

Recombinant Human DLL1 Protein(His Tag)

Catalog Number: PDMH100303

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

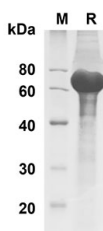
Description

Species	Human
Source	Mammalian-derived Human DLL1 proteins Ser22-Gly540, with C-terminal His
Mol_Mass	56.9 kDa
Accession	O00548
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 °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 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 DLL1 proteins , 2µg/lane of Recombinant Human DLL1 proteins was resolved with SDS-PAGE under reducing conditions , showing bands at 70 KD

Background

For Research Use Only

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Delta-like protein 1(DLL1), also known as Delta1, a single-pass type I membrane protein which contains one DSL domain and eight EGF-like domains, acts as a ligand for Notch receptors, and positively regulates T-cell development. DLL1 is proteolytically processed in a similar manner to the Notch receptor, and it has been speculated to participate in bidirectional signaling. The proteolytic processing of DLL1 helps achieve an asymmetry in Notch signaling in initially equivalent myogenic cells and helps sustain the balance between differentiation and self-renewal. Interactions between DLL1 and Notch in trans activate the Notch pathway, whereas DLL1 binding to Notch in cis inhibits Notch signaling. DLL1 undergoes proteolytic processing in its extracellular domain by ADAM10. It had been demonstrated that DLL1 represents a substrate for several other members of the ADAM family. In co-transfected cells, DLL1 is constitutively cleaved by ADAM12, and the N-terminal fragment of DLL1 is released to medium. ADAM12-mediated cleavage of DLL1 is cell density-dependent, takes place in cis orientation, and does not require the presence of the cytoplasmic domain of ADAM12. Full-length DLL1, but not its N-or C-terminal proteolytic fragment, co-immunoprecipitates with ADAM12. By using a Notch reporter construct, we show that DLL1 processing by ADAM12 increases Notch signaling in a cell-autonomous manner. Furthermore, ADAM9 and ADAM17 have the ability to process DLL1. In contrast, ADAM15 does not cleave DLL1, although the two proteins still co-immunoprecipitate with each other. During fetal development, DLL1 is an essential Notch ligand in the vascular endothelium of large arteries to activate Notch1 and maintain arterial identity. DLL1-Notch signaling was required for VEGF receptor expression in fetal arteries.

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