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PKC eta Antibody (N-term) Blocking Peptide

Synthetic peptide
Catalog # BP7020a

Specification

PKC eta Antibody (N-term) Blocking Peptide - Product Information

Primary Accession P24723

PKC eta Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 5583

Other Names
Protein kinase C eta type, PKC-L, nPKC-eta, PRKCH, PKCL, PRKCL

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7020a was selected from the N-term region of human PKC eta . 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 eta Antibody (N-term) Blocking Peptide - Protein Information

Name PRKCH

Synonyms PKCL, PRKCL

Function
Calcium-independent, phospholipid- and diacylglycerol (DAG)- dependent serine/threonine-protein kinase that is involved in the regulation of cell differentiation in keratinocytes and pre-B cell receptor, mediates regulation of epithelial tight junction integrity and foam cell formation, and is required for glioblastoma proliferation and apoptosis prevention in MCF-7 cells. In keratinocytes, binds and activates the tyrosine kinase FYN, which in turn blocks epidermal growth factor receptor (EGFR) signaling and leads to keratinocyte growth arrest and differentiation. Associates with the cyclin CCNE1- CDK2-CDKN1B complex and inhibits CDK2 kinase activity, leading to RB1 dephosphorylation and thereby G1 arrest in keratinocytes. In association with RALA activates actin depolymerization, which is necessary for keratinocyte differentiation. In the pre-B cell receptor signaling, functions downstream of BLNK by up-regulating IRF4, which in turn activates L chain

gene rearrangement. Regulates epithelial tight junctions (TJs) by phosphorylating occludin (OCLN) on threonine residues, which is necessary for the assembly and maintenance of TJs. In association with PLD2 and via TLR4 signaling, is involved in lipopolysaccharide (LPS)-induced RGS2 down-regulation and foam cell formation. Upon PMA stimulation, mediates glioblastoma cell proliferation by activating the mTOR pathway, the PI3K/AKT pathway and the ERK1-dependent phosphorylation of ELK1. Involved in the protection of glioblastoma cells from irradiation-induced apoptosis by preventing caspase-9 activation. In camptothecin-treated MCF-7 cells, regulates NF-kappa-B upstream signaling by activating IKBKB, and confers protection against DNA damage-induced apoptosis. Promotes oncogenic functions of ATF2 in the nucleus while blocking its apoptotic function at mitochondria. Phosphorylates ATF2 which promotes its nuclear retention and transcriptional activity and negatively regulates its mitochondrial localization.

Cellular Location
Cytoplasm.

Tissue Location
Most abundant in lung, less in heart and skin.

PKC eta Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PKC eta Antibody (N-term) Blocking Peptide - Images

PKC eta Antibody (N-term) Blocking Peptide - Background

Protein kinase C (PKC) is a family of serine- and threonine-specific protein kinases that can be activated by calcium and the 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 eta is one of the PKC family members. This protein is most abundant in lung, less in heart and skin.

PKC eta Antibody (N-term) Blocking Peptide - References

Palmer, R.H., et al., FEBS Lett. 356(1):5-8 (1994).Bacher, N., et al., Mol. Cell. Biol. 11(1):126-133 (1991).Bacher, N., et al., Mol. Cell. Biol. 12 (3), 1404 (1992).