

Recombinant Mouse CD302/CLEC13A Protein (Fc Tag)

Catalog No. PKSM040534

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

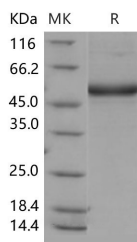
Description

Synonyms	1110055L24Rik;AI159627
Species	Mouse
Expression Host	HEK293 Cells
Sequence	Met 1-His 156
Accession	Q9DCG2-2
Calculated Molecular Weight	42.6 kDa
Observed molecular weight	48 kDa
Tag	C-hFc
Bioactivity	Not validated for activity

Properties

Purity	> 93 % 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°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 sterile PBS, pH 7.4 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 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



> 93 % as determined by reducing SDS-PAGE.

Background

CD302/CLEC13A (C-type lectin domain family 13 member A), also known as C-type lectin receptor DCL-1, is a type I transmembrane C-type lectin DCL-1/CD302. DCL-1 protein was highly conserved among the human, mouse, and rat

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orthologs. DCL-1 ectodomain contains only one CRD, whereas other type I transmembrane C-type lectins contain more than one domain (e.g. selectins and MMR). DCL-1 CP contains several putative motifs, including a Tyr-based internalization, a cluster of acidic amino acids, and Ser and Tyr phosphorylation motifs, suggesting that DCL-1 CP mediates not only endocytosis and late endosome targeting but also signaling. DCL-1 may be another cell/matrix adhesion receptor integrated in cell adhesion complexes and that DCL-1 dysfunction may affect APC adhesion and migration, causing suppression of APC function.