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SAP1 Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP8429a

Specification

SAP1 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession	Q9HD43
Other Accession	Q15426

SAP1 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 5794

Other Names
Receptor-type tyrosine-protein phosphatase H, R-PTP-H, Stomach cancer-associated protein tyrosine phosphatase 1, SAP-1, Transmembrane-type protein-tyrosine phosphatase type H, PTPRH, SAP1

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8429a was selected from the C-term region of human SAP1 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

SAP1 Antibody (C-term) Blocking Peptide - Protein Information

Name PTPRH

Synonyms SAP1

Function
Protein phosphatase that may contribute to contact inhibition of cell growth and motility by mediating the dephosphorylation of focal adhesion-associated substrates and thus negatively regulating integrin- promoted signaling processes. Induces apoptotic cell death by at least two distinct mechanisms: inhibition of cell survival signaling mediated by PI 3-kinase, Akt, and ILK and activation of a caspase-dependent proapoptotic pathway. Inhibits the basal activity of LCK and its activation in response to TCR stimulation and TCR-induced activation of MAP kinase and surface expression of CD69. Inhibits TCR-induced tyrosine phosphorylation of LAT and ZAP70. Inhibits both

basal activity of DOK1 and its CD2-induced tyrosine phosphorylation. Induces dephosphorylation of BCAR1, focal adhesion kinase and SRC. Reduces migratory activity of activity of Jurkat cells. Reduces tyrosine phosphorylation of CEACAM20 and thereby contributes to suppress the intestinal immune response CEACAM20 (By similarity).

Cellular Location
Cell projection, microvillus membrane {ECO:0000250|UniProtKB:E9Q0N2}; Single-pass type I membrane protein. Apical cell membrane {ECO:0000250|UniProtKB:E9Q0N2}; Single-pass type I membrane protein. Cytoplasm. Note=Colocalizes with CEACAM20 at the apical brush border of intestinal cells {ECO:0000250|UniProtKB:E9Q0N2}

Tissue Location
Expressed at high levels in the brain, spleen and liver and at lower levels in the heart and stomach. Expressed in pancreatic and colorectal cancer cells, but not in normal pancreas or colon. Expression in hepatocellular carcinoma is related to the differentiation status of the tumor and expression is inversely related to tumor aggressiveness.

SAP1 Antibody (C-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

SAP1 Antibody (C-term) Blocking Peptide - Images

SAP1 Antibody (C-term) Blocking Peptide - Background

Phosphorylation of receptors by protein kinases is a process that can be reversed by a group of enzymes called protein phosphatases. Coordinated control of kinases and phosphatases provides the cell with the capacity to rapidly switch between phosphorylated and dephosphorylated protein states in dynamic response to environmental stimuli. Activation of critical enzymes by kinase phosphorylation alone is not enough to provide adequate regulation ? it is the combination with phosphatase dephosphorylation that effectively creates on/off switches to control cellular events. Errors in control, either through kinases or their counterpart phosphatases, can lead to unchecked cell growth attributable to human cancers and developmental disorders. Potential mechanisms to control dephosphorylation include changes in the expression of protein phosphatases, their subcellular localization, phosphorylation of phosphatase catalytic and regulatory subunits and regulation by endogenous phosphatase inhibitors. Most protein phosphatases are not stringently specific for their substrates. Consequently, changes in phosphatase activity may have a broad impact on dephosphorylation and turnover of phosphoproteins that are substrates for different kinases. This may be an important point of control to connect cellular circuitry of interrelated signaling pathways, and to synchronize physiological responses.