

Human VEGFR2/KDR/Flk-1 Antibody

Monoclonal Mouse IgG₁ Clone # 89106 Catalog Number: MAB3572

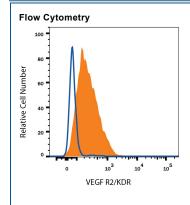
DESCRIPTION	
Species Reactivity	Human
Specificity	Detects human VEGFR2/KDR/Flk-1 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human (rh) VEGFR1, rhVEGFR3 or recombinant mouse VEGFR2 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 89106
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	S. frugiperda insect ovarian cell line Sf 21-derived recombinant human VEGFR2/KDR/Flk-1 Ala20-Glu764 Accession # P35968
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

APPLICATIONS

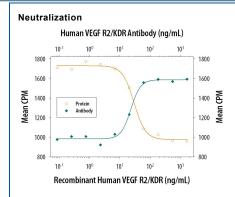
Please Note: Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.

	Recommended Sample Concentration
Flow Cytometry	0.25 μg/10 ⁶ cells See Below
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.
Neutralization	Measured by its ability to neutralize VEGFR2/KDR/Flk-1-mediated inhibition of proliferation in HUVEC human umbilical vein endothelial cells. The Neutralization Dose (ND ₅₀) is typically 10-50 ng/mL in the presence of 50 ng/mL Recombinant Human VEGFR2/KDR/Flk-1 Fc Chimera and 10 ng/mL Recombinant Human VEGF ₁₆₅ .

DATA



Detection of VEGFR2/KDR/FIk-1 in HUVEC Human Cells by Flow Cytometry. HUVEC human umbilical vein endothelial cells were stained with Mouse Anti-Human VEGFR2/KDR/Fik-1 Monoclonal Antibody (Catalog # MAB3572, filled histogram) or isotype control antibody (Catalog # MAB002, open histogram), followed by Phycoerythrin-conjugated Anti-Mouse IgG Secondary Antibody (Catalog # F0102B).



VEGFR2/KDR Inhibition of VEGF-dependent Cell Proliferation and Neutralization by Human VEGFR2/KDR Antibody. Recombinant Human VEGFR2/KDR Fc Chimera (Catalog # 357-KD) inhibits Recombinant Human VEGF₁₆₅ (Catalog # 293-VE) induced proliferation in HUVEC human umbilical vein endothelial cells in a dosedependent manner (orange line). Inhibition of Recombinant Human VEGF₁₆₅ (10 ng/mL) activity elicited by Recombinant Human VEGFR2/KDR Fc Chimera (50 ng/mL) is neutralized (green line) by increasing concentrations of Mouse Anti-Human VEGFR2/KDR Monoclonal Antibody (Catalog # MAB3572). The ND_{50} is

typically 10-50 ng/mL.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 0.5 mg/mL in sterile PBS.

Shipping

The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

*Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C

Stability & Storage L

Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 6 months, -20 to -70 °C under sterile conditions after reconstitution

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BACKGROUND

VEGFR2 (KDR/Flk-1), VEGFR1 (Flt-1) and VEGFR3 (Flt-4) belong to the class III subfamily of receptor tyrosine kinases (RTKs). All three receptors contain seven immunoglobulin-like repeats in their extracellular domains and kinase insert domains in their intracellular regions. The expression of VEGFR1, 2, and 3 is almost exclusively restricted to the endothelial cells. These receptors are likely to play essential roles in vasculogenesis and angiogenesis. Mature VEGFR2 is composed of a 745 aa extracellular domain, a 25 aa transmembrane domain and a 567 aa cytoplasmic domain. In contrast to VEGFR1 which binds both P/GF and VEGF with high affinity, VEGFR2 binds VEGF but not P/GF with high affinity. The recombinant soluble VEGFR2/Fc chimera binds VEGF with high affinity and is a potent VEGF antagonist.

References:

1. Ferra, N. and R. Davis-Smyth (1997) Endocrine Reviews 18:4.

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