Antibody (C-term) - Additional Information**Gene ID** 5294**Other Names**

Phosphatidylinositol 4, 5-bisphosphate 3-kinase catalytic subunit gamma isoform, PI3-kinase subunit gamma, PI3K-gamma, PI3Kgamma, PtdIns-3-kinase subunit gamma, Phosphatidylinositol 4, 5-bisphosphate 3-kinase 110 kDa catalytic subunit gamma, PtdIns-3-kinase subunit p110-gamma, p110gamma, Phosphoinositide-3-kinase catalytic gamma polypeptide, Serine/threonine protein kinase PIK3CG, p120-PI3K, PIK3CG

Target/Specificity

This PI3KCG antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1074-1102 amino acids from the C-terminal region of human PI3KCG.

Dilution

IHC-P~~1:50~100

WB~~1:1000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

PI3KCG Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

PI3KCG Antibody (C-term) - Protein Information

Name PIK3CG

Function Phosphoinositide-3-kinase (PI3K) that phosphorylates PtdIns(4,5)P₂ (Phosphatidylinositol 4,5-bisphosphate) to generate phosphatidylinositol 3,4,5-trisphosphate (PIP₃). PIP₃ plays a key role by recruiting PH domain-containing proteins to the membrane, including AKT1 and PDK1, activating signaling cascades involved in cell growth, survival, proliferation, motility and morphology. Links G-protein coupled receptor activation to PIP₃ production. Involved in immune, inflammatory and allergic responses. Modulates leukocyte chemotaxis to inflammatory sites and in response to chemoattractant agents. May control leukocyte polarization and migration by regulating the spatial accumulation of PIP₃ and by regulating the organization of F-actin formation and integrin-based adhesion at the leading edge. Controls motility of dendritic cells. Together with PIK3CD is involved in natural killer (NK) cell development and migration towards the sites of inflammation. Participates in T-lymphocyte migration. Regulates T- lymphocyte proliferation, activation, and cytokine production. Together with PIK3CD participates in T-lymphocyte development. Required for B- lymphocyte development and signaling. Together with PIK3CD participates in neutrophil respiratory burst. Together with PIK3CD is involved in neutrophil chemotaxis and extravasation. Together with PIK3CB promotes platelet aggregation and thrombosis. Regulates alpha-IIb/beta-3 integrins (ITGA2B/ ITGB3) adhesive function in platelets downstream of P2Y₁₂ through a lipid kinase activity-independent mechanism. May have also a lipid kinase activity-dependent function in platelet aggregation. Involved in endothelial progenitor cell migration. Negative regulator of cardiac contractility. Modulates cardiac contractility by anchoring protein kinase A (PKA) and PDE3B activation, reducing cAMP levels. Regulates cardiac contractility also by promoting beta-adrenergic receptor internalization by binding to GRK2 and by non- muscle tropomyosin phosphorylation. Also has serine/threonine protein kinase activity: both lipid and protein kinase activities are required for beta-adrenergic receptor endocytosis. May also have a scaffolding role in modulating cardiac contractility. Contributes to cardiac hypertrophy under pathological stress. Through simultaneous binding of PDE3B to RAPGEF3 and PIK3R6 is assembled in a signaling complex in which the PI3K gamma complex is activated by RAPGEF3 and which is involved in angiogenesis. In neutrophils, participates in a phospholipase C-activating N-formyl peptide-activated GPCR (G protein- coupled receptor) signaling pathway downstream of RASGRP4-mediated Ras- activation, to promote neutrophil functional responses (By similarity).

Cellular Location

Cytoplasm. Cell membrane

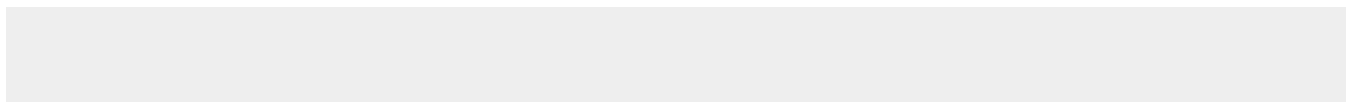
Tissue Location

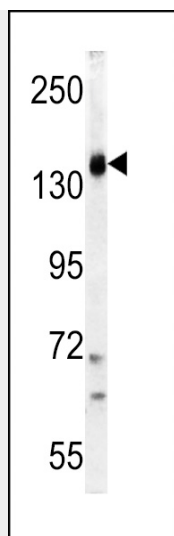
Pancreas, skeletal muscle, liver and heart.

PI3KCG Antibody (C-term) - Protocols

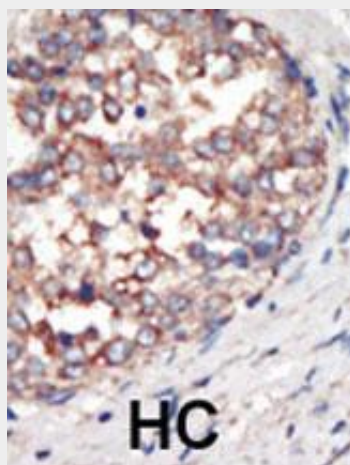
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

PI3KCG Antibody (C-term) - Images



Western blot analysis of PI3KCG Antibody (C-term) (Cat.# AP8021b) in K562 cell line lysates (35ug/lane). PI3KCG (arrow) was detected using the purified Pab.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

PI3KCG Antibody (C-term) - Background

PI3KCG belongs to the pi3/pi4-kinase family of proteins. This protein is an enzyme that phosphorylates phosphoinositides on the 3-hydroxyl group of the inositol ring. It is an important modulator of extracellular signals, including those elicited by E-cadherin-mediated cell-cell adhesion, which plays an important role in maintenance of the structural and functional integrity of epithelia. In addition to its role in promoting assembly of adherens junctions, the protein is thought to play a pivotal role in the regulation of cytotoxicity in NK cells.

PI3KCG Antibody (C-term) - References

- Resendiz, J.C., et al., Mol. Pharmacol. 63(3):639-645 (2003).
- Francois, F., et al., J. Virol. 77(4):2539-2549 (2003).
- Chandrasekar, N., et al., Oncogene 22(3):392-400 (2003).
- Ishibashi, Y., et al., Cell. Microbiol. 4(12):825-833 (2002).
- Semba, S., et al., Clin. Cancer Res. 8(12):3824-3831 (2002).