

**Phospho-PIK3CG(S1100) Antibody**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP3464a**

**Specification**

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**Phospho-PIK3CG(S1100) Antibody - Product Information**

Application	DB,E
Primary Accession	<a href="#">P48736</a>
Other Accession	<a href="#">O02697</a> , <a href="#">O9JHG7</a>
Reactivity	Human
Predicted	Mouse, Pig
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	126454

**Phospho-PIK3CG(S1100) Antibody - 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 PIK3CG Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S1100 of human PIK3CG.

**Dilution**

DB~~1:500

E~~Use at an assay dependent concentration.

**Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**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**

Phospho-PIK3CG(S1100) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Phospho-PIK3CG(S1100) Antibody - Protein Information**

**Name** PIK3CG

**Function** Phosphoinositide-3-kinase (PI3K) that phosphorylates PtdIns(4,5)P<sub>2</sub> (Phosphatidylinositol 4,5-bisphosphate) to generate phosphatidylinositol 3,4,5-trisphosphate (PIP<sub>3</sub>). PIP<sub>3</sub> 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 PIP<sub>3</sub> 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 PIP<sub>3</sub> 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 P2Y<sub>12</sub> 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

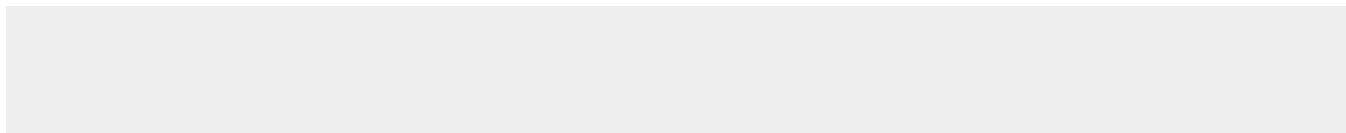
**Tissue Location**

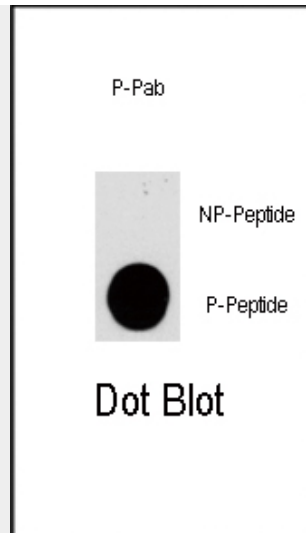
Pancreas, skeletal muscle, liver and heart.

**Phospho-PIK3CG(S1100) Antibody - 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](#)

**Phospho-PIK3CG(S1100) Antibody - Images**



Dot blot analysis of anti-PIK3CG-pS1100 Phospho-specific Pab (RB13320) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

### **Phospho-PIK3CG(S1100) Antibody - Background**

PIK3CG belongs to the pi3/pi4-kinase family of proteins. It 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. The gene for this protein is located in a commonly deleted segment of chromosome 7 previously identified in myeloid leukemias.

### **Phospho-PIK3CG(S1100) Antibody - References**

- Andreozzi, F., et al., Endocrinology 145(6):2845-2857 (2004).
- Reddy, S.A., et al., Biochem. Biophys. Res. Commun. 316(4):1022-1028 (2004).
- Osaki, M., et al., J. Cancer Res. Clin. Oncol. 130(1):8-14 (2004).
- Khan, N.A., et al., J. Neurovirol. 9(6):584-593 (2003).
- Theberge, J.F., et al., Arch. Biochem. Biophys. 420(1):9-17 (2003).