

Recombinant Human DLL4 Protein (Fc Tag)

Catalog Number:PKSH031806



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

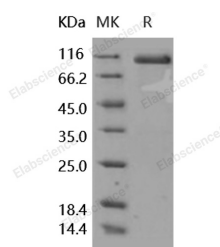
Description

Synonyms	Delta-like protein 4;Drosophila Delta homolog 4;Delta4;DLL4
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Pro 524
Accession	NP_061947.1
Calculated Molecular Weight	81.0 kDa
Observed molecular weight	100-110 kDa
Tag	C-hFc
Bioactivity	1. Immobilized human DLL4 at 10 µg/mL (100 µL/well) can bind biotinylated mouse NOTCH1-his. The EC50 of biotinylated mouse NOTCH1-his is 40 ng/mL. 2. Measured by the ability of the immobilized protein to enhance BMP2-induced alkaline phosphatase activity in C3H10T1/2 mouse embryonic fibroblast cells. The ED50 for this effect is typically 1-8 µg/mL in the presence of 500 ng/mL recombinant human BMP2.

Properties

Purity	> 95 % 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°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C 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% Tween80 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



> 95 % as determined by reducing SDS-PAGE.

Background

Delta-like protein 4 (DLL4; Delta4); a type I membrane-bound Notch ligand; is one of five known Notch ligands in mammals and interacts predominantly with Notch 1; which has a key role in vascular development. Recent studies yield

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substantial insights into the role of DLL4 in angiogenesis. DLL4 is induced by vascular endothelial growth factor (VEGF) and acts downstream of VEGF as a 'brake' on VEGF-induced vessel growth; forming an autoregulatory negative feedback loop inactivating VEGF. DLL4 is downstream of VEGF signaling and its activation triggers a negative feedback that restrains the effects of VEGF. Attenuation of DLL4/Notch signaling results in chaotic vascular network with excessive branching and sprouting. DLL4 is widely distributed in tissues other than vessels including many malignancies. Furthermore; the molecule is internalized on binding its receptor and often transported to the nucleus. In pathological conditions; such as cancer; DLL4 is up-regulated strongly in the tumour vasculature. Blockade of DLL4-mediated Notch signaling strikingly increases nonproductive angiogenesis; but significantly inhibits tumor growth in preclinical mouse models. In preclinical studies; blocking of DLL4/Notch signaling is associated with a paradoxical increase in tumor vessel density; yet causes marked growth inhibition due to functionally defective vasculature. Thus; DLL4 blockade holds promise as an additional strategy for angiogenesis-based cancer therapy.

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