SANTA CRUZ BIOTECHNOLOGY, INC.

p-PTEN (Ser 380/Thr 382/383): sc-101789

BACKGROUND

As human tumors progress to advanced stages, one genetic alteration that occurs at high frequency is a loss of heterozygosity (LOH) at chromosome 10q23.31. Mapping of homozygous deletions on this chromosome led to the isolation of the PTEN gene, also designated MMAC1 (for mutated in multiple advanced cancers) and TEP1. This candidate tumor suppressor gene exhibits a high frequency of mutations in human glioblastomas and is also mutated in other cancers, including sporadic brain, breast, kidney and prostate cancers. PTEN has been associated with Cowden disease, an autosomal dominant cancer predisposition syndrome. The PTEN gene product is a putative protein tyrosine phosphatase that is localized to the cytoplasm, and it shares extensive homology with the cytoskeletal proteins tensin and auxilin. Gene transfer studies have indicated that the phosphatase domain of PTEN is essential for growth suppression of glioma cells. PTEN contains five major phosphorylation sites and these are all located in the C-terminal 50 amino acid tail region (Ser 370, Ser 380, Thr 382, Thr 383 and Ser 385) and have been implicated in controlling PTEN activity.

REFERENCES

1. Bigner, S.H., et al. 1988. Specific chromosomal abnormalities in malignant human gliomas. Cancer Res. 48: 405-411.
2. James, C.D., et al. 1988. Clonal genomic alterations in glioma malignancy stages. Cancer Res. 48: 5546-5551.
3. Steck, P.A., et al. 1997. Identification of a candidate tumour suppressor gene, MMAC1, at chromosome 10q23.3 that is mutated in multiple advanced cancers. Nat. Genet. 15: 356-362.
4. Liaw, D., et al. 1997. Germline mutations of the PTEN gene in Cowden disease, an inherited breast and thyroid cancer syndrome. Nat. Genet. 16: 64-67.

CHROMOSOMAL LOCATION

Genetic locus: PTEN (human) mapping to 10q23.31; Pten (mouse) mapping to 19 C1.

SOURCE

p-PTEN (Ser 380/Thr 382/383) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Ser 380/Thr 382/383 phosphorylated PTEN of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

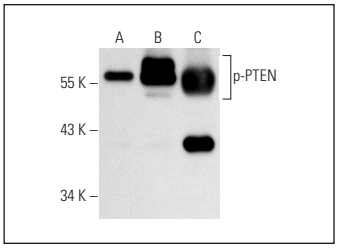
p-PTEN (Ser 380/Thr 382/383) is recommended for detection of Ser 380/Thr 382/383 phosphorylated PTEN of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for PTEN siRNA (h): sc-29459, PTEN siRNA (m): sc-36326, PTEN siRNA (r): sc-61873, PTEN shRNA Plasmid (h): sc-29459-SH, PTEN shRNA Plasmid (m): sc-36326-SH, PTEN shRNA Plasmid (r): sc-61873-SH, PTEN shRNA (h) Lentiviral Particles: sc-29459-V, PTEN shRNA (m) Lentiviral Particles: sc-36326-V and PTEN shRNA (r) Lentiviral Particles: sc-61873-V.

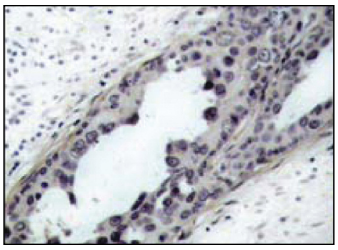
Molecular Weight of p-PTEN: 55 kDa.

Positive Controls: PTEN (h): 293T Lysate: sc-159790 or WI-38 whole cell lysate: sc-364260.

DATA



p-PTEN (Ser 380/Thr 382/383): sc-101789. Western blot analysis of PTEN phosphorylation in non-transfected 293T: sc-117752 (**A**), human PTEN transfected 293T: sc-159790 (**B**) and WI-38 (**C**) whole cell lysates.



p-PTEN (Ser 380/Thr 382/383): sc-101789. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human breast carcinoma tissue showing cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. Riggio, M., et al. 2012. PI3K/AKT pathway regulates phosphorylation of steroid receptors, hormone independence and tumor differentiation in breast cancer. Carcinogenesis 33: 509-518.
2. Fuente-Martín, E., et al. 2013. Hypothalamic inflammation without astrogliosis in response to high sucrose intake is modulated by neonatal nutrition in male rats. Endocrinology 154: 2318-2330.
3. Yang, L., et al. 2014. Identification of prolidase as a high affinity ligand of the ErbB2 receptor and its regulation of ErbB2 signaling and cell growth. Cell Death Dis. 5: e1211.