

Product datasheet

PTEN peptide ab157804

Overview

Product name PTEN peptide

Description

Nature Synthetic

Specifications

Our [Abpromise guarantee](#) covers the use of **ab157804** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications Blocking - Blocking peptide for Anti-PTEN antibody [Y184] ([ab32199](#))

Form Liquid

Additional notes

- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Information available upon request.

General Info

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 $\text{PtdIns}(3,4,5)\text{P}_3 > \text{PtdIns}(3,4)\text{P}_2 > \text{PtdIns}3\text{P} > \text{Ins}(1,3,4,5)\text{P}_4$. 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. In motile cells, suppresses the formation of lateral pseudopods and thereby promotes cell polarization and directed movement.

Isoform alpha: Functional kinase, like isoform 1 it antagonizes the PI3K-AKT/PKB signaling pathway. Plays a role in mitochondrial energetic metabolism by promoting COX activity and ATP production, via collaboration with isoform 1 in increasing protein levels of PINK1.

Tissue specificity

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

Involvement in disease

Cowden syndrome 1

Lhermitte-Duclos disease

Bannayan-Riley-Ruvalcaba syndrome

Squamous cell carcinoma of the head and neck

Endometrial cancer

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.

Glioma 2

VACTERL association with hydrocephalus

Prostate cancer

Macrocephaly/autism syndrome

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.

Sequence similarities

Contains 1 C2 tensin-type domain.

Contains 1 phosphatase tensin-type domain.

Domain

The C2 domain binds phospholipid membranes in vitro in a $\text{Ca}(2+)$ -independent manner; this binding is important for its tumor suppressor function.

Post-translational modifications

Constitutively phosphorylated by CK2 under normal conditions. Phosphorylated in vitro by MAST1, MAST2, MAST3 and STK11. 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.

Cellular localization

Secreted. May be secreted via a classical signal peptide and reenter into cells with the help of a poly-Arg motif and Cytoplasm. Nucleus. Nucleus, PML body. Monoubiquitinated form is nuclear. Nonubiquitinated form is cytoplasmic. Colocalized with PML and USP7 in PML nuclear bodies. XIAP/BIRC4 promotes its nuclear localization.

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