Recombinant Human CLEC12A/CLL-1/DCAL2 Protein (His Tag)

Catalog No. PKSH030977

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

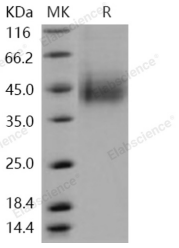
Description

Synonyms	BDCA2;CD303;CLECSF11;CLECSF7;CLL-1;CLL1;DCAL-2;DLEC;HECL;MICL;PRO34150
Species	Human
Expression Host	HEK293 Cells
Sequence	His 75-Ala 275
Accession	EAW96132.1
Calculated Molecular Weight	26 kDa
Observed molecular weight	40-45 kDa
Tag	N-His
Bioactivity	Not validated for activity

Properties

Purity	> 95 % 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℃. 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 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



> 95 % as determined by reducing SDS-PAGE.

Background

CLEC12A is a member of the C-type lectin/C-type lectin-like domain (CTL/CTLD) superfamily. Members of this family

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share a common protein fold and have diverse functions, such as cell adhesion, cell-cell signaling, glycoprotein turnover, and roles in inflammation and immune response. CLEC12A is a negative regulator of granulocyte and monocyte function. Several alternatively spliced transcript variants of this gene have been described, but the full-length nature of some of these variants has not been determined. C-type lectins are the most diverse and prevalent lectin family in immunity. Using a novel CLEC12A -specific monoclonal antibody, experiments had shown that human CLEC12A was expressed primarily on myeloid cells, including granulocytes, monocytes, macrophages, and dendritic cells. Although CLEC12A was highly N-glycosylated in primary cells, the level of glycosylation was found to vary between cell types. CLEC12A surface expression was down-regulated during inflammatory/activation conditions in vitro, as well as during an in vivo model of acute inflammation. This suggests that CLEC12A may be involved in the control of myeloid cell activation during inflammation.

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