Active Vascular Endothelial Growth Factor Receptor 2 (VEGFR2)

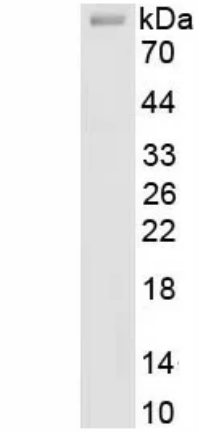
Catalog#:PHH12659

概述（Summary）

别称（Alternative Names）	CD309; FLK1; VEGFR; KDR; A Type III Receptor Tyrosine Kinase; Kinase Insert Domain Receptor; Kinase Insert Domain Receptor; Fetal Liver Kinase-1
纯度（Purity）	Greater than 97% as determined by reducing SDS-PAGE.
内毒素（Endotoxin level）	Please contact with the lab for this information
蛋白构建（Construction）	【Ala20-Glu764】
Accession #	P35968
表达宿主（Host）	293F
种属（Species）	Homo sapiens (Human)
预测分子量（Predicted MW）	84.9 kDa
制剂（Form）	Lyophilized from a 0.2 μm filtered solution of PBS, pH7.4, containing 0.01% SKL, 1mM DTT, 5% Trehalose and Proclin300.
运输方式（Shipping）	The product is shipped with ice bag. Upon receipt, store it immediately at the temperature listed below.
稳定性&储存（Stability &Storage）	12 months at -20°C to -80°C in lyophilized state.6 months at -20°C to -80°C under sterile conditions after reconstitution.7-10 days at 2°C to 8°C under sterile conditions after reconstitution.Please minimize freeze-thaw cycles.
复溶（Reconstitution）	Reconstitute in 10mM PBS (pH7.4) to a concentration of 0.1-1.0 mg/mL. Do not vortex.
背景介绍（References）	Tyrosine-protein kinase that acts as a cell-surface receptor for VEGFA, VEGFC and VEGFD. Plays an essential role in the regulation of angiogenesis, vascular development, vascular permeability, and embryonic hematopoiesis. Promotes proliferation, survival, migration and differentiation of endothelial cells. Promotes reorganization of the actin cytoskeleton. Isoforms lacking a transmembrane domain, such as isoform 2 and isoform 3, may function as decoy receptors for VEGFA, VEGFC and/or VEGFD. Isoform 2 plays an important role as negative regulator of VEGFA- and VEGFC-mediated lymphangiogenesis by limiting the

amount of free VEGFA and/or VEGFC and preventing their binding to FLT4. Modulates FLT1 and FLT4 signaling by forming heterodimers. Binding of vascular growth factors to isoform 1 leads to the activation of several signaling cascades. Activation of PLCG1 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate and the activation of protein kinase C. Mediates activation of MAPK1/ERK2, MAPK3/ERK1 and the MAP kinase signaling pathway, as well as of the AKT1 signaling pathway. Mediates phosphorylation of PIK3R1, the regulatory subunit of phosphatidylinositol 3-kinase, reorganization of the actin cytoskeleton and activation of PTK2/FAK1. Required for VEGFA-mediated induction of NOS2 and NOS3, leading to the production of the signaling molecule nitric oxide (NO) by endothelial cells. Phosphorylates PLCG1. Promotes phosphorylation of FYN, NCK1, NOS3, PIK3R1, PTK2/FAK1 and SRC.

电泳图（SDS-PAGE image）



Greater than 97% as determined by reducing SDS-PAGE.

活性图（Bioactivity image）

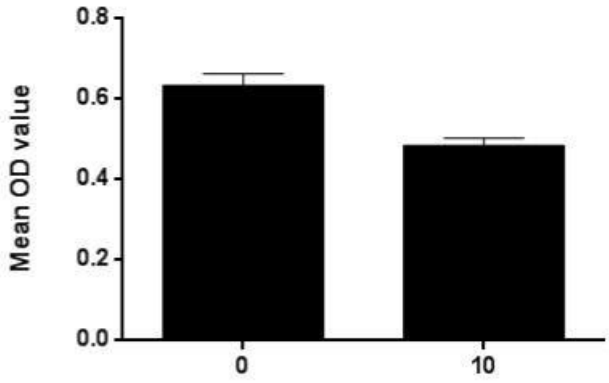
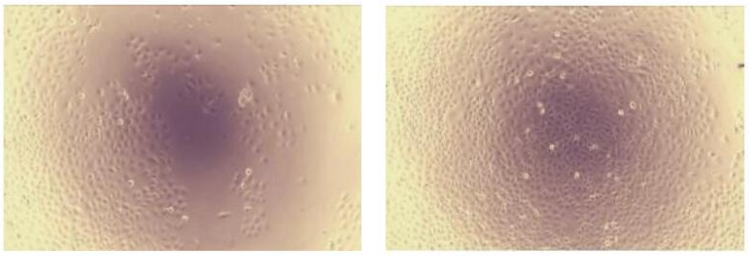


Figure. Cell proliferation of ECV-304 cells inhibit by VEGFR2. Vascular Endothelial Growth Factor Receptor 2 (VEGFR2) also known as kinase insert domain receptor acts as a cell-surface receptor for VEGFA, VEGFC and VEGFD. VEGFR2 functions as the primary mediator of vascular endothelial growth factor activation in endothelial cells. Regulation of VEGFR-2 expression appears critical in mitogenesis, differentiation, and angiogenesis. To test the effect on inhibit the VEGF-dependent proliferation of endothelium cells, ECV-304 cells were seeded into triplicate wells of 96-well plates at a density of 5,000 cells/well and allowed to attach, replaced with serum-free overnight, then the medium was replaced with 2% serum standard DMEM including 1μg/mL Vascular Endothelial Growth Factor C (VEGFC) and various concentrations of recombinant human VEGFR2. After incubated for 96h, cells were observed by inverted microscope and cell proliferation was measured by Cell Counting Kit-8 (CCK-8). Briefly, 10μL of CCK-8 solution was added to each well of the plate, then the absorbance at 450nm was measured using a microplate reader after incubating the plate for 1-4 hours at 37℃. Proliferation of ECV-304 cells after incubation with VEGFR2 for 96h observed by inverted microscope was shown in Figure 1. Cell viability was assessed by CCK-8 (Cell Counting Kit-8) assay after incubation with recombinant VEGFR2 for 96h. The result was shown in Figure 2. It was obvious that VEGFR2 significantly inhibit cell viability of ECV-304. (A) ECV-304 cells cultured in DMEM, stimulated with 10ng/mL VEGFR2 for 96h; (B) Unstimulated ECV-304 cells cultured in DMEM for 96h. Figure. VEGFR2 inhibit VEGF-dependent proliferation of ECV-304 cells.



注意事项（Note）

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