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Rabbit Anti-Phospho-PTEN (Ser385) antibody

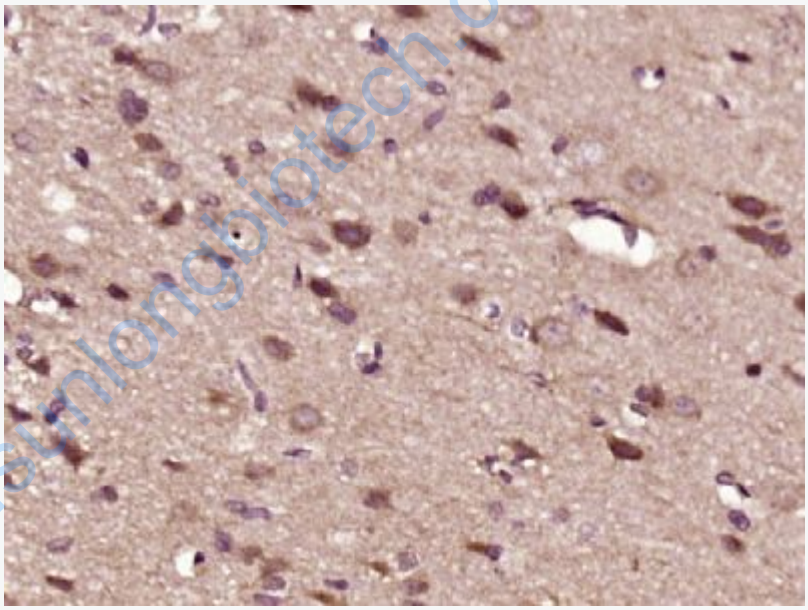
SL20195R

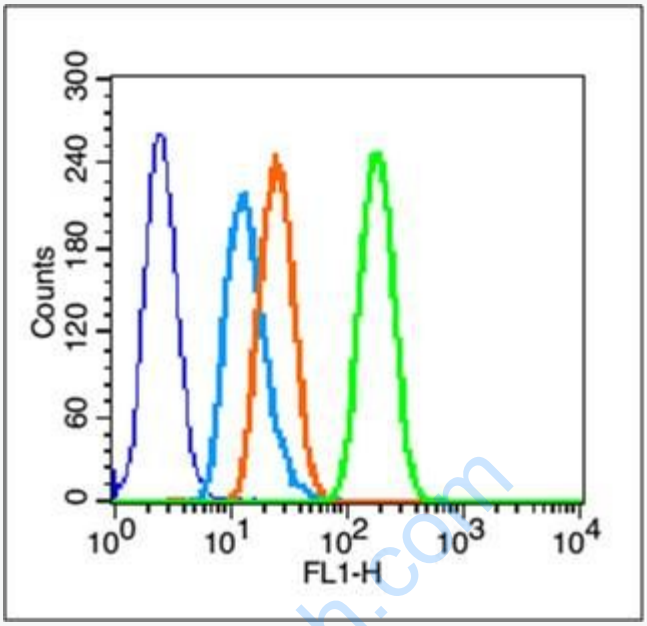
Product Name:	Phospho-PTEN (Ser385)
Chinese Name:	磷酸化Tumour抑制基因PTEN抗体
Alias:	PTEN(phospho Ser385); PTEN(phospho S385); ITGA 2; MGC11227; MHAM; MMAC 1; MMAC1; Bannayan Zonana; BZS a; Multiple hamartoma (Cowden syndrome); Mutated in Mutiple Advanced Cancers 1; Phosphatase and Tensin Homolog; Phosphatidylinositol 345 trisphosphate 3 phosphatase and dual specificity protein phosphatase PTEN; Phosphatidylinositol 345 trisphosphate 3 phosphatase; Platelet antigen BR; PTEN 1; PTEN1; Tensin homolog; TEP 1; TEP1; VLA 2 Receptor Alpha Subunit.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,
Applications:	ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	44kDa
Cellular localization:	The nucleuscytoplasmic
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthesised phosphopeptide derived from human PTEN around the phosphorylation site of Ser385:TD(p-S)DP
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	Preservative: 15mM Sodium Azide, Constituents: 1% BSA, 0.01M PBS, pH 7.4
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

PubMed:	PubMed
Product Detail:	Potential tumor suppressor. Acts as a phosphoinositide3-phosphatase by regulating PtdIns (3,4,5)P3 levels. Involved in regulation of the AKT1 signaling pathway. The unphosphorylated form cooperates with AIP1 to suppress AKT1 activation.The PTEN/MMAC1 discovers the first to have the suppress of the phosphoric acid enzyme activity cancer gene currently.The gene of PTEN locates the chromosome10q23 area, sending forth sex tumor and a few households cancers with the variety to suffer from the comprehensive disease easilyrelevant.The activity that passes to repress the Akt regulates the cell period, the cell ground rule decease and glues to connect.This text discussed PTEN structure, function and its correlationses, the PTEN is in tumor repress function mechanism. **Function:** Tumor suppressor. Acts as a dual-specificity protein phosphatase, dephosphorylating tyrosine-, serine- and threonine-phosphorylated proteins. Also acts as a lipid phosphatase, removing the phosphate in the D3 position of the inositol ring from phosphatidylinositol 3,4,5-trisphosphate, phosphatidylinositol 3,4-diphosphate, phosphatidylinositol 3-phosphate and inositol 1,3,4,5-tetrakisphosphate with order of substrate preference in vitro PtdIns(3,4,5)P3 > PtdIns(3,4)P2 > PtdIns3P > Ins(1,3,4,5)P4. The lipid phosphatase activity is critical for its tumor suppressor function. Antagonizes the PI3K-AKT/PKB signaling pathway by dephosphorylating phosphoinositides and thereby modulating cell cycle progression and cell survival. The unphosphorylated form cooperates with AIP1 to suppress AKT1 activation. Dephosphorylates tyrosine-phosphorylated focal adhesion kinase and inhibits cell migration and integrin-mediated cell spreading and focal adhesion formation. 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. 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. **Subunit:** Monomer. The unphosphorylated form interacts with the second PDZ domain of AIP1 and with DLG1 and MAST2 in vitro. Interacts with MAGI2, MAGI3, MAST1 and MAST3, but neither with MAST4 nor with DLG5. Interaction with MAGI2 increases protein stability. Interacts with NEDD4. Interacts with NDFIP1 and NDFIP2; in the presence of NEDD4 or ITCH, this interaction promotes PTEN ubiquitination. Interacts (via C2 domain) with FRK. Interacts with USP7; the interaction is direct. Interacts with ROCK1. Interacts with XIAP/BIRC4. **Subcellular Location:** Cytoplasm. Nucleus. Nucleus, PML body. Note=Monoubiquitinated form is nuclear. Nonubiquitinated form is cytoplasmic. Colocalized with PML and USP7 in PML nuclear bodies. XIAP/BIRC4 promotes its nuclear localization.

	Tissue Specificity: Expressed at a relatively high level in all adult tissues, including heart, brain, placenta, lung, liver, muscle, kidney and pancreas. **Post-translational modifications:** Constitutively phosphorylated by CK2 under normal conditions. Phosphorylated in vitro by MAST1, MAST2 and MAST3. Phosphorylation results in an inhibited activity towards PIP3. Phosphorylation can both inhibit or promote PDZ-binding. Phosphorylation at Tyr-336 by FRK/PTK5 protects this protein from ubiquitin-mediated degradation probably by inhibiting its binding to NEDD4. Phosphorylation by ROCK1 is essential for its stability and activity. Phosphorylation by PLK3 promotes its stability and prevents its degradation by the proteasome. Monoubiquitinated; monoubiquitination is increased in presence of retinoic acid. Deubiquitinated by USP7; leading to its nuclear exclusion. Monoubiquitination of one of either Lys-13 and Lys-289 amino acid is sufficient to modulate PTEN compartmentalization. Ubiquitinated by XIAP/BIRC4. **DISEASE:** Defects in PTEN are a cause of Cowden disease (CD) [MIM:158350]; also known as Cowden syndrome (CS). CD is an autosomal dominant cancer predisposition syndrome associated with elevated risk for tumors of the breast, thyroid and skin. The predominant phenotype for CD is multiple hamartoma syndrome, in many organ systems including the breast (70% of CD patients), thyroid (40-60%), skin, CNS (40%), gastrointestinal tract. Affected individuals are at an increased risk of both breast and thyroid cancers. Trichilemmomas (benign tumors of the hair follicle infundibulum), and mucocutaneous papillomatosis (99%) are hallmarks of CD. Defects in PTEN are the cause of Lhermitte-Duclos disease (LDD) [MIM:158350]; also known as cerebelloparenchymal disorder VI. LDD is characterized by dysplastic gangliocytoma of the cerebellum which often results in cerebellar signs and seizures. LDD and CD seem to be the same entity, and are considered as hamartoma-neoplasia syndromes. Defects in PTEN are a cause of Bannayan-Zonana syndrome (BZS) [MIM:153480]; also known as Ruvalcaba-Myhre-Smith syndrome (RMSS) or Bannayan-Riley-Ruvalcaba syndrome (BRRS). In BZS there seems not to be an increased risk of malignancy. It has a partial clinical overlap with CD. BZS is characterized by the classic triad of macrocephaly, lipomatosis and pigmented macules of the gland penis. Defects in PTEN are a cause of head and neck squamous cell carcinomas (HNSCC) [MIM:275355]; also known as squamous cell carcinoma of the head and neck. Defects in PTEN are a cause of susceptibility to endometrial cancer (ENDMC) [MIM:608089]. Note=PTEN mutations are found in a subset of patients with Proteus syndrome, a genetically heterogeneous condition. The molecular diagnosis of PTEN mutation positive cases classifies Proteus syndrome patients as part of the PTEN hamartoma syndrome spectrum. As such, patients surviving the early years of Proteus syndrome are likely at a greater risk of developing malignancies. Defects in PTEN are a cause of susceptibility to glioma type 2 (GLM2) [MIM:613028].

	Gliomas are central nervous system neoplasms derived from glial cells and comprise astrocytomas, glioblastoma multiforme, oligodendrogliomas, and ependymomas. [DISEASE] Defects in PTEN are a cause of VACTERL association with hydrocephalus (VACTERL-H) [MIM:276950]. VACTERL is an acronym for vertebral anomalies, anal atresia, congenital cardiac disease, tracheoesophageal fistula, renal anomalies, radial dysplasia, and other limb defects. Defects in PTEN may be a cause of susceptibility to prostate cancer (PC) [MIM:176807]. It is a malignancy originating in tissues of the prostate. Most prostate cancers are adenocarcinomas that develop in the acini of the prostatic ducts. Other rare histopathologic types of prostate cancer that occur in approximately 5% of patients include small cell carcinoma, mucinous carcinoma, prostatic ductal carcinoma, transitional cell carcinoma, squamous cell carcinoma, basal cell carcinoma, adenoid cystic carcinoma (basaloid), signet-ring cell carcinoma and neuroendocrine carcinoma. Defects in PTEN are a cause of macrocephaly/autism syndrome (MCEPHAS) [MIM:605309]. Patients have autism spectrum disorders and macrocephaly, with head circumferences ranging from +2.5 to +8 SD for age and sex (average head circumference +4.0 SD). Note=A microdeletion of chromosome 10q23 involving BMPR1A and PTEN is a cause of chromosome 10q23 deletion syndrome, which shows overlapping features of the following three disorders: Bannayan-Zonana syndrome, Cowden disease and juvenile polyposis syndrome. **Similarity:** Contains 1 C2 tensin-type domain. Contains 1 phosphatase tensin-type domain. **SWISS:** P60484 **Gene ID:** 5728 **Database links:** Entrez Gene: 5728Human Entrez Gene: 19211Mouse Entrez Gene: 50557Rat Omim: 601728Human SwissProt: P60484Human SwissProt: O08586Mouse

	Unigene: 500466Human Unigene: 245395Mouse Unigene: 444861Mouse **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications. PTEN是一种Tumour抑制基因(N端抗体)，在维持细胞的增殖、分化和凋亡平衡起重要作用，该基因在许多Tumour常表现为突变或杂合性丢失，NMAC1主要用于胶质瘤、乳腺、前列腺癌、非何杰金氏淋巴瘤各种恶性Tumour的研究.
Picture:	 Paraformaldehyde-fixed, paraffin embedded (rat brain tissue); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (PTEN(Ser385)) Polyclonal Antibody, Unconjugated (SL20195R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructionsand DAB staining.



Blank control (blue line): A431 cells (fixed with 70% methanol (Overnight at 4℃) and then permeabilized with 90% ice-cold methanol for 20 min at -20℃).

Primary Antibody (green line): Rabbit Anti-Phospho-PTEN(Ser385) antibody (SL20195R),Dilution: 3µg /10^6 cells;

Isotype Control Antibody (orange line): Rabbit IgG .

Secondary Antibody (white blue line): Goat anti-rabbit IgG-FITC,Dilution: 1µg /test.