abcam

Product datasheet

Anti-cGKI antibody - Carboxyterminal end ab37709

1 References 2 Images

Overview

Product name	Anti-cGKI antibody - Carboxyterminal end
Description	Rabbit polyclonal to cGKI - Carboxyterminal end
Tested applications	**Suitable for:** ELISA, WB, IHC-P, ICC/IF
Species reactivity	**Reacts with:** Mouse, Human
Immunogen	Synthetic peptide conjugated to KLH, located within C terminal amino acids 600-670 of Human cGKI
Positive control	293 cell lysate. Mouse small intestine tissue lysate.

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer	Preservative: 0.09% Sodium Azide Constituents: PBS
Purity	Protein G purified
Clonality	Polyclonal
Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab37709** in the following tested applications.

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

Application	Abreviews	Notes
ELISA		1/1000.
WB		1/100 - 1/500. Predicted molecular weight: 76 kDa.
IHC-P		1/50 - 1/100.

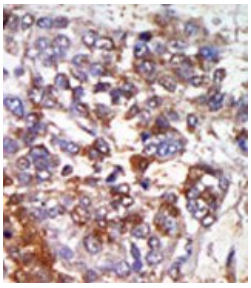
Application	Abreviews	Notes
ICC/IF		1/50. PubMed: 22158710

Target

Function	Serine/threonine protein kinasethat 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. Smoth 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 MRVI1/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/PKGI signaling involves PKGI-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	Autophosphorylation increases kinase activity.

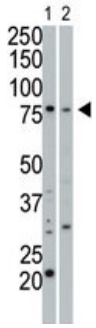
modifications	65 kDa monomer is produced by proteolytic cleavage.
Cellular localization	Cytoplasm. Colocalized with TRPC7 in the plasma membrane.

Images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - PKG antibody - Carboxyterminal end (ab37709)

Immunohistochemical detection of PKG expression in formalin-fixed and paraffin-embedded human hepatocarcinoma tissue using 1/50 ab37709.



Western blot - PKG antibody - Carboxyterminal end (ab37709)

Lane 1 : Anti-cGKI antibody - Carboxyterminal end (ab37709) at 1/100 dilution
Lane 2 : Anti-cGKI antibody - Carboxyterminal end (ab37709) at 1/1000 dilution

Lane 1 : 293 cell lysate
Lane 2 : mouse small intestine tissue lysate

Predicted band size : 76 kDa
Observed band size : 76 kDa
Additional bands at : 20 kDa,30 kDa. We are unsure as to the identity of these extra bands.

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