

PKD2 Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP7934a**Specification**

PKD2 Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [Q9BZL6](#)**PKD2 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 25865**Other Names**

Serine/threonine-protein kinase D2, nPKC-D2, PRKD2, PKD2

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP7934a](/product/products/AP7934a) was selected from the C-term region of human PKD2 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PKD2 Antibody (C-term) Blocking Peptide - Protein Information**Name** PRKD2**Synonyms** PKD2**Function**

Serine/threonine-protein kinase that converts transient diacylglycerol (DAG) signals into prolonged physiological effects downstream of PKC, and is involved in the regulation of cell proliferation via MAPK1/3 (ERK1/2) signaling, oxidative stress-induced NF-kappa-B activation, inhibition of HDAC7 transcriptional repression, signaling downstream of T-cell antigen receptor (TCR) and cytokine production, and plays a role in Golgi membrane trafficking, angiogenesis, secretory granule release and cell adhesion (PubMed: [14743217](http://www.uniprot.org/citations/14743217), PubMed: [15604256](http://www.uniprot.org/citations/15604256), PubMed: [16928771](http://www.uniprot.org/citations/16928771), PubMed: [17077180](http://www.uniprot.org/citations/17077180), PubMed: [17951978](http://www.uniprot.org/citations/17951978))

target="_blank">17951978, PubMed:17962809, PubMed:18262756, PubMed:19001381, PubMed:19192391, PubMed:23503467, PubMed:28428613). May potentiate mitogenesis induced by the neuropeptide bombesin by mediating an increase in the duration of MAPK1/3 (ERK1/2) signaling, which leads to accumulation of immediate-early gene products including FOS that stimulate cell cycle progression (By similarity). In response to oxidative stress, is phosphorylated at Tyr-438 and Tyr-717 by ABL1, which leads to the activation of PRKD2 without increasing its catalytic activity, and mediates activation of NF-kappa-B (PubMed:15604256, PubMed:28428613). In response to the activation of the gastrin receptor CCKBR, is phosphorylated at Ser-244 by CSNK1D and CSNK1E, translocates to the nucleus, phosphorylates HDAC7, leading to nuclear export of HDAC7 and inhibition of HDAC7 transcriptional repression of NR4A1/NUR77 (PubMed:17962809). Upon TCR stimulation, is activated independently of ZAP70, translocates from the cytoplasm to the nucleus and is required for interleukin-2 (IL2) promoter up-regulation (PubMed:17077180). During adaptive immune responses, is required in peripheral T-lymphocytes for the production of the effector cytokines IL2 and IFNG after TCR engagement and for optimal induction of antibody responses to antigens (By similarity). In epithelial cells stimulated with lysophosphatidic acid (LPA), is activated through a PKC-dependent pathway and mediates LPA-stimulated interleukin-8 (IL8) secretion via a NF-kappa-B-dependent pathway (PubMed:16928771). During TCR-induced T- cell activation, interacts with and is activated by the tyrosine kinase LCK, which results in the activation of the NFAT transcription factors (PubMed:19192391). In the trans-Golgi network (TGN), regulates the fission of transport vesicles that are on their way to the plasma membrane and in polarized cells is involved in the transport of proteins from the TGN to the basolateral membrane (PubMed:14743217). Plays an important role in endothelial cell proliferation and migration prior to angiogenesis, partly through modulation of the expression of KDR/VEGFR2 and FGFR1, two key growth factor receptors involved in angiogenesis (PubMed:19001381). In secretory pathway, is required for the release of chromogranin-A (CHGA)-containing secretory granules from the TGN (PubMed:18262756). Downstream of PRKCA, plays important roles in angiotensin-2-induced monocyte adhesion to endothelial cells (PubMed:17951978). Plays a regulatory role in angiogenesis and tumor growth by phosphorylating a downstream mediator CIB1 isoform 2, resulting in vascular endothelial growth factor A (VEGFA) secretion (PubMed:23503467).

Cellular Location

Cytoplasm. Cell membrane {ECO:0000250|UniProtKB:Q15139}. Nucleus. Golgi apparatus, trans-Golgi network. Note=Translocation to the cell membrane is required for kinase activation. Accumulates in the nucleus upon CK1- mediated phosphorylation after activation of G-protein-coupled receptors. Nuclear accumulation is regulated by blocking nuclear export of active PRKD2 rather than by increasing import

Tissue Location

Widely expressed..

PKD2 Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

PKD2 Antibody (C-term) Blocking Peptide - Images

PKD2 Antibody (C-term) Blocking Peptide - Background

PKD2, a member of the polycystin family, functions as a calcium permeable cation channel. PKD1 and PKD2 may function through a common signaling pathway that is necessary for normal tubulogenesis. PKD2 interacts with PKD1, potentially through the C-terminal region. PKD1 requires the presence of PKD2 for stable expression. PKD2 also interacts with CD2AP. This protein is strongly expressed in ovary, fetal and adult kidney, testis, and small intestine, but is not detected in peripheral leukocytes. Defects in PKD2 are the cause of autosomal dominant polycystic kidney disease type II (ADPKD-2) which represent approximately 15% of cases of autosomal dominant polycystic kidney disease, a common autosomal dominant genetic disease affecting about 1 out of 1000 individuals. ADPKD is characterized by progressive formation and enlargement of cysts in both kidneys, typically leading to end-stage renal disease in adult life. Cysts also occur in the liver and other organs. All mutations, scattered between exons 1 and 11, result in a truncated PKD2 that lacks both the calcium-binding EF-hand domain and the two cytoplasmic domains required for the interaction of PKD2 with PKD1 and with itself. ADPKD type II is clinically milder than ADPKD type I, but it has a deleterious impact on overall life expectancy.

PKD2 Antibody (C-term) Blocking Peptide - References

Stayner, C., et al., Trends Pharmacol. Sci. 22(11):543-546 (2001). Lehtonen, S., et al., J. Biol. Chem. 275(42):32888-32893 (2000). Watnick, T., et al., Nat. Genet. 25(2):143-144 (2000). Torra, R., et al., Kidney Int. 56(1):28-33 (1999). Hayashi, T., et al., Genomics 44(1):131-136 (1997).