

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide
Rabbit Polyclonal Antibody [Clone]
Catalog # AH12140

Specification

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Product Information

Application	,1,3,4,
Primary Accession	P60484
Other Accession	5728 , 500466
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit / Ig
Calculated MW	55kDa KDa

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Additional Information

Gene ID 5728

Other Names

Phosphatidylinositol 3, 4, 5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN, 3.1.3.16, 3.1.3.48, 3.1.3.67, Mutated in multiple advanced cancers 1, Phosphatase and tensin homolog, PTEN, MMAC1, TEP1

Storage

Store at 2 to 8°C. Antibody is stable for 24 months.

Precautions

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Protein Information

Name PTEN

Synonyms MMAC1, TEP1

Function

Dual-specificity protein phosphatase, dephosphorylating tyrosine-, serine- and threonine-phosphorylated proteins (PubMed: [9187108](http://www.uniprot.org/citations/9187108), PubMed: [9256433](http://www.uniprot.org/citations/9256433), PubMed: [9616126](http://www.uniprot.org/citations/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: [9811831](http://www.uniprot.org/citations/9811831), PubMed: [16824732](http://www.uniprot.org/citations/16824732))

target="_blank">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). 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

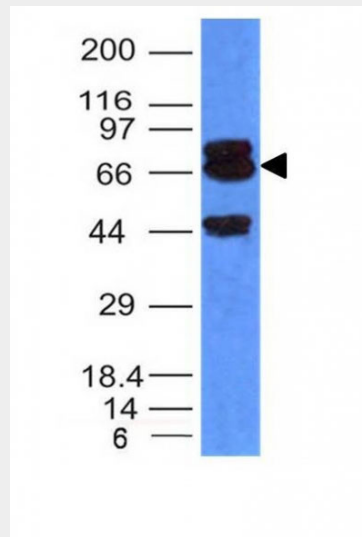
Expressed at a relatively high level in all adult tissues, including heart, brain, placenta, lung, liver, muscle, kidney and pancreas.

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Images



Formalin-fixed, paraffin-embedded human Prostate Carcinoma stained with PTEN Polyclonal Antibody.

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - Background

Recognizes a protein of 55kDa, which is identified as PTEN. PTEN is one of the most commonly lost tumor suppressors in human cancer; in fact, up to 70% of men with prostate cancer are estimated to have lost a copy of the PTEN gene at the time of diagnosis. During tumor development, mutations and deletions of PTEN occur that inactivate its enzymatic activity leading to increased cell proliferation and reduced cell death. Frequent genetic inactivation of PTEN occurs in glioblastoma, endometrial cancer, and prostate cancer; and reduced expression is found in many other tumor types such as lung and breast cancer. In breast and prostate cancer, loss of PTEN expression has been shown to correlate positively with advanced stage. Furthermore, PTEN mutation also causes a variety of inherited predispositions to cancer.

PTEN (Tumor Suppressor Protein) Antibody - With BSA and Azide - References

Steck P, et al. Identification of a candidate tumour suppressor gene, MMAC1, at chromosome 10q23.3 that is mutated in multiple advanced cancers . Nature Genetics. 1997;15 (4): 356-62. 2