Rat VEGFR2 / Flk-1 / CD309 / KDR Protein (Fc Tag)

Catalog Number: **80113-R02H**

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General Information

Gene Name Synonym:

CD309; Flk-1; FLK1; VEGFR; VEGFR2; 6130401C07; Flk-1; Flk1; Krd-1; Ly73; sVEGFR-2; VEGFR-2; VEGFR2

Protein Construction:

A DNA sequence encoding the rat KDR (AAH87029.1) (Met1-Glu760) was expressed with the Fc region of human IgG1 at the C-terminus.

Source: Rat

Expression Host: Human Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE

Bio Activity:

Immobilized mouse VEGF164 (Cat:50159-MNAB) at 10 μg/ml (100 μl/well) can bind rat KDR-Fc, The EC_{50} of rat KDR-Fc is 0.07-0.15 μg/ml.

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Ala 20

Molecular Mass:

The recombinant rat KDR comprises 982 amino acids and has a predicted molecular mass of 110 kDa.

Formulation:

Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

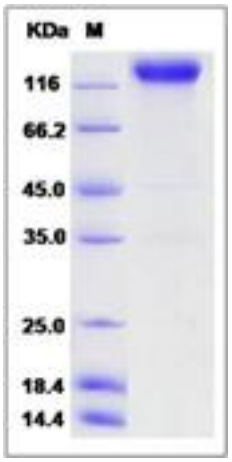
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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.

References

1.Shibuya M. (2006) Vascular endothelial growth factor (VEGF)-Receptor2: its biological functions, major signaling pathway, and specific ligand VEGF-E. Endothelium. 13(2): 63-9.
2.Marwick JA, *et al.* (2010) Cigarette smoke regulates VEGFR2-mediated survival signaling in rat lungs. J Inflamm (Lond). 7(1): 11.
3.Bruns AF, *et al.* (2010) Ligand-stimulated VEGFR2 signaling is regulated by co-ordinated trafficking and proteolysis. Traffic. 11(1): 161-74.