

Flk-1 (C-1158): sc-504

BACKGROUND

Three cell membrane receptor tyrosine kinases, Flt (also designated VEGF-R1), Flk-1 (also designated VEGF-R2) and Flt-4, putatively involved in the growth of endothelial cells, are characterized by the presence of seven immunoglobulin-like sequences in their extracellular domain. These receptors exhibit high degrees of sequence relatedness to each other as well as lesser degrees of relatedness to the class III receptors including CSF-1/Fms, PDGR, SLFR/Kit and Flt-3/Flk-2. Two members of this receptor class, Flt-1 and Flk-1, have been shown to represent high affinity receptors for vascular endothelial growth factors (VEGFs). On the basis of structural similarity to Flt and Flk-1, it has been speculated that Flt-4 might represent a third receptor for either VEGF or a VEGF-related ligand.

CHROMOSOMAL LOCATION

Genetic locus: KDR (human) mapping to 4q12; Kdr (mouse) mapping to 5 C3.3.

SOURCE

Flk-1 (C-1158) is a rabbit polyclonal antibody raised against amino acids 1158-1345 of Flk-1 of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Available as agarose conjugate for immunoprecipitation, sc-504 AC, 500 µg/0.25 ml agarose in 1 ml.

APPLICATIONS

Flk-1 (C-1158) is recommended for detection of Flk-1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Flk-1 siRNA (h): sc-29318, Flk-1 siRNA (m): sc-35390, Flk-1 shRNA Plasmid (h): sc-29318-SH, Flk-1 shRNA Plasmid (m): sc-35390-SH, Flk-1 shRNA (h) Lentiviral Particles: sc-29318-V and Flk-1 shRNA (m) Lentiviral Particles: sc-35390-V.

Molecular Weight of immature Flk-1: 150 kDa.

Molecular Weight of intermediate glycosylated Flk-1: 200 kDa.

Molecular Weight of mature glycosylated Flk-1: 230 kDa.

Positive Controls: ECV304 cell lysate: sc-2269, Flk-1 (m): 293T Lysate: sc-120289 or c4 whole cell lysate: sc-364186.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

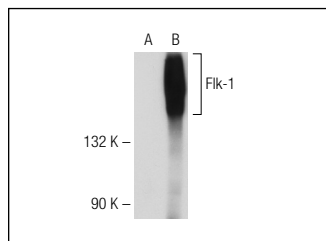
PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

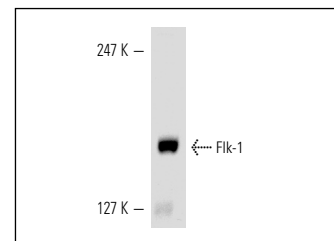
STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



Flk-1 (C-1158): sc-504. Western blot analysis of Flk-1 expression in non-transfected: sc-117752 (A) and mouse Flk-1 transfected: sc-120289 (B) 293T whole cell lysates.



Flk-1 (C-1158): sc-504. Western blot analysis of Flk-1 expression in c4 whole cell lysate.

SELECT PRODUCT CITATIONS

- Daniel, T.O., et al. 1996. ELK and LERK-2 in developing kidney and microvascular endothelial assembly. *Kidney Int. Suppl.* 57: S73-S81.
- Ugarte-Berzal, E., et al. 2010. VEGF/VEGFR2 interaction down-regulates matrix metalloproteinase-9 via STAT1 activation and inhibits B chronic lymphocytic leukemia cell migration. *Blood* 115: 846-849.
- Palmieri, D., et al. 2010. Osteoblasts extracellular matrix induces vessel like structures through glycosylated collagen I. *Exp. Cell Res.* 316: 789-799.
- Gorbunova, E., et al. 2010. Pathogenic hantaviruses Andes virus and Hantaan virus induce adherens junction disassembly by directing vascular endothelial cadherin internalization in human endothelial cells. *J. Virol.* 84: 7405-7411.
- Herzog, B., et al. 2011. VEGF binding to NRP1 is essential for VEGF stimulation of endothelial cell migration, complex formation between NRP1 and VEGFR2, and signaling via FAK Tyr407 phosphorylation. *Mol. Biol. Cell* 22: 2766-2776.
- Anselmi, F., et al. 2012. c-Abl modulates MAP kinases activation downstream of VEGFR-2 signaling by direct phosphorylation of the adaptor proteins GRB2 and NCK1. *Angiogenesis* 15: 187-197.
- Albrecht-Schgoer, K., et al. 2012. The angiogenic factor secretoneurin induces coronary angiogenesis in a model of myocardial infarction by stimulation of vascular endothelial growth factor signaling in endothelial cells. *Circulation* 126: 2491-2501.

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