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VEGFB

Reactivity: Human Mouse Rat

Tested applications:WB IHC

Recommended Dilution:WB 1:500 - 1:2000 IHC 1:50 - 1:200

Calculated MW:19kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human VEGFB

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Concentration:

b

Synonym:

VEGFB; VEGFL; VRF; Vascular endothelial growth factor B; VEGF-related factor ;

Catalog #:A2132

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID:7423

Isotype: IgG

Swiss Prot:P49765

Purity: Affinity purification

For research use only.

Background:

Vascular endothelial growth factor (VEGF) is a highly specific mitogen for vascular endothelial cells. VEGF and its close relatives VEGF-B, -C and -D form a subfamily within PDGF family of growth factors, which belongs to the cysteine knot class of cytokines. Five VEGF isoforms of 121, 145, 165, 189 and 206 amino acids (VEGF121206) are generated as a result of alternative splicing from a single VEGF gene (1).The various VEGF forms bind to three tyrosine-kinase receptors, VEGFR-1, VEGFR-2 and VEGFR-3 which are expressed almost exclusively in endothelial cells. VEGFR-2 is the main angiogenic signal transducer for VEGF, while VEGFR-3 is specific for VEGF-C and -D and is necessary and sufficient for lymphangiogenic signaling. However, upon proteolytic processing VEGF-C and -D gain the ability to also bind and activate VEGFR-2 (2). Guided by the binding properties of the ligands, the VEGFRs are able to form both homodimers and heterodimers. Receptor dimerization is accompanied by activation of receptor kinase activity leading to receptor autophosphorylation. Phosphorylated receptors recruit interacting proteins and induce downstream signaling (3). Recently, tumor therapies based on neutralizing anti-VEGF antibodies and small molecule tyrosine kinase inhibitors targeting VEGFRs have been developed. These new strategies for tumor treatment show the clinical relevance of inhibiting VEGF signal transduction pathways that are exaggerated in pathological angiogenesis (4).

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