Recombinant Protein Technical Manual

Recombinant Human DLL4 Protein (His Tag)(Active)

RPES1017

Product Data:

Product SKU: RPES1017

Size: 50µg

Species: Human

Expression host: HEK293 Cells

Uniprot: NP_061947.1

Protein Information:

Molecular Mass:	55.7 kDa
AP Molecular Mass:	55-70 kDa
Tag:	C-His
Bio-activity:	1. Measured by its ability to bind human Notch1 in a functional ELISA.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 20 ¦Ìg/mL in the presence of 500 ng/mL recombinant human BMP2.
Purity:	> 98 % as determined by reducing SDS-PAGE.
Endotoxin:	< 1.0 EU per µg 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:	Delta-like protein 4; Drosophila Delta homolog 4; Delta4; DLL4

Immunogen Information:

Sequence: Met 1-Pro 524

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.