

PLK2 (SNK) Antibody (C-term) Blocking peptide

Synthetic peptide Catalog # BP7252a

Specification

PLK2 (SNK) Antibody (C-term) Blocking peptide - Product Information

Primary Accession

Q9NYY3

PLK2 (SNK) Antibody (C-term) Blocking peptide - Additional Information

Gene ID 10769

Other Names

Serine/threonine-protein kinase PLK2, Polo-like kinase 2, PLK-2, hPlk2, Serine/threonine-protein kinase SNK, hSNK, Serum-inducible kinase, PLK2, SNK

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP7252a was selected from the C-term region of human SNK . 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.

PLK2 (SNK) Antibody (C-term) Blocking peptide - Protein Information

Name PLK2

Synonyms SNK

Function

Tumor suppressor serine/threonine-protein kinase involved in synaptic plasticity, centriole duplication and G1/S phase transition. Polo-like kinases act by binding and phosphorylating proteins that are already phosphorylated on a specific motif recognized by the POLO box domains. Phosphorylates CENPJ, NPM1, RAPGEF2, RASGRF1, SNCA, SIPA1L1 and SYNGAP1. Plays a key role in synaptic plasticity and memory by regulating the Ras and Rap protein signaling: required for overactivity-dependent spine remodeling by phosphorylating the Ras activator RASGRF1 and the Rap inhibitor SIPA1L1 leading to their degradation by the proteasome. Conversely, phosphorylates the Rap activator RAPGEF2 and the Ras inhibitor SYNGAP1, promoting their activity. Also regulates synaptic plasticity independently of kinase activity, via its interaction with NSF that disrupts the



interaction between NSF and the GRIA2 subunit of AMPARs, leading to a rapid rundown of AMPAR-mediated current that occludes long term depression. Required for procentriole formation and centriole duplication by phosphorylating CENPJ and NPM1, respectively. Its induction by p53/TP53 suggests that it may participate in the mitotic checkpoint following stress.

Cellular Location

Cytoplasm, cytoskeleton, microtubule organizing center, centrosome, centriole. Cell projection, dendrite Note=Localizes to centrosomes during early G1 phase where it only associates to the mother centriole and then distributes equally to both mother and daughter centrioles at the onset of S phase

Tissue Location

Expressed at higher level in the fetal lung, kidney, spleen and heart.

PLK2 (SNK) Antibody (C-term) Blocking peptide - Protocols

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

• Blocking Peptides

PLK2 (SNK) Antibody (C-term) Blocking peptide - Images

PLK2 (SNK) Antibody (C-term) Blocking peptide - Background

Plks (polo-like kinases) encode serine/threonine kinases that are closelyrelated to polo and CDC5, genes that are required for passage through mitosisin Drosophila and Saccharomyces, respectively. Polo-like kinases, whichinclude Plk, Snk (for serum-inducible kinase, also designated Plk2) and Fnk(for FGF-inducible kinase, also designated Plk3 or PRK), play a role in cellproliferation. Plk protein accumulates in the cell during S and G2 phases ofthe cell cycle, and both protein content and catalytic activity peak at theonset of mitosis, followed by a rapid reduction after mitosis. Snk and Fnk are immediate-early response genes that are first expressed during G1 phase. SNK may play a role in the division of at least some cell types, such as fibroblasts, and could function in embryogenesis, wound healing or neoplasia SNK mRNA is rapidly induced in human lung fibroblasts upon reintroduction of serum following 36 hours of serum deprivation.

PLK2 (SNK) Antibody (C-term) Blocking peptide - References

Strausberg, R.L., et al., Proc. Natl. Acad. Sci. U.S.A. 99(26):16899-16903 (2002).Liby, K., et al., DNA Seq. 11:527-533 (2001).