

## Product datasheet

### Recombinant human cGKI protein ab96403

[2 Images](#)

#### Description

<b>Product name</b>	Recombinant human cGKI protein
<b>Biological activity</b>	The specific activity of ab96403 was determined to be 780 nmol/min/mg. The specific activity was determined to be 780 nmol/min/mg.
<b>Purity</b>	> 95 % Densitometry. Affinity purified.
<b>Expression system</b>	Baculovirus infected insect cells
<b>Accession</b>	<b><u>Q13976</u></b>
<b>Protein length</b>	Full length protein
<b>Animal free</b>	No
<b>Nature</b>	Recombinant
<b>Species</b>	Human
<b>Predicted molecular weight</b>	100 kDa including tags
<b>Amino acids</b>	2 to 686

#### Specifications

Our **Abpromise guarantee** covers the use of **ab96403** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

<b>Applications</b>	Functional Studies SDS-PAGE
<b>Form</b>	Liquid

#### Preparation and Storage

<b>Stability and Storage</b>	Shipped on dry ice. Upon delivery aliquot and store at -80°C. Avoid freeze / thaw cycles. pH: 7.50 Constituents: 0.307% Glutathione, 0.00174% PMSF, 0.00385% DTT, 0.79% Tris HCl, 0.00292% EDTA, 25% Glycerol (glycerin, glycerine), 0.87% Sodium chloride This product is an active protein and may elicit a biological response in vivo, handle with caution.
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## General Info

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### Function

Serine/threonine protein kinase that acts as 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 MRV11/IRAG 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/PKG1 signaling involves PKG1-mediated inactivation of the Ras homolog gene family member A (RhoA). Phosphorylation of RHOA by PRKG1 blocks the action of this protein in myriad processes: regulation of RHOA translocation; decreasing contraction; controlling vesicle trafficking, reduction of myosin light chain phosphorylation resulting in vasorelaxation. Activation of PRKG1 by NO signaling alters also 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 phosphoprotein (VASP) functions in platelets and smooth muscle.

### Tissue specificity

Primarily expressed in lung and placenta.

### Sequence similarities

Belongs to the protein kinase superfamily. AGC Ser/Thr protein kinase family. cGMP subfamily. Contains 1 AGC-kinase C-terminal domain. Contains 2 cyclic nucleotide-binding domains. Contains 1 protein kinase domain.

### Domain

Composed of an N-terminal leucine-zipper domain followed by an autoinhibitory domain, which mediate homodimer formation and inhibit kinase activity, respectively. Next, two cGMP-binding domains are followed by the catalytic domain at the C-terminus. Binding of cGMP to cGMP-binding domains results in a conformational change that activates kinase activity by removing the autoinhibitory domain from the catalytic cleft leaving the catalytic domain free to phosphorylate downstream substrates. Isoforms alpha and beta have identical cGMP-binding and catalytic domains but differ in their leucine zipper and autoinhibitory sequences and therefore differ in their dimerization substrates and kinase enzyme activity. Heterotetramerization is mediated by the interaction between a coiled-coil of PRKG1 and the leucine/isoleucine zipper of PPP1R12A/MBS, the myosin-binding subunit of the myosin phosphatase.

### Post-translational modifications

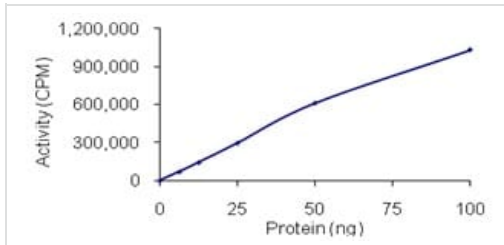
Autophosphorylation increases kinase activity.  
65 kDa monomer is produced by proteolytic cleavage.

### Cellular localization

Cytoplasm. Colocalized with TRPC7 in the plasma membrane.

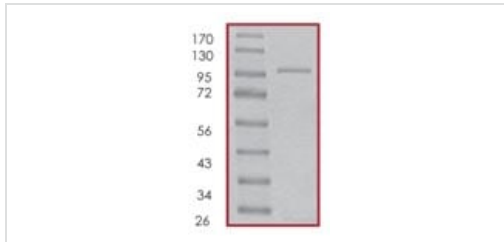
## Images

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Kinase Assay demonstrating specific activity of ab96403.

Functional Studies - Recombinant human cGKI protein (ab96403)



SDS-PAGE showing ab96403 at approximately 100kDa.

SDS-PAGE - Recombinant human cGKI protein (ab96403)

**Please note:** All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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