Recombinant Human VEGFR2/Flk-1/KDR Protein (Fc Tag)

Catalog Number: PKSH031931

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

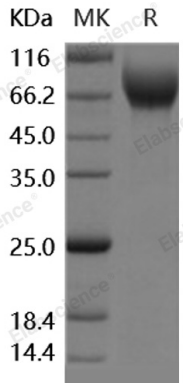
Description

Species	Human
Source	HEK293 Cells-derived Human VEGFR2/Flk-1/KDR protein Met 1-Glu 764, with an C-terminal hFc
Calculated MW	109 kDa
Observed MW	150-160 kDa
Accession	NP_002244.1
Bio-activity	1. Immobilized recombinant human VEGFA at 1. 25 μg/ml (100 μl/well) can bind VEGFR2 with a linear range of 1.25-40.0 ng/ml. 2. Measured by its ability to inhibit the VEGF-dependent proliferation of human umbilical vein endothelial cells (HUVEC). The ED_{50} for this effect is typically 20-120 ng/mL in the presence of 10 ng/mL recombinant human VEGF165.

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 90 % as determined by reducing SDS-PAGE.

Background

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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