

# PTEN mouse Monoclonal Antibody(2C10)

Catalog # AP63769

### **Product Information**

Application IHC-P
Primary Accession P60484
Reactivity Human
Host Mouse
Clonality Monoclonal
Calculated MW 47166

## **Additional Information**

**Gene ID** 5728

Other Names PTEN

**Dilution** IHC-P~~N/A

Format Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium

azide.

Storage Conditions -20°C

## **Protein Information**

Name PTEN

Synonyms MMAC1, TEP1

**Function** Dual-specificity protein phosphatase, dephosphorylating tyrosine-, serine-

and threonine-phosphorylated proteins (PubMed:9187108, PubMed:9256433,

PubMed: 9616126). Also functions as a lipid phosphatase, removing the

phosphate in the D3 position of the inositol ring of PtdIns(3,4,5)P3/phosphatidylinositol 3,4,5- trisphosphate, PtdIns(3,4)P2/phosphatidylinositol 3,4-diphosphate and

PtdIns3P/phosphatidylinositol 3-phosphate with a preference for

PtdIns(3,4,5)P3 (PubMed:<u>16824732</u>, PubMed:<u>26504226</u>, PubMed:<u>9593664</u>, PubMed:<u>9811831</u>). Furthermore, this enzyme can also act as a cytosolic inositol 3-phosphatase acting on Ins(1,3,4,5,6)P5/inositol 1,3,4,5,6 pentakisphosphate and possibly Ins(1,3,4,5)P4/1D-myo-inositol 1,3,4,5-tetrakisphosphate (PubMed:<u>11418101</u>, PubMed:<u>15979280</u>). Antagonizes the PI3K-AKT/PKB signaling pathway by dephosphorylating phosphoinositides and

thereby modulating cell cycle progression and cell survival

(PubMed:<u>31492966</u>, PubMed:<u>37279284</u>). The unphosphorylated form cooperates with MAGI2 to suppress AKT1 activation (PubMed:<u>11707428</u>). In motile cells, suppresses the formation of lateral pseudopods and thereby

promotes cell polarization and directed movement (PubMed:22279049). Dephosphorylates tyrosine-phosphorylated focal adhesion kinase and inhibits cell migration and integrin-mediated cell spreading and focal adhesion formation (PubMed:22279049), Required for growth factor-induced epithelial cell migration; growth factor stimulation induces PTEN phosphorylation which changes its binding preference from the p85 regulatory subunit of the PI3K kinase complex to DLC1 and results in translocation of the PTEN-DLC1 complex to the posterior of migrating cells to promote RHOA activation (PubMed: 26166433). Meanwhile, TNS3 switches binding preference from DLC1 to p85 and the TNS3-p85 complex translocates to the leading edge of migrating cells to activate RAC1 activation (PubMed: 26166433). 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 (By similarity). Involved in the regulation of synaptic function in excitatory hippocampal synapses. Recruited to the postsynaptic membrane upon NMDA receptor activation, is required for the modulation of synaptic activity during plasticity. Enhancement of lipid phosphatase activity is able to drive depression of AMPA receptor-mediated synaptic responses, activity required for NMDA receptor-dependent long-term depression (LTD) (By similarity). 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 (PubMed: 10468583, PubMed: 18716620).

#### **Cellular Location**

Cytoplasm. Nucleus. Nucleus, PML body. Cell projection, dendritic spine {ECO:0000250 | UniProtKB:O54857}. Postsynaptic density {ECO:0000250 | UniProtKB:O54857}. Note=Monoubiquitinated form is nuclear Nonubiquitinated form is cytoplasmic. Colocalized with PML and USP7 in PML nuclear bodies (PubMed:18716620). XIAP/BIRC4 promotes its nuclear localization (PubMed:19473982). Associares with the postsynaptic density in response to NMDAR activation (By similarity) {ECO:0000250 | UniProtKB:O54857, ECO:0000269 | PubMed:18716620, ECO:0000269 | PubMed:19473982}

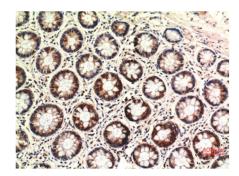
### **Tissue Location**

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

# **Background**

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.

# **Images**



Immunohistochemical analysis of paraffin-embedded Human Colon Carcinoma Tissue using PTEN Mouse mAb diluted at 1:200.

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