Immunotag™ PTEN (Acetyl Lys402) Polyclonal Antibody

Antibody Specification

Catalog No.	ITK0053
Product Description	Immunotag™ PTEN (Acetyl Lys402) Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	PTEN (Lys402)
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	Western Blot: 1/500 - 1/2000. ELISA: 1/20000. Not yet tested in other applications.
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat
Host Species	Rabbit
Immunogen	Synthesized acetyl-peptide derived from the human PTEN around the acetylation site of K402.
Specificity	Acetyl-PTEN (K402) Polyclonal AntibodySynthesized peptide derived from the human PTEN around the acetylation site of K402.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	PTEN
Accession No.	P60484 O08586
Alternate Names	PTEN; MMAC1; TEP1; Phosphatidylinositol 3,4,5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN; Mutated in multiple advanced cancers 1; Phosphatase and tensin homolog

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Description	phosphatase and tensin homolog(PTEN) Homo sapiens This gene was identified as a tumor suppressor that is mutated in a large number of cancers at high frequency. The protein encoded by this gene is a phosphatidylinositol-3,4,5-trisphosphate 3-phosphatase. It contains a tensin 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. The use of a non-canonical (CUG) upstream initiation site produces a longer isoform that initiates translation with a leucine, and is thought to be preferentially associated with the mitochondrial inner membrane. This longer isoform may help regulate ener
Cell Pathway/ Category	Inositol phosphate metabolism,Phosphatidylinositol signaling system,p53,Focal adhesion,Tight junction,Pathways in cancer,Endometrial cancer,Glioma,Prostate cancer,Melanoma,Small cell lung cancer,
Protein Expression	Epithelium,Lung,
Subcellular Localization	extracellular region,nucleus,nucleoplasm,cytoplasm,mitochondrion,cytosol,plasma membrane,cytoplasmic side of plasma membrane,apical plasma membrane,PML body,myelin sheath adaxonal region,cell projection,neuron

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Protein Function	catalytic activity:A phosphoprotein + H(2)O = a protein + phosphate.,catalytic activity:Phosphatidylinositol 3,4,5-trisphosphate + H(2)O = phosphatidylinositol 4,5-bisphosphate + phosphate.,catalytic activity:Protein tyrosine phosphate + H(2)O = protein tyrosine + phosphate.,cofactor:Magnesium.,disease:A microdeletion of chromosome 10q23 involving PTEN and BMPR1A is a cause of chromosome 10q23 deletion syndrome [MIM:612242]. This syndrome shows overlapping features of the following three disorders: Bannayan-Zonana syndrome, Cowden disease and juvenile polyposis syndrome.,disease:Defects in PTEN are a cause of Bannayan-Zonana syndrome (BZS) [MIM:153480]; also known as Ruvalcaba-Riley-Smith 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.,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.,disease:Defects in PTEN are a cause of macrocephaly/autism syndrome [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).,disease:Defects in PTEN are a cause of oligodendroglioma [MIM:137800]; also called oligodendroblastoma or familial glioma of brain. Oligodendroglioma is a usually benign neoplasm derived from and composed of oligodendrogliocytes in varying stages of differentiation. The majority are seen in adults in the white matter of the brain.,disease:Defects in PTEN are a cause of Proteus syndrome [MIM:176920]. Proteus syndrome is a hamartomatous disorder characterized by overgrowth of multiple tissues, connective tissue and epidermal naevi, and vascular malformations. These presentations are usually apparent at birth or soon after and continue to develop as the patient ages. It is named after the Greek god Proteus who, legend has it, could change his shape at will to avoid capture. Tumors, mostly benign but some malignant, have also been reported in Proteus syndrome, generally presenting by the age of 20 years and including papillary adenocarcinoma of the testis, meningioma, and cystadenoma of the ovaries.,disease:Defects in PTEN are a cause of squamous cell carcinoma of the head and neck (HNSCC) [MIM:275355].,disease:Defects in PTEN are a cause of susceptibility to endometrial cancer [MIM:608089].,disease:Defects in PTEN are a cause of VACTERL association with hydrocephalus [MIM:276950]; which includes also VATER association with hydrocephalus. VACTERL is an acronym for vertebral anomalies, anal atresia, congenital cardiac disease, tracheoesophageal fistula, renal anomalies, radial dysplasia, and other limb defects.,disease:Defects in PTEN are involved in prostate cancer [MIM:176807].,disease: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.,disease:Mutations of PTEN are found in a large number of cancers.,domain:The C2 domain binds phospholipid membranes in vitro in a Ca(2+)-independent manner; this binding is important for its tumor suppressor function.,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

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Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.