

PPP6C Antibody (N-term) Blocking Peptide

Synthetic peptide Catalog # BP8477a

Specification

PPP6C Antibody (N-term) Blocking Peptide - Product Information

Primary Accession O00743
Other Accession NP 006238

PPP6C Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 5537

Other Names

Serine/threonine-protein phosphatase 6 catalytic subunit, PP6C, Serine/threonine-protein phosphatase 6 catalytic subunit, N-terminally processed, PPP6C, PPP6

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP8477a was selected from the N-term region of human PPP6C. 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.

PPP6C Antibody (N-term) Blocking Peptide - Protein Information

Name PPP6C {ECO:0000303|PubMed:29053956, ECO:0000312|HGNC:HGNC:9323}

Function

Catalytic subunit of protein phosphatase 6 (PP6) (PubMed:17079228, PubMed:29053956, PubMed:32474700). PP6 is a component of a signaling pathway regulating cell cycle progression in response to IL2 receptor stimulation (PubMed:<a href="http://www.uniprot.org/citations/10227379"

target="_blank">10227379). N-terminal domain restricts G1 to S phase progression in cancer cells, in part through control of cyclin D1 (PubMed:17568194). During mitosis, regulates spindle positioning (PubMed:<a



href="http://www.uniprot.org/citations/27335426" target="_blank">27335426). Down-regulates MAP3K7 kinase activation of the IL1 signaling pathway by dephosphorylation of MAP3K7 (PubMed:17079228). Participates also in the innate immune defense against viruses by desphosphorylating RIGI, an essential step that triggers RIGI-mediated signaling activation (PubMed:29053956). Also regulates innate immunity by acting as a negative regulator of the cGAS-STING pathway: mediates dephosphorylation and inactivation of CGAS and STING1 (PubMed:32753499, PubMed:32474700). CGAS dephosphorylation at 'Ser-435' impairs its ability to bind GTP, thereby inactivating it (PubMed:32474700).

Cellular Location

Mitochondrion. Cytoplasm

Tissue Location

Ubiquitously expressed in all tissues tested with highest expression levels in testis, heart, kidney, brain, stomach, liver and skeletal muscle and lowest in placenta, lung colon and spleen.

PPP6C Antibody (N-term) Blocking Peptide - Protocols

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

• Blocking Peptides

PPP6C Antibody (N-term) Blocking Peptide - Images

PPP6C Antibody (N-term) Blocking Peptide - Background

PPP6C belongs to the PPP phosphatase family, PP-V subfamily. Reversible phosphorylation of proteins on serine and threonine residues is an important biochemical event that regulates a broad variety of intracellular processes. The phosphorylation state is determined by the well-controlled balance of activities of serine/threonine-specific protein kinases and protein phosphatases, including PPP6C. Expression levels are highest in testis, heart, and skeletal muscle and lowest in placenta, lung, and kidney. PPP6C can complement mutations in the S. cerevisiae Sit4 and S. pombe ppe1 genes, indicating that PPP6C is the functional homolog of yeast Sit4p and ppe1. Since Sit4p is required for the G1 to S transition of the cell cycle and ppe1 is involved in cell shape control and mitotic division, it has been suggested that PPP6C functions in cell cycle regulation.

PPP6C Antibody (N-term) Blocking Peptide - References

Yang, J., et al., EMBO J. 24(1):1-10 (2005).Zhou, G., et al., J. Biol. Chem. 279(45):46595-46605 (2004).Huang, S., et al., J. Biol. Chem. 279(35):36490-36496 (2004).Swingle, M.R., et al., J. Biol. Chem. 279(32):33992-33999 (2004).Wechsler, T., et al., Proc. Natl. Acad. Sci. U.S.A. 101(5):1247-1252 (2004).