10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Phospho-PTEN-S380 Antibody Blocking Peptide
Synthetic peptide
Catalog # BP3220a

Specification

Phospho-PTEN-S380 Antibody Blocking Peptide - Product Information

Primary Accession P60484

Phospho-PTEN-S380 Antibody Blocking Peptide - Additional Information

Gene ID 5728

Other Names
Phosphatidylinositol 3, 5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN, Mutated in multiple advanced cancers 1, Phosphatase and tensin homolog, PTEN, MMAC1, TEP1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP3220a was selected from the region of human Phospho-PTEN-S380. 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.

Phospho-PTEN-S380 Antibody Blocking Peptide - Protein Information

Name PTEN

Synonyms MMAC1, TEP1

Function
Dual-specificity protein phosphatase, dephosphorylating tyrosine-, serine- and threonine-phosphorylated proteins (PubMed:9187108, PubMed:9256433, PubMed:9616126). Also functions as a lipid phosphatase, removing the phosphate in the D3 position of the inositol ring of PtdIns(3,4,5)P3/phosphatidylinositol 3,4,5- trisphosphate, PtdIns(3,4)P2/phosphatidylinositol 3,4-diphosphate and PtdIns3P/phosphatidylinositol 3-phosphate with a preference for PtdIns(3,4,5)P3 (PubMed:<a href="http://www.uniprot.org/citations/9811831"

target="_blank">9811831, PubMed:16824732, PubMed:26504226, PubMed:9593664). Furthermore, this enzyme can also act as a cytosolic inositol 3-phosphatase acting on Ins(1,3,4,5,6)P5/inositol 1,3,4,5,6 pentakisphosphate and possibly Ins(1,3,4,5)P4/1D-myo-inositol 1,3,4,5- tetrakisphosphate (PubMed:11418101, PubMed:15979280). Antagonizes the PI3K-AKT/PKB signaling pathway by dephosphorylating phosphoinositides and thereby modulating cell cycle progression and cell survival (PubMed:31492966, PubMed:37279284). The unphosphorylated form cooperates with MAGI2 to suppress AKT1 activation (PubMed:11707428). In motile cells, suppresses the formation of lateral pseudopods and thereby promotes cell polarization and directed movement (PubMed:22279049). Dephosphorylates tyrosine-phosphorylated focal adhesion kinase and inhibits cell migration and integrin-mediated cell spreading and focal adhesion formation (PubMed:22279049). Required for growth factor-induced epithelial cell migration; growth factor stimulation induces PTEN phosphorylation which changes its binding preference from the p85 regulatory subunit of the PI3K kinase complex to DLC1 and results in translocation of the PTEN-DLC1 complex to the posterior of migrating cells to promote RHOA activation (PubMed:26166433). Meanwhile, TNS3 switches binding preference from DLC1 to p85 and the TNS3-p85 complex translocates to the leading edge of migrating cells to activate RAC1 activation (PubMed:26166433). Plays a role as a key modulator of the AKT-mTOR signaling pathway controlling the tempo of the process of newborn neurons integration during adult neurogenesis, including correct neuron positioning, dendritic development and synapse formation (By similarity). Involved in the regulation of synaptic function in excitatory hippocampal synapses. Recruited to the postsynaptic membrane upon NMDA receptor activation, is required for the modulation of synaptic activity during plasticity. Enhancement of lipid phosphatase activity is able to drive depression of AMPA receptor-mediated synaptic responses, activity required for NMDA receptor-dependent long-term depression (LTD) (By similarity). May be a negative regulator of insulin signaling and glucose metabolism in adipose tissue. The nuclear monoubiquitinated form possesses greater apoptotic potential, whereas the cytoplasmic nonubiquitinated form induces less tumor suppressive ability (PubMed:10468583, PubMed:18716620).

Cellular Location
Cytoplasm. Nucleus. Nucleus, PML body. Cell projection, dendritic spine {ECO:0000250|UniProtKB:O54857}. Postsynaptic density {ECO:0000250|UniProtKB:O54857}. Note=Monoubiquitinated form is nuclear Nonubiquitinated form is cytoplasmic. Colocalized with PML and USP7 in PML nuclear bodies (PubMed:18716620). XIAP/BIRC4 promotes its nuclear localization (PubMed:19473982). Associares with the postsynaptic density in response to NMDAR activation (By similarity) {ECO:0000250|UniProtKB:O54857, ECO:0000269|PubMed:18716620, ECO:0000269|PubMed:19473982}

Tissue Location
Expressed at a relatively high level in all adult tissues, including heart, brain, placenta, lung, liver, muscle, kidney and pancreas.

Phospho-PTEN-S380 Antibody Blocking Peptide - Protocols

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

- Blocking Peptides

Phospho-PTEN-S380 Antibody Blocking Peptide - Images

Phospho-PTEN-S380 Antibody Blocking Peptide - Background

PTEN is a tumor suppressor that is mutated in a large number of cancers at high frequency. PTEN is a phosphatidylinositol-3,4,5-trisphosphate 3-phosphatase. It contains a tension like domain as well as a catalytic domain similar to that of the dual specificity protein tyrosine phosphatases. Unlike most of the protein tyrosine phosphatases, this protein preferentially dephosphorylates phosphoinositide substrates. It negatively regulates intracellular levels of phosphatidylinositol-3,4,5-trisphosphate in cells and functions as a tumor suppressor by negatively regulating AKT/PKB signaling pathway.

Phospho-PTEN-S380 Antibody Blocking Peptide - References

Garcia, J.M., et al., Genes Chromosomes Cancer 41(2):117-124 (2004).Chu, E.C., et al., Biochem. Biophys. Res. Commun. 320(3):875-879 (2004).Minaguchi, T., et al., Cancer Lett. 210(1):57-62 (2004).Cheung, T.H., et al., Gynecol. Oncol. 93(3):621-627 (2004).Goel, A., et al., Cancer Res. 64(9):3014-3021 (2004).