Recombinant Mouse DLL4 Protein (His Tag)

Catalog Number: PKSM040570

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

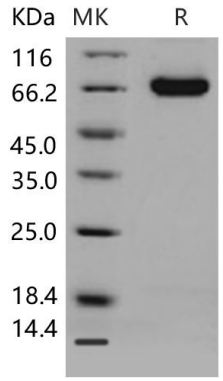
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse DLL4 protein Met 1-Pro 525, with an C-terminal His
Calculated MW	55.7 kDa
Observed MW	68 kDa
Accession	NP_062327.2
Bio-activity	1. Immobilized mouse DLL4-his at 10 μg/mL (100 μL/well) can bind biotinylated mouse NOTCH1-his. The EC_{50} of biotinylated mouse NOTCH1-his is 30 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 ED_{50} for this effect is typically 0.2-3 μg/mL.

Properties

Purity	> 96 % 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



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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 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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