Recombinant Human DLL4 Protein(Fc Tag)

Catalog Number: PDMH100304

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

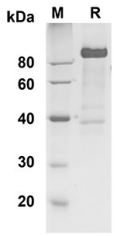
Description

Species	Human
Source	Mammalian-derived Human DLL4 proteins Ser27-Pro524,with an C-terminal Fc
Mol_Mass	79.7 kDa
Accession	Q9NR61
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg 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 a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human DLL4 proteins , 2μg/lane of Recombinant Human DLL4 proteins was resolved with SDS-PAGE under reducing conditions , showing bands at 90 KD

Background

For Research Use Only

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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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