

Recombinant Human VEGFR2/Flk-1/KDR Protein (His &GSTTag)

Catalog Number: PKSH030427

Note: Centrifuge before opening to ensure complete recovery of vial contents.

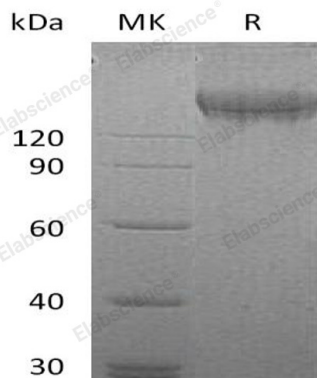
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human VEGFR2/Flk-1/KDR protein Asp807-Val1356, with an N-terminal His & GST
Mol_Mass	89.3 kDa
Accession	NP_002244
Bio-activity	The specific activity was determined to be 10 nmol/min/mg using Poly(Glu, Tyr) 4:1 as substrate.

Properties

Purity	> 78 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < -20°C.
Formulation	Supplied as sterile solution of 50mM Tris, 100mM NaCl, pH 8.0, 10% glycerol, 2mM GSH
Reconstitution	Not Applicable

Data



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Background

For Research Use Only

Toll-free: 1-888-852-8623
Web: www.elabscience.com

Tel: 1-832-243-6086
Email: techsupport@elabscience.com

Fax: 1-832-243-6017

Rev. V3.4

VEGFR2, also called as KDR or Flk-1, is identified as the receptor for VEGF and VEGFC and an early marker for endothelial cell progenitors, whose expression is restricted to endothelial cells in vivo. VEGFR2 was shown to be the primary signal transducer for angiogenesis and the development of pathological conditions such as cancer and diabetic retinopathy. It has been shown that VEGFR2 is expressed mainly in the endothelial cells, and the expression is upregulated in the tumor vasculature. Thus the inhibition of VEGFR2 activity and its downstream signaling are important targets for the treatment of diseases involving angiogenesis. VEGFR2 transduces the major signals for angiogenesis via its strong tyrosine kinase activity. However, unlike other representative tyrosine kinase receptors, VEGFR2 does not use the Ras pathway as a major downstream signaling but rather uses the phospholipase C-protein kinase C pathway to signal mitogen-activated protein (MAP)-kinase activation and DNA synthesis. VEGFR2 is a direct and major signal transducer for pathological angiogenesis, including cancer and diabetic retinopathy, in cooperation with many other signaling partners; thus, VEGFR2 and its downstream signaling appear to be critical targets for the suppression of these diseases. VEGF and VEGFR2-mediated survival signaling is critical to endothelial cell survival, maintenance of the vasculature and alveolar structure and regeneration of lung tissue. Reduced VEGF and VEGFR2 expression in emphysematous lungs has been linked to increased endothelial cell death and vascular regression.

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