

PIP5K2G Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP8043a

Specification

PIP5K2G Antibody (C-term) - Product Information

Application WB, IHC-P,E
Primary Accession Other Accession O88370

Reactivity Human, Mouse

Predicted Rat
Host Rabbit
Clonality Polyclonal
Isotype Rabbit Ig
Antigen Region 333-364

PIP5K2G Antibody (C-term) - Additional Information

Gene ID 79837

Other Names

Phosphatidylinositol 5-phosphate 4-kinase type-2 gamma, Phosphatidylinositol 5-phosphate 4-kinase type II gamma, PI(5)P 4-kinase type II gamma, PIP4KII-gamma, PIP4K2C, PIP5K2C

Target/Specificity

This PIP5K2G antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 333-364 amino acids from the C-terminal region of human PIP5K2G.

Dilution

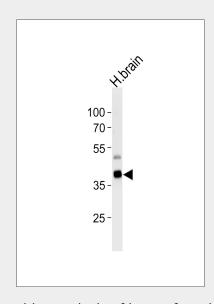
WB~~1:1000 IHC-P~~1:50~100

Format

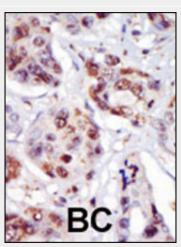
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

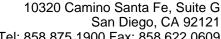
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.



Western blot analysis of lysate from human brain tissue lysate, using PIP5K2G Antibody(E348)(Cat. #AP8043a). AP8043a was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody. Lysate at 35ug per lane.



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



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Precautions

PIP5K2G Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

PIP5K2G Antibody (C-term) - Protein Information

Name PIP4K2C (HGNC:23786)

Synonyms PIP5K2C

Function

Phosphatidylinositol 5-phosphate 4-kinase with low enzymatic activity. May be a GTP sensor, has higher GTP-dependent kinase activity than ATP-dependent kinase activity. PIP4Ks negatively regulate insulin signaling through a catalytic-independent mechanism. They interact with PIP5Ks and suppress PIP5K-mediated PtdIns(4,5)P2 synthesis and insulin- dependent conversion to PtdIns(3,4,5)P3 (PubMed:<a h ref="http://www.uniprot.org/citations/31091 439" target=" blank">31091439).

Cellular Location

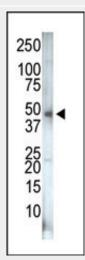
Endoplasmic reticulum {ECO:0000250|UniProtKB:088370}. Cytoplasm {ECO:0000250|UniProtKB:088370}

PIP5K2G 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
- <u>Immunoprecipitation</u>
- Flow Cvtometv
- Cell Culture

immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.



Western blot analysis of anti-PIP5K2G Pab (Cat. #AP8043a) in mouse brain cell lysate. PIP5K2G (arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.

PIP5K2G Antibody (C-term) - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the g phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains.

PIP5K2G Antibody (C-term) - References

Blume-Jensen P, et al. Nature 2001. 411: 355. Cantrell D, J. Cell Sci. 2001. 114: 1439. Jhiang S Oncogene 2000. 19: 5590. Manning G, et al. Science 2002. 298: 1912. Moller, D, et al. Am. J. Physiol. 1994. 266: C351-C359. Robertson, S. et al. Trends Genet. 2000. 16:





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Robinson D, et al. Oncogene 2000. 19: 5548. Van der Ven, P, et al. Hum. Molec. Genet. 1993. 2: 1889.

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