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PKN Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP7938a

Specification

PKN Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Q16512

PKN Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 5585

Other Names
Serine/threonine-protein kinase N1, Protease-activated kinase 1, PAK-1, Protein kinase C-like 1, Protein kinase C-like PKN, Protein kinase PKN-alpha, Protein-kinase C-related kinase 1, Serine-threonine protein kinase N, PKN1, PAK1, PKN, PRK1, PRKCL1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7938a was selected from the C-term region of human PKN . 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.

PKN Antibody (C-term) Blocking Peptide - Protein Information

Name PKN1

Synonyms PAK1, PKN, PRK1, PRKCL1

Function
PKC-related serine/threonine-protein kinase involved in various processes such as regulation of the intermediate filaments of the actin cytoskeleton, cell migration, tumor cell invasion and transcription regulation. Part of a signaling cascade that begins with the activation of the adrenergic receptor ADRA1B and leads to the activation of MAPK14. Regulates the cytoskeletal network by phosphorylating proteins such as VIM and neurofilament proteins NEFH, NEFL and NEFM, leading to inhibit their polymerization. Phosphorylates 'Ser-575', 'Ser-637' and 'Ser-669' of MAPT/Tau, lowering its ability to bind to microtubules, resulting in disruption of tubulin assembly. Acts as a key coactivator of androgen receptor (AR)-dependent transcription, by being recruited to

AR target genes and specifically mediating phosphorylation of 'Thr-11' of histone H3 (H3T11ph), a specific tag for epigenetic transcriptional activation that promotes demethylation of histone H3 'Lys-9' (H3K9me) by KDM4C/JMJD2C. Phosphorylates HDAC5, HDAC7 and HDAC9, leading to impair their import in the nucleus. Phosphorylates 'Thr-38' of PPP1R14A, 'Ser-159', 'Ser-163' and 'Ser-170' of MARCKS, and GFAP. Able to phosphorylate RPS6 in vitro.

Cellular Location
Cytoplasm. Nucleus Endosome. Cell membrane {ECO:0000250|UniProtKB:Q63433}; Peripheral membrane protein {ECO:0000250|UniProtKB:Q63433}. Cleavage furrow. Midbody Note=Associates with chromatin in a ligand-dependent manner Localization to endosomes is mediated via its interaction with RHOB Association to the cell membrane is dependent on Ser-377 phosphorylation. Accumulates during telophase at the cleavage furrow and finally concentrates around the midbody in cytokinesis {ECO:0000250|UniProtKB:Q63433, ECO:0000269|PubMed:17332740}

Tissue Location
Found ubiquitously. Expressed in heart, brain, placenta, lung, skeletal muscle, kidney and pancreas. Expressed in numerous tumor cell lines, especially in breast tumor cells

PKN Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PKN Antibody (C-term) Blocking Peptide - Images

PKN Antibody (C-term) Blocking Peptide - Background

PKN, a member of the PKC subfamily of Ser/Thr protein kinases, can phosphorylate ribosomal protein S6. It mediates GTPase Rho dependent intracellular signalling. This protein is activated by lipids, particularly cardiolipin and to a lesser extent by other acidic phospholipids This cytoplasmic protein is expressed ubiquitously, including heart, brain, placenta, lung, skeletal muscle, kidney and pancreas.

PKN Antibody (C-term) Blocking Peptide - References

Palmer, R.H., et al., FEBS Lett. 356(1):5-8 (1994).Mukai, H., et al., Biochem. Biophys. Res. Commun. 199(2):897-904 (1994).Palmer, R.H., et al., Eur. J. Biochem. 227 (1-2), 344-351 (1995).