

# cGKI (cGKI beta) Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP8000a

## **Product Information**

IHC-P, WB, E Q13976 P00516, P14619 Human, Rat, Mouse Bovine Rabbit Polyclonal Rabbit IgG
Rabbit IgG
76364
629-660

### **Additional Information**

Gene ID	5592
Other Names	cGMP-dependent protein kinase 1, cGK 1, cGK1, cGMP-dependent protein kinase I, cGKI, PRKG1, PRKG1B, PRKGR1A, PRKGR1B
Target/Specificity	This cGKI (cGKI beta) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 629-660 amino acids from the C-terminal region of human cGKI beta.
Dilution	IHC-P~~1:100~500 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 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	cGKI (cGKI beta) Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

#### **Protein Information**

Name	PRKG1
Synonyms	PRKG1B, PRKGR1A, PRKGR1B
Function	Serine/threonine protein kinase that acts as a key mediator of the nitric

	oxide (NO)/cGMP signaling pathway. GMP binding activates PRKG1, which phosphorylates serines and threonines on many cellular proteins. Numerous protein targets for PRKG1 phosphorylation are implicated in modulating cellular calcium, but the contribution of each of these targets may vary substantially among cell types. Proteins that are phosphorylated by PRKG1 regulate platelet activation and adhesion, smooth muscle contraction, cardiac function, gene expression, feedback of the NO-signaling pathway, and other processes involved in several aspects of the CNS like axon guidance, hippocampal and cerebellar learning, circadian rhythm and nociception. Smooth muscle relaxation is mediated through lowering of intracellular free calcium, by desensitization of contractile proteins to calcium, and by decrease in the contractile state of smooth muscle or in platelet activation. Regulates intracellular calcium levels via several pathways: phosphorylates IRAG1 and inhibits IP3-induced Ca(2+) release from intracellular stores, phosphorylation of KCNMA1 (BKCa) channels decreases intracellular ca(2+) levels, which leads to increased opening of this channel. PRKG1 phosphorylates the canonical transient receptor potential channel (TRPC) family which inactivates the associated inward calcium current. Another mode of action of NO/cGMP/PKGI signaling involves PKG1-mediated inactivation of the Ras homolog gene family member A (RhoA). Phosphorylation resulting in vasorelaxation. Activation of myosin light chain phosphorylation resulting in vasorelaxation. Activation of PRKG1 by NO signaling also alters gene expression in a number of tissues. In smooth muscle cells, increased cGMP and PRKG1 activity influence expression of smooth muscle-specific contractile proteins, levels of proteins in the NO/cGMP signaling pathway, down- regulation of the matrix proteins osteopontin and thrombospondin-1 to limit smooth muscle cell migration and phenotype. Regulates vasodilator-stimulated phosphorytein (VASP) functions in platelets and smooth muscl
Cellular Location	Cytoplasm. Note=Colocalized with TRPC7 in the plasma membrane.
Tissue Location	Primarily expressed in lung and placenta.

### 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.

#### References

Orstavik, S., et al., Genomics 42(2):311-318 (1997). Tamura, N., et al., Hypertension 27 (3 Pt 2), 552-557 (1996).

#### Images

Immunohistochemical analysis of AP8000A on paraffin-embedded Human hepato carcinoma tissue. Tissue was fixed with formaldehyde at room temperature. Heat induced epitope retrieval was



performed by EDTA buffer (pH9. 0). Samples were incubated with primary antibody(1:100) for 1 hour at room temperature. Undiluted CRF Anti-Polyvalent HRP Polymer antibody was used as the secondary antibody.



Immunohistochemical analysis of AP8000A on paraffin-embedded Human placenta tissue. Tissue was fixed with formaldehyde at room temperature. Heat induced epitope retrieval was performed by EDTA buffer (pH9. 0). Samples were incubated with primary antibody(1:100) for 1 hour at room temperature. Undiluted CRF Anti-Polyvalent HRP Polymer antibody was used as the secondary antibody.



All lanes : Anti-cGKI (cGKI beta) Antibody (C-term) at 1:1000 dilution Lane 1: 293 whole cell lysate Lane 2: U-87 MG whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 76 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



All lanes : Anti-cGKI (cGKI beta) Antibody (C-term) at 1:2000 dilution Lane 1: 293 whole cell lysate Lane 2: U-87 MG whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 76 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

## Citations

• <u>A molecular mechanism for therapeutic effects of cGMP-elevating agents in pulmonary arterial hypertension.</u>

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.