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# PKG / PRKG1 Antibody (clone 2B3)

**Mouse Monoclonal Antibody**
**Catalog # ALS13378**

## Specification

### PKG / PRKG1 Antibody (clone 2B3) - Product Information

| | |
|---|---|
| Application | **IHC** |
| Primary Accession | Q13976 |
| Reactivity | **Human** |
| Host | **Mouse** |
| Clonality | **Monoclonal** |
| Calculated MW | **76kDa KDa** |

### PKG / PRKG1 Antibody (clone 2B3) - Additional Information

**Gene ID** 5592

**Other Names**
cGMP-dependent protein kinase 1, cGK 1, cGK1, 2.7.11.12, cGMP-dependent protein kinase I, cGKI, PRKG1, PRKG1B, PRKGR1A, PRKGR1B

**Reconstitution & Storage**
Store at -20°C. Aliquot to avoid freeze/thaw cycles.

**Precautions**
PKG / PRKG1 Antibody (clone 2B3) is for research use only and not for use in diagnostic or therapeutic procedures.

### PKG / PRKG1 Antibody (clone 2B3) - 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 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.

**Cellular Location**
Cytoplasm. Note=Colocalized with TRPC7 in the plasma membrane.

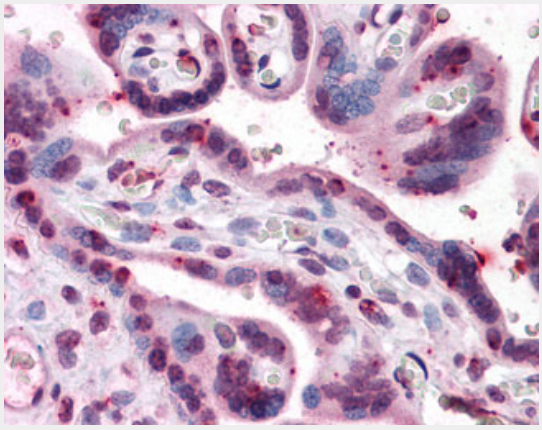
**Tissue Location**
Primarily expressed in lung and placenta.

## PKG / PRKG1 Antibody (clone 2B3) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

## PKG / PRKG1 Antibody (clone 2B3) - Images



Anti-PRKG1 / CGKI antibody IHC of human placenta.

## PKG / PRKG1 Antibody (clone 2B3) - Background

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 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.

**PKG / PRKG1 Antibody (clone 2B3) - References**

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Tamura N.,et al.Hypertension 27:552-557(1996).
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