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VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide

Synthetic peptide
Catalog # BP7645b

Specification

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Product Information

Primary Accession P35916

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Additional Information

Gene ID 2324

Other Names
Vascular endothelial growth factor receptor 3, VEGFR-3, Fms-like tyrosine kinase 4, FLT-4, Tyrosine-protein kinase receptor FLT4, FLT4, VEGFR3

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7645b was selected from the C-term region of human FLT4 . 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.

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Protein Information

Name FLT4

Synonyms VEGFR3

Function
Tyrosine-protein kinase that acts as a cell-surface receptor for VEGFC and VEGFD, and plays an essential role in adult lymphangiogenesis and in the development of the vascular network and the cardiovascular system during embryonic development. Promotes proliferation, survival and migration of endothelial cells, and regulates angiogenic sprouting. Signaling by activated FLT4 leads to enhanced production of VEGFC, and to a lesser degree VEGFA, thereby creating a positive feedback loop that enhances FLT4 signaling. Modulates KDR signaling by forming heterodimers. The secreted isoform 3 may function as a decoy receptor for VEGFC and/or VEGFD and play an important role as a negative regulator of VEGFC-mediated lymphangiogenesis and angiogenesis. Binding of vascular growth factors to isoform 1 or isoform 2 leads to the activation of several

signaling cascades; isoform 2 seems to be less efficient in signal transduction, because it has a truncated C-terminus and therefore lacks several phosphorylation sites. Mediates activation of the MAPK1/ERK2, MAPK3/ERK1 signaling pathway, of MAPK8 and the JUN signaling pathway, and of the AKT1 signaling pathway. Phosphorylates SHC1. Mediates phosphorylation of PIK3R1, the regulatory subunit of phosphatidylinositol 3-kinase. Promotes phosphorylation of MAPK8 at 'Thr-183' and 'Tyr-185', and of AKT1 at 'Ser-473'.

Cellular Location
Cell membrane; Single-pass type I membrane protein Cytoplasm Nucleus. Note=Ligand-mediated autophosphorylation leads to rapid internalization [Isoform 2]: Cell membrane; Single-pass type I membrane protein

Tissue Location
Detected in endothelial cells (at protein level). Widely expressed. Detected in fetal spleen, lung and brain. Detected in adult liver, muscle, thymus, placenta, lung, testis, ovary, prostate, heart, and kidney.

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Images

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - Background

FLT4, a member of the CSF-1/PDGF receptor subfamily of Tyr protein kinases, is a receptor for VEGFC. This Type I membrane protein is expressed in placenta, lung, heart, and kidney, but does not seem to be expressed in pancreas and brain. Defects in FLT4 are the cause of hereditary lymphedema I, also known as Nonne-Milroy lymphedema or Milroy disease. Hereditary lymphedema is a chronic disabling condition which results in swelling of the extremities due to altered lymphatic flow. Patients with lymphedema suffer from recurrent local infections and physical impairment. Hereditary lymphedema I shows autosomal dominant inheritance and is characterized by onset usually at birth. Defects in FLT4 are also found in juvenile hemangioma. Juvenile hemangiomas are the most common tumors of infancy, occurring as many as 10/% of all births. These benign vascular lesions enlarge rapidly during the first year of life by hyperplasia of endothelial cells and attendant pericytes, and then spontaneously involute over a period of years, leaving loose fibrofatty tissue.

VEGFR-3 (FLT4) Antibody (C-term) Blocking peptide - References

Walter, J.W., et al., Genes Chromosomes Cancer 33(3):295-303 (2002).Karkkainen, M.J., et al., Nat. Genet. 25(2):153-159 (2000).Irrthum, A., et al., Am. J. Hum. Genet. 67(2):295-301 (2000).Ferrell, R.E., et al., Hum. Mol. Genet. 7(13):2073-2078 (1998).Galland, F., et al., Oncogene 8(5):1233-1240 (1993).