10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide

Synthetic peptide
Catalog # BP3107a

Specification

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Product Information

Primary Accession P35968

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Additional Information

Gene ID 3791

Other Names
Vascular endothelial growth factor receptor 2, VEGFR-2, Fetal liver kinase 1, FLK-1, Kinase insert domain receptor, KDR, Protein-tyrosine kinase receptor flk-1, CD309, KDR, FLK1, VEGFR2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP3107a was selected from the 992-1001 <CR>region of human Phospho-KDR/FLK1-Y996. 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.

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Protein Information

Name KDR (HGNC:6307)

Synonyms FLK1, VEGFR2

Function
Tyrosine-protein kinase that acts as a cell-surface receptor for VEGFA, VEGFC and VEGFD. Plays an essential role in the regulation of angiogenesis, vascular development, vascular permeability, and embryonic hematopoiesis. Promotes proliferation, survival, migration and differentiation of endothelial cells. Promotes reorganization of the actin cytoskeleton. Isoforms lacking a transmembrane domain, such as isoform 2 and isoform 3, may function as decoy receptors for VEGFA, VEGFC and/or VEGFD. Isoform 2 plays an important role as negative regulator of VEGFA- and VEGFC-mediated lymphangiogenesis by limiting the amount of free VEGFA and/or VEGFC and preventing their binding to FLT4. Modulates FLT1 and FLT4 signaling by forming heterodimers. Binding of vascular growth factors to isoform 1 leads to the activation of several signaling

cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate and the activation of protein kinase C. Mediates activation of MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. Mediates phosphorylation of PIK3R1, the regulatory subunit of phosphatidylinositol 3-kinase, reorganization of the actin cytoskeleton and activation of PTK2/FAK1. Required for VEGFA-mediated induction of NOS2 and NOS3, leading to the production of the signaling molecule nitric oxide (NO) by endothelial cells. Phosphorylates PLCG1. Promotes phosphorylation of FYN, NCK1, NOS3, PIK3R1, PTK2/FAK1 and SRC.

Cellular Location
Cell junction. Endoplasmic reticulum. Cell membrane. Note=Localized with RAP1A at cell-cell junctions (By similarity). Colocalizes with ERN1 and XBP1 in the endoplasmic reticulum in endothelial cells in a vascular endothelial growth factor (VEGF)-dependent manner (PubMed:23529610). {ECO:0000250, ECO:0000269|PubMed:23529610} [Isoform 2]: Secreted.

Tissue Location
Detected in cornea (at protein level). Widely expressed.

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Protocols

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

- Blocking Peptides

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Images

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - Background

KDR (FLK1) is a receptor for VEGF or VEGFC. This protein has a tyrosine-protein kinase activity. The VEGF-kinase ligand/receptor signaling system plays a key role in vascular development and regulation of vascular permeability.

Phospho-KDR/FLK1(Y996) Antibody Blocking peptide - References

Le Boeuf, F., et al., J. Biol. Chem. 279(37):39175-39185 (2004).Lee, Y.K., et al., Blood 104(3):788-794 (2004).Sulpice, E., et al., Eur. J. Biochem. 271(16):3310-3318 (2004).Murdaca, J., et al., J. Biol. Chem. 279(25):26754-26761 (2004).List, A.F., et al., Exp. Hematol. 32(6):526-535 (2004).