

SPHK1 Antibody (C-term R414) Blocking Peptide

Synthetic peptide Catalog # BP7237b

Specification

SPHK1 Antibody (C-term R414) Blocking Peptide - Product Information

Primary Accession Q9NYA1
Other Accession Q8N632

SPHK1 Antibody (C-term R414) Blocking Peptide - Additional Information

Gene ID 8877

Other Names

Sphingosine kinase 1, SK 1, SPK 1, SPHK1, SPHK, SPK

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.

SPHK1 Antibody (C-term R414) Blocking Peptide - Protein Information

Name SPHK1 (HGNC:11240)

Function

Catalyzes the phosphorylation of sphingosine to form sphingosine 1-phosphate (SPP), a lipid mediator with both intra- and extracellular functions. Also acts on D-erythro-sphingosine and to a lesser extent sphinganine, but not other lipids, such as D,L-threo- dihydrosphingosine, N,N-dimethylsphingosine, diacylglycerol, ceramide, or phosphatidylinositol (PubMed:20577214, PubMed:23602659, PubMed:29662056, PubMed:24929359, PubMed:11923095). In contrast to proapoptotic SPHK2, has a negative effect on intracellular ceramide levels, enhances cell growth and inhibits apoptosis (PubMed: 16118219). Involved in the regulation of inflammatory response and neuroinflammation. Via the product sphingosine 1-phosphate, stimulates TRAF2 E3 ubiquitin ligase activity, and promotes activation of NF- kappa-B in response to TNF signaling leading to IL17 secretion (PubMed: 20577214). In response to TNF and in parallel to NF-kappa-B activation, negatively regulates RANTES induction through p38 MAPK signaling pathway (PubMed: <a



href="http://www.uniprot.org/citations/23935096" target=" blank">23935096). Involved in endocytic membrane trafficking induced by sphingosine, recruited to dilate endosomes, also plays a role on later stages of endosomal maturation and membrane fusion independently of its kinase activity (PubMed: 28049734, PubMed:24929359). In Purkinje cells, seems to be also involved in the regulation of autophagosome-lysosome fusion upon VEGFA (PubMed:25417698).

Cellular Location

Cytoplasm. Nucleus. Cell membrane. Endosome membrane; Peripheral membrane protein. Membrane, clathrin-coated pit. Synapse {ECO:0000250|UniProtKB:Q8CI15} Note=Translocated from the cytoplasm to the plasma membrane in a CIB1- dependent manner (PubMed:19854831). Binds to membranes containing negatively charged lipids but not neutral lipids (PubMed:24929359) Recruited to endocytic membranes by sphingosine where promotes membrane fusion (By similarity). {ECO:0000250|UniProtKB:Q8CI15, ECO:0000269|PubMed:19854831, ECO:0000269|PubMed:24929359}

Tissue Location

Widely expressed with highest levels in adult liver, kidney, heart and skeletal muscle. Expressed in brain cortex (at protein level) (PubMed:29662056).

SPHK1 Antibody (C-term R414) Blocking Peptide - Protocols

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

• Blocking Peptides

SPHK1 Antibody (C-term R414) Blocking Peptide - Images

SPHK1 Antibody (C-term R414) Blocking Peptide - Background

SPHK1 is a novel lipid messenger with both intracellular and extracellular functions. Intracellularly, it regulates proliferation and survival, and extracellularly, it is a ligand for EDG1 (MIM 601974). Various stimuli increase cellular levels of SPP by activation of sphingosine kinase (SPHK), the enzyme that catalyzes the phosphorylation of sphingosine. Competitive inhibitors of SPHK block formation of SPP and selectively inhibit cellular proliferation induced by a variety of factors, including platelet-derived growth factor (e.g., MIM 173430) and serum.

SPHK1 Antibody (C-term R414) Blocking Peptide - References

Hengst, J.A., et al. Arch. Biochem. Biophys. 494(1):23-31(2010) Jarman, K.E., et al. J. Biol. Chem. 285(1):483-492(2010)Gamble, J.R., et al. Am. J. Pathol. 175(5):2217-2225(2009)