PRODUCT INFORMATION

VEGFR2 Extracellular Domain (human, recombinant; aa 20-327)

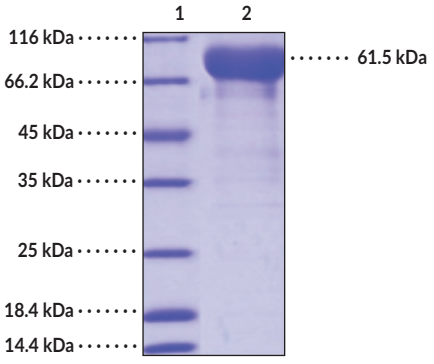
Item No. 31845

Overview and Properties

Synonyms:	CD309, Fetal Liver Kinase 1, FLK-1, KDR, Kinase Insert Domain Receptor, Vascular Endothelial Growth Factor Receptor 2
Source:	Active recombinant C-terminal human IgG1 Fc-tagged VEGFR2 expressed in HEK293 cells
Amino Acids:	20-327
Uniprot No.:	P35968
Molecular Weight:	61.5 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	***batch specific*** (≥95% estimated by SDS-PAGE)
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/µg, determined by the LAL endotoxin assay
Protein Concentration:	***batch specific*** mg/ml
Bioactivity:	See figures for details

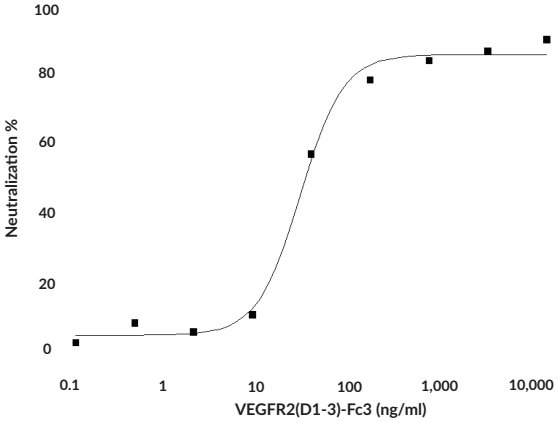
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: VEGFR2

SDS-PAGE Analysis of VEGFR2 . This protein has a calculated molecular weight of 61.5 kDa.



Ability of VEGFR to inhibit VEGF-dependent proliferation of human umbilical vein endothelial cells (HUVEC) in the presence of 10 ng/mL rhVEGF165. The ED_{50} value for this effect is 40-120 ng/mg.

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Description

VEGFR2, also known as kinase insert domain receptor (KDR), is a receptor tyrosine kinase that has roles in vascular permeability, cell proliferation and survival, and angiogenesis.[1] It is composed of an N-terminal extracellular ligand-binding domain, which contains seven immunoglobulin-like (Ig-like) domains, a transmembrane domain, and an intracellular tyrosine kinase domain, which is subject to phosphorylation and contains a kinase insert domain and the C-terminal domain.[1,2] VEGFR2 is expressed primarily in vascular and lymphatic endothelial cells, where it is bound by the growth factors VEGF-A, VEGF-C, and VEGF-D.[1] Upon ligand binding, VEGFR2 forms homodimers or heterodimers with VEGFR1 or VEGFR3, resulting in VEGFR2 phosphorylation and activation of a variety of intracellular signaling pathways, including the ERK/MAPK pathway, to promote endothelial cell survival, proliferation, and migration. VEGFR2 also exists in a soluble form that is generated by alternative splicing of the *KDR* pre-mRNA or proteolytic shedding and decreases angiogenesis by functioning as a decoy receptor for VEGF-A.[3,4] VEGFR2 expression is increased in the tumor vasculature of patients with a variety of solid tumors, including colorectal, lung, pancreatic, and breast cancer.[5] Cayman's VEGFR2 Extracellular Domain (human, recombinant; aa 20-327) protein can be used for enzyme activity assays. This protein is a disulfide-linked homodimer. The reduced monomer, composed of VEGFR2 (amino acids 20-327) fused to human IgG1 Fc at its C-terminus, consists of 549 amino acids, has a calculated molecular weight of 61.5 kDa, and a predicted N terminus of Ala20 after signal peptide cleavage. As a result of glycosylation, the monomer migrates at greater than 61.5 kDa by SDS-PAGE under reducing conditions.

References

1. Koch, S. and Claesson-Welsh, L. Signal transduction by vascular endothelial growth factor receptors. *Cold Spring Harb. Perspect. Med.* **2(7)**, a006502 (2012).
2. Park, S.A., Jeon, M.S., Ha, K.-T., *et al.* Structure and function of vascular endothelial growth factor and its receptor system. *BMB Rep.* **51(2)**, 73-78 (2018).
3. Bowler, E. and Oltean, S. Alternative splicing in angiogenesis. *Int. J. Mol. Sci.* **20(9)**, 2067 (2019).
4. Stevens, M. and Oltean, S. Modulation of receptor tyrosine kinase activity through alternative splicing of ligands and receptors in the VEGF-A/VEGFR axis. *Cells* **8(4)**, 288 (2019).
5. Smith, N.R., Baker, D., James, N.H., *et al.* Vascular endothelial growth factor receptors VEGFR-2 and VEGFR-3 are localized primarily to the vasculature in human primary solid cancers. *Clin. Cancer Res.* **16(14)**, 3548-3561 (2010).

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