Recombinant Protein Technical Manual

Recombinant Mouse DLL4 Protein (His Tag)(Active)

RPES0582

Product Data:

Product SKU: RPES0582

Size: 50µg

Species: Mouse

Expression host: HEK293 Cells

Uniprot: NP_062327.2

Protein Information:

Molecular Mass:	55.7 kDa
AP Molecular Mass:	68 kDa
Tag:	C-His
Bio-activity:	1. Measured by its binding ability in a functional ELISA. Immobilized mouse DLL4-his at 10 µg/mL (100 µL/well) can bind biotinylated mouse NOTCH1-his. The EC50 of biotinylated mouse NOTCH1-his is 30 ng/mL.2. Measured by the ability of the immobilized prot
Purity:	> 96 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage:	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℃ 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
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	Functional ELISA
Synonyms:	Delta4

Immunogen Information:

Sequence: Met 1-Pro 525

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.