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PTEN Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8436c

Product Information

Application	WB, IF, E
Primary Accession	P60484
Reactivity	Human
Predicted	Mouse, Rat, Bovine, Canine, Rabbit, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB5080
Calculated MW	47166
Antigen Region	371-403

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	This PTEN antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 371-403 amino acids from the C-terminal region of human PTEN.
Dilution	WB~~1:1000 IF~~1:10~50 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PTEN Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

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:16824732, PubMed:26504226, PubMed:9593664, PubMed:9811831). 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). Associates 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.

Background

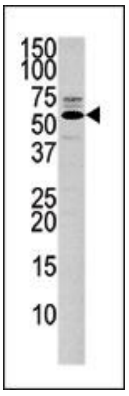
PTEN, (phosphatase and tensin homolog deleted on chromosome 10), also known as MMAC1 (mutated in

multiple advanced cancers 1), is a tumor suppressor implicated in a large number of human tumors. The PTEN phosphatase incorporates the catalytic motif (HCXXGXXRS/T) that is a signature of the protein tyrosine phosphatase family. Recombinant human PTEN is a dual phosphatase with ability to dephosphorylate both tyrosine and serine/threonine residues. PTEN functions primarily as a lipid phosphatase to regulate signal transduction pathways, with a primary target identified as phosphatidylinositol 3,4,5 trisphosphate. In addition, PTEN presents weak tyrosine phosphatase activity, which may downregulate signaling pathways involving focal adhesion kinase or Shc. PTEN negatively regulates activation of the serine/threonine kinase Akt/PKB by blocking its phosphorylation, thereby inhibiting the PI 3 kinase Akt signaling pathway, which is important for cell survival. In vivo, the majority of PTEN missense mutations detected in tumor specimens target the phosphatase domain and cause a loss in PTEN phosphatase activity. Mutations in PTEN are associated with several common cancers including prostate, brain and breast cancer, and with Cowden's disease, an autosomal dominant disorder conferring susceptibility to benign and malignant tumors. Germline mutations of PTEN are also linked Lhermitte-Duclos disease and Bannayan-Zonana syndrome. Mutations of PTEN occur in 60 to 80% of prostate cancers. PTEN is also essential for embryonic development.

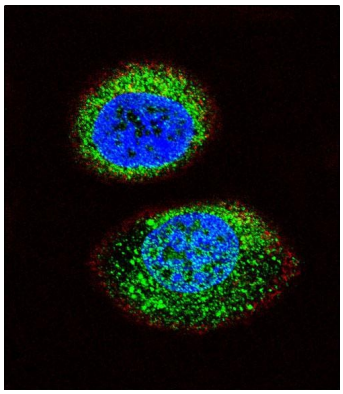
References

Smith, J.M., et al., J. Med. Genet. 39(12):937-940 (2002).
Poetsch, M., et al., Cancer Genet. Cytogenet. 132(1):20-24 (2002).
Staal, F.J., et al., Br. J. Cancer 86(10):1586-1591 (2002).
Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).
Reardon, W., et al., J. Med. Genet. 38(12):820-823 (2001).

Images



Western blot analysis of anti-PTEN Pab (Cat. #AP8436c) in 293 cell line lysate (35ug/lane). PTEN(arrow) was detected using the purified Pab.



Confocal immunofluorescent analysis of PTEN Antibody (C-term)(Cat#AP8436c) with MCF-7 cell followed by Alexa Fluor 488-conjugated goat anti-rabbit lgG (green).Actin filaments have been labeled with Alexa Fluor 555 phalloidin (red).DAPI was used to stain the cell nuclear (blue).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.