

**PKC beta1/2 Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP7016a****Specification**

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**PKC beta1/2 Antibody (Center) Blocking peptide - Product Information**Primary Accession [P05771](#)**PKC beta1/2 Antibody (Center) Blocking peptide - Additional Information****Gene ID** 5579**Other Names**

Protein kinase C beta type, PKC-B, PKC-beta, PRKCB, PKCB, PRKCB1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7016a](/product/products/AP7016a) was selected from the C-term region of human PKC beta1/2 . 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.

**PKC beta1/2 Antibody (Center) Blocking peptide - Protein Information****Name** PRKCB**Synonyms** PKCB, PRKCB1**Function**

Calcium-activated, phospholipid- and diacylglycerol (DAG)- dependent serine/threonine-protein kinase involved in various cellular processes such as regulation of the B-cell receptor (BCR) signalosome, oxidative stress-induced apoptosis, androgen receptor-dependent transcription regulation, insulin signaling and endothelial cells proliferation. Plays a key role in B-cell activation by regulating BCR- induced NF-kappa-B activation. Mediates the activation of the canonical NF-kappa-B pathway (NFKB1) by direct phosphorylation of CARD11/CARMA1 at 'Ser-559', 'Ser-644' and 'Ser-652'. Phosphorylation induces CARD11/CARMA1 association with lipid rafts and recruitment of the BCL10-MALT1 complex as well as MAP3K7/TAK1, which then activates IKK complex, resulting in nuclear translocation and activation of NFKB1. Plays a direct role in the negative feedback regulation of the BCR signaling, by down-modulating BTK function via direct

phosphorylation of BTK at 'Ser-180', which results in the alteration of BTK plasma membrane localization and in turn inhibition of BTK activity (PubMed:<a href="http://www.uniprot.org/citations/11598012" target="\_blank">11598012</a>). Involved in apoptosis following oxidative damage: in case of oxidative conditions, specifically phosphorylates 'Ser-36' of isoform p66Shc of SHC1, leading to mitochondrial accumulation of p66Shc, where p66Shc acts as a reactive oxygen species producer. Acts as a coactivator of androgen receptor (AR)-dependent transcription, by being recruited to AR target genes and specifically mediating phosphorylation of 'Thr-6' of histone H3 (H3T6ph), a specific tag for epigenetic transcriptional activation that prevents demethylation of histone H3 'Lys-4' (H3K4me) by LSD1/KDM1A (PubMed:<a href="http://www.uniprot.org/citations/20228790" target="\_blank">20228790</a>). In insulin signaling, may function downstream of IRS1 in muscle cells and mediate insulin-dependent DNA synthesis through the RAF1-MAPK/ERK signaling cascade. Participates in the regulation of glucose transport in adipocytes by negatively modulating the insulin-stimulated translocation of the glucose transporter SLC2A4/GLUT4. Phosphorylates SLC2A1/GLUT1, promoting glucose uptake by SLC2A1/GLUT1 (PubMed:<a href="http://www.uniprot.org/citations/25982116" target="\_blank">25982116</a>). Under high glucose in pancreatic beta-cells, is probably involved in the inhibition of the insulin gene transcription, via regulation of MYC expression. In endothelial cells, activation of PRKCB induces increased phosphorylation of RB1, increased VEGFA-induced cell proliferation, and inhibits PI3K/AKT-dependent nitric oxide synthase (NOS3/eNOS) regulation by insulin, which causes endothelial dysfunction. Also involved in triglyceride homeostasis (By similarity). Phosphorylates ATF2 which promotes cooperation between ATF2 and JUN, activating transcription (PubMed:<a href="http://www.uniprot.org/citations/19176525" target="\_blank">19176525</a>). Phosphorylates KLHL3 in response to angiotensin II signaling, decreasing the interaction between KLHL3 and WNK4 (PubMed:<a href="http://www.uniprot.org/citations/25313067" target="\_blank">25313067</a>).

#### Cellular Location

Cytoplasm. Nucleus. Membrane; Peripheral membrane protein

### PKC beta1/2 Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

### PKC beta1/2 Antibody (Center) Blocking peptide - Images

### PKC beta1/2 Antibody (Center) Blocking peptide - Background

Protein kinase C (PKC) is a family of serine- and threonine-specific protein kinases that can be activated by calcium and second messenger diacylglycerol. PKC family members phosphorylate a wide variety of protein targets and are known to be involved in diverse cellular signaling pathways. PKC family members also serve as major receptors for phorbol esters, a class of tumor promoters. Each member of the PKC family has a specific expression profile and is believed to play a distinct role in cells. PKC beta is one of the PKC family members. This protein kinase has been reported to be involved in many different cellular functions, such as B cell activation, apoptosis induction, endothelial cell proliferation, and intestinal sugar absorption. Studies in mice also suggest that this kinase may also regulate neuronal functions and correlate fear-induced conflict behavior after stress. Alternatively spliced transcript variants encoding distinct isoforms have been reported. Isoform 1 uses an alternate exon at the 3' end compared to isoform 2, which includes a part of the coding region. The resulting isoform 1 has a distinct and shorter C-terminus, as compared to isoform 2.

### PKC beta1/2 Antibody (Center) Blocking peptide - References

Chen, D., et al., J. Biol. Chem. 279(6):4829-4839 (2004).Hug, B.A., et al., J. Biol. Chem. 279(2):825-830 (2004).Wu, H., et al., Biochem. Biophys. Res. Commun. 311(4):948-953 (2003).Becker, K.P., et al., J. Biol. Chem. 278(52):52747-52754 (2003).Birikh, K.R., et al., Proc. Natl. Acad. Sci. U.S.A. 100(1):283-288 (2003).