SANTA CRUZ BIOTECHNOLOGY, INC.

# p-Flk-1 (Tyr 951): sc-16628

SANTA CRUZ BIOTECHNOLOGY

*The Power to Question*

## 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 951) is available as either goat (sc-16628) or rabbit (sc-16628-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing Tyr 951 phosphorylated of Flk-1 human origin.

## PRODUCT

Each vial contains either 100 µg (sc-16628) or 200 µg (sc-16628)-R IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-16628 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## APPLICATIONS

p-Flk-1 (Tyr 951) is recommended for detection of Tyr 951 phosphorylated Flk-1 of human origin, correspondingly phosphorylated Tyr 949 Flk-1 of mouse origin and correspondingly phosphorylated Tyr 947 Flk-1of 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.

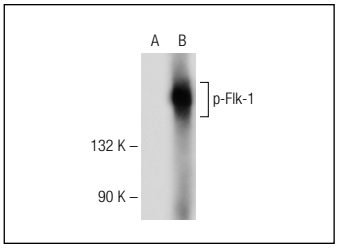
## RESEARCH USE

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

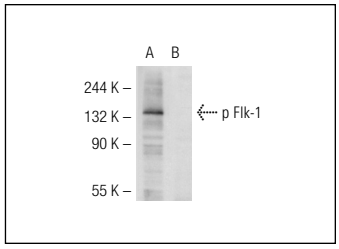
## STORAGE

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

## DATA




p-Flk-1 (Tyr 951)-R: sc-16628-R. Western blot analysis of Flk-1 phosphorylation in non-transfected: sc-117752 (**A**) and mouse Flk-1 transfected: sc-120289 (**B**) 293T whole cell lysates.




p-Flk-1 (Tyr 951)-R: sc-16628-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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4. Iacob, D., et al. 2008. Decorin-mediated inhibition of proliferation and migration of the human trophoblast via different tyrosine kinase receptors. Endocrinology 149: 6187-6197.
5. 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.
6. Smith, V.E., et al. 2009. A novel mechanism of sodium iodide symporter repression in differentiated thyroid cancer. J. Cell Sci. 122: 3393-3402.
7. Trinh, X.B., et al. 2009. The VEGF pathway and the AKT/mTOR/p70S6K1 signalling pathway in human epithelial ovarian cancer. Br. J. Cancer 100: 971-978.
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9. Ling, Y., et al. 2011. Baicalein potently suppresses angiogenesis induced by vascular endothelial growth factor through the p53/Rb signaling pathway leading to $G_1$/S cell cycle arrest. Exp. Biol. Med. 236: 851-858.