Rhesus CLEC5A / MDL1 / MDL-1 Protein (Fc Tag)

Catalog Number: 90157-C04H

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General Information

Gene Name Synonym:

CLECSF5; MDL-1; MDL1; Clecsf5; Ly100; MDL-1; Mdl1

Protein Construction:

A DNA sequence encoding the rhesus CLEC5A (XP_001085243.1) (Pro28-Arg188) was expressed with the Fc region of mouse IgG1 at the N-terminus.

Source: Rhesus

Expression Host: Human Cells

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Asp

Molecular Mass:

The recombinant rhesus CLEC5A comprises 397 amino acids and has a calculated molecular mass of 45.3 KDa.

Formulation:

Lyophilized from sterile PBS, pH 7.4.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

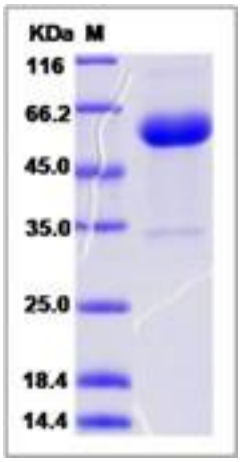
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

CLEC5A, also known as MDL1 and MDL-1, 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 signalling, glycoprotein turnover, and roles in inflammation and immune response. CLEC5A with dnax-activation protein 12 and may play a role in cell activation. It also functions as a positive regulator of osteoclastogenesis. CLEC5A acts as a key regulator of synovial injury and bone erosion during autoimmune joint inflammation .The binding of dengue virus to CLEC5A triggers signaling through the phosphylation of TYROBP, this interaction does not result in viral entry, but stimulates proinflammatory cytokine release.

References

1.Chen ST. et al., 2008, Nature. 453 (7195): 672-6.
2.Davila S. et al., 2010, Genes Immun. 11 (3): 232-8.
3.Hillier LW. et al., 2003, Nature. 424 (6945): 157-64.