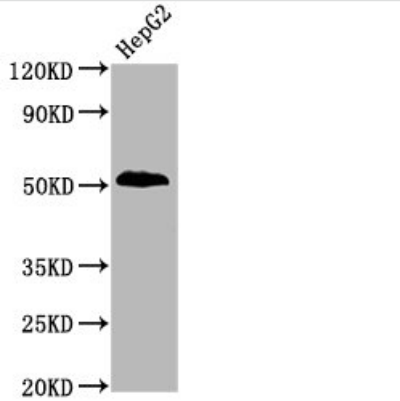
Phospho-PTEN (S380) Antibody

Product Code	CSB-RA018964A380phHU
Abbreviation	Phosphatidylinositol 3,4,5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN
Storage	Upon receipt, store at -20°C or -80°C. Avoid repeated freeze.
Uniprot No.	P60484
Immunogen	A synthesized peptide derived from Human Phospho-PTEN (S380)
Species Reactivity	Human
Tested Applications	ELISA, WB; Recommended dilution: WB:1:500-1:5000
Relevance	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 (PubMed:26504226). 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.
Form	Liquid
Conjugate	Non-conjugated
Storage Buffer	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Purification Method	Affinity-chromatography
Isotype	Rabbit IgG
Clonality	Monoclonal
Alias	Phosphatidylinositol 3, 4, 5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN, Mutated in multiple advanced cancers 1, Phosphatase and tensin homolog, PTEN, MMAC1, TEP1

Immunogen Species	Homo sapiens (Human)
Research Area	Cell Biology
Gene Names	PTEN
Accession NO.	4D10

Image



Western Blot
Positive WB detected in HepG2 whole cell lysate
All lanes Phospho-PTEN antibody at 1.9μg/ml
Secondary
Goat polyclonal to rabbit IgG at 1/50000 dilution
Predicted band size: 54 KDa
Observed band size: 54 KDa

Description

CUSABIO cloned PTEN antibody-coding genes into plasma vectors and then transfected these vector clones into mammalian cells using a lipid-based transfection reagent. Following transient expression, the recombinant antibodies against PTEN were harvested and characterized. The recombinant PTEN antibody was purified by affinity-chromatography from the culture medium. It can be used to detect PTEN protein from Human in the ELISA, WB.

PTEN is a protein-coding gene that encodes Phosphatidylinositol 3,4,5-trisphosphate 3-phosphatase and dual-specificity protein phosphatase PTEN. Disorders associated with PTEN include Cowden syndrome 1 and macrocephaly/autism syndrome. Its related pathways include metabolism and T cell receptor and co-stimulatory signaling. According to some studies, PTEN may have the following characteristics.
PTEN may inhibit tumor cell growth by antagonizing protein tyrosine kinases, and may regulate tumor cell invasion and metastasis through interactions at focal adhesions. PTEN is frequently disrupted in a variety of sporadic tumors and is targeted by germline mutations in patients with cancer susceptibility syndromes. The mechanisms regulating PTEN expression and function, including transcriptional regulation, post-transcriptional regulation of noncoding RNAs, post-translational modifications, and protein-protein interactions, are altered in cancer.