

p-Flk-1 (Tyr 996)-R: sc-16629-R

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 homology to each other as well as lesser degrees of homology 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). In response to VEGF binding, Flk-1 undergoes auto-phosphorylation in the kinase insert domain on Tyr 951 and Tyr 996 and in the tyrosine kinase catalytic domain on Tyr 1054 and Tyr 1059. Upon activation, Flk-1 recruits several adapter proteins, including Shc, GRB2, Nck and protein tyrosine phosphatases SHP-1 and SHP-2. The mediation of VEGF signaling by Flk-1 promotes proliferation, chemotaxis, prouting and angiogenesis.

CHROMOSOMAL LOCATION

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

SOURCE

p-Flk-1 (Tyr 996)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing phosphorylated Tyr 996 of Flk-1 of human origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-16629 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-Flk-1 (Tyr 996)-R is recommended for detection of Tyr 996 phosphorylated Flk-1 of human origin, correspondingly phosphorylated Tyr 994 Flk-1 of mouse origin and correspondingly phosphorylated Tyr 992 Flk-1 of rat 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 p-Flk-1: 150 kDa.

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

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

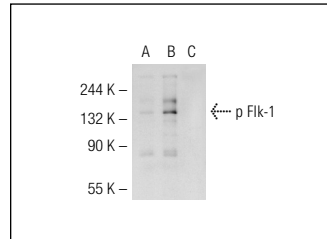
STORAGE

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

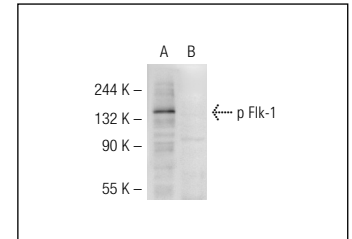
RESEARCH USE

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

DATA



p-Flk-1 (Tyr 996)-R: sc-16629-R. Western blot analysis of Flk-1 phosphorylation in non-transfected: sc-117752 (A), untreated mouse Flk-1 transfected: sc-120289 (B) and lambda protein phosphatase (sc-200312A) treated mouse Flk-1 transfected: sc-120289 (C) 293T whole cell lysates.



p-Flk-1 (Tyr 996)-R: sc-16629-R. Western blot analysis of Flk-1 phosphorylation in untreated (A) and lambda protein phosphatase (sc-200312A) treated (B) SK-OV-3 whole cell lysates.

SELECT PRODUCT CITATIONS

1. Yoo, M.H., et al. 2005. Riluzole inhibits VEGF-induced endothelial cell proliferation *in vitro* and hyperoxia-induced abnormal vessel formation *in vivo*. *Invest. Ophthalmol. Vis. Sci.* 46: 4780-4787.
2. Guan, F., et al. 2006. Autocrine VEGF-A system in podocytes regulates podocin and its interaction with CD2AP. *Am. J. Physiol. Renal Physiol.* 291: F422-F428.
3. Jesmin, S., et al. 2007. Endothelin antagonism normalizes VEGF signaling and cardiac function in STZ-induced diabetic rat hearts. *Am. J. Physiol. Endocrinol. Metab.* 292: E1030-E1040.
4. Tufro, A., et al. 2007. Crosstalk between VEGF-A/VEGFR2 and GDNF/RET signaling pathways. *Biochem. Biophys. Res. Commun.* 358: 410-416.
5. Lange, A., et al. 2009. Detergent fractionation with subsequent subtractive suppression hybridization as a tool for identifying genes coding for plasma membrane proteins. *Exp. Dermatol.* 18: 527-535.
6. Sörensen, I., et al. 2009. DLL1-mediated Notch activation regulates endothelial identity in mouse fetal arteries. *Blood* 113: 5680-5688.
7. Sinha, S., et al. 2009. Dopamine regulates phosphorylation of VEGF receptor 2 by engaging Src-homology-2-domain-containing protein tyrosine phosphatase 2. *J. Cell Sci.* 122: 3385-3392.
8. Verma, A., et al. 2010. Endothelial cell-specific chemotaxis receptor (ecscr) promotes angioblast migration during vasculogenesis and enhances VEGF receptor sensitivity. *Blood* 115: 4614-4622.
9. Byeon, S.H., et al. 2010. Vascular endothelial growth factor as an autocrine survival factor for retinal pigment epithelial cells under oxidative stress via the VEGF-R2/PI3K/Akt. *Invest. Ophthalmol. Vis. Sci.* 51: 1190-1197.

PROTOCOLS

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