

KDR antibody - N-terminal region

Rabbit Polyclonal Antibody Catalog # Al16219

Specification

KDR antibody - N-terminal region - Product Information

Application WB, IHC Primary Accession P35968

Other Accession NM 002253, NP 002244

Reactivity
Predicted
Human, Pig, Horse, Bovine, Dog
Human, Pig, Horse, Bovine, Dog

Host Rabbit
Clonality Polyclonal
Calculated MW 151kDa KDa

KDR antibody - N-terminal region - Additional Information

Gene ID 3791

Alias Symbol CD309, FLK1, VEGFR, VEGFR2

Other Names

Vascular endothelial growth factor receptor 2, VEGFR-2, 2.7.10.1, Fetal liver kinase 1, FLK-1, Kinase insert domain receptor, KDR, Protein-tyrosine kinase receptor flk-1, CD309, KDR, FLK1, VEGFR2

Format

Liquid. Purified antibody supplied in 1x PBS buffer with 0.09% (w/v) sodium azide and 2% sucrose.

Reconstitution & Storage

Add 50 ul of distilled water. Final anti-KDR antibody concentration is 1 mg/ml in PBS buffer with 2% sucrose. For longer periods of storage, store at 20°C. Avoid repeat freeze-thaw cycles.

Precautions

KDR antibody - N-terminal region is for research use only and not for use in diagnostic or therapeutic procedures.

KDR antibody - N-terminal region - 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

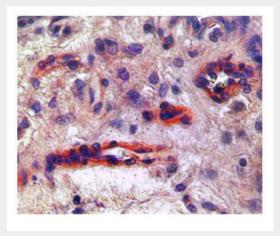
Detected in cornea (at protein level). Widely expressed.

KDR antibody - N-terminal region - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- <u>Immunoprecipitation</u>
- Flow Cytomety
- Cell Culture

KDR antibody - N-terminal region - Images



KDR in endothelial cells in blood vessale in placenta was detected using HRP/AEC red color stain.recommended for IHC on human tissue .5-10 μ g/ml





WB Suggested Anti-KDR Antibody Titration: 0.2-1 μg/ml

ELISA Titer: 1:62500

Positive Control: Human Lung

KDR antibody - N-terminal region - Background

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KDR antibody - N-terminal region - References

Jin P., et al. Arthritis Res. Ther. 10:R73-R73(2008). Albuquerque R.J., et al. Nat. Med. 15:1023-1030(2009). Yin L.Y., et al. Submitted (DEC-1997) to the EMBL/GenBank/DDBJ databases. Yu Y., et al. Submitted (MAY-1998) to the EMBL/GenBank/DDBJ databases. Hillier L.W., et al. Nature 434:724-731(2005).