

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP7047a**Specification**

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Product InformationPrimary Accession [P22694](#)**PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Additional Information****Gene ID** 5567**Other Names**

cAMP-dependent protein kinase catalytic subunit beta, PKA C-beta, PRKACB

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP7047a](/product/products/AP7047a) was selected from the N-term region of human PRKACB. 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.

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Protein Information**Name** PRKACB**Function**

Mediates cAMP-dependent signaling triggered by receptor binding to GPCRs (PubMed: [12420224](http://www.uniprot.org/citations/12420224), PubMed: [21423175](http://www.uniprot.org/citations/21423175), PubMed: [31112131](http://www.uniprot.org/citations/31112131)). PKA activation regulates diverse cellular processes such as cell proliferation, the cell cycle, differentiation and regulation of microtubule dynamics, chromatin condensation and decondensation, nuclear envelope disassembly and reassembly, as well as regulation of intracellular transport mechanisms and ion flux (PubMed: [12420224](http://www.uniprot.org/citations/12420224), PubMed: [21423175](http://www.uniprot.org/citations/21423175)). Regulates the abundance of compartmentalized pools of its regulatory subunits through phosphorylation of PJA2 which binds and ubiquitinates these subunits, leading to their subsequent proteolysis

(PubMed:12420224, PubMed:21423175). Phosphorylates GPKOW which regulates its ability to bind RNA (PubMed:21880142). Acts as a negative regulator of mTORC1 by mediating phosphorylation of RPTOR (PubMed:31112131).

Cellular Location

Cytoplasm. Cell membrane. Membrane; Lipid- anchor. Nucleus {ECO:0000250|UniProtKB:P05131} Note=Translocates into the nucleus (monomeric catalytic subunit). The inactive holoenzyme is found in the cytoplasm {ECO:0000250|UniProtKB:P05131}

Tissue Location

Isoform 1 is most abundant in the brain, with low level expression in kidney. Isoform 2 is predominantly expressed in thymus, spleen and kidney. Isoform 3 and isoform 4 are only expressed in the brain.

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Images**PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - Background**

cAMP is a signaling molecule important for a variety of cellular functions. cAMP exerts its effects by activating the cAMP-dependent protein kinase (AMPK), which transduces the signal through phosphorylation of different target proteins. The inactive holoenzyme of AMPK is a tetramer composed of two regulatory and two catalytic subunits. cAMP causes the dissociation of the inactive holoenzyme into a dimer of regulatory subunits bound to four cAMP and two free monomeric catalytic subunits. Four different regulatory subunits and three catalytic subunits of AMPK have been identified in humans. PRKACB is a member of the Ser/Thr protein kinase family and is a catalytic subunit of AMPK.

PKA C-beta (PRKACB) Antibody (N-term) Blocking peptide - References

Dwivedi, Y., et al., Biol. Psychiatry 55(3):234-243 (2004). Cartier, C., et al., J. Biol. Chem. 278(37):35211-35219 (2003). Higuchi, H., et al., EMBO J. 22(8):1790-1800 (2003). Wu, K.J., et al., Oncogene 21(51):7872-7882 (2002). Jiang, C.H., et al., Proc. Natl. Acad. Sci. U.S.A. 98(4):1930-1934 (2001).