abcam

Product datasheet

Anti-cGKI antibody ab111930

1 Abreviews

Overview

Product name	Anti-cGKI antibody
Description	Rabbit polyclonal to cGKI
Host species	Rabbit
Tested applications	**Suitable for:** WB, ICC
Species reactivity	**Reacts with:** Mouse **Predicted to work with:** Rat, Horse, Human, Pig
Immunogen	Synthetic peptide: LIKEAILDNDFMKNL , corresponding to amino acids 93-107 of Cow cGKI (P00516) Run BLAST with Run BLAST with

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	Preservative: 0.05% Sodium azide
Purity	Whole antiserum
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab111930 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
WB	(1)	1/400. Predicted molecular weight: 76 kDa.
ICC		1/200.

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

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