Recombinant Protein Technical Manual

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

RPES4856

Product Data:

Product SKU: RPES4856

Size: 10µg

Species: Human

Expression host: HEK293 Cells

Uniprot: EAW96132.1

Protein Information:

Molecular Mass: 26 kDa

AP Molecular Mass: 40-45 kDa

Tag: N-His

Bio-activity:

Purity: > 95 % 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:

Synonyms: BDCA2;CD303;CLECSF11;CLECSF7;CLL;CLL1;DCAL-2;DLEC;HECL;MICL;PRO34150

Immunogen Information:

Sequence: His 75-Ala 275

Background:

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