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Recombinant Mouse Leukocyte Ig-Like Receptor B4/LILRB4/CD85k/ILT3 (C-Fc)

Catalog Number: PKSM041393

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

Description

Species Mouse

Source HEK293 Cells-derived Mouse LILRB4/CD85k/ILT3 protein Gly24-Lys238, with an C-

terminal Fc

Calculated MW 51.0 kDa
Observed MW 65-85 kDa
Accession Q64281

Bio-activity 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

°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.

ShippingThis product is provided as lyophilized powder which is shipped with ice packs.FormulationLyophilized from a 0.2 μm filtered solution of 50 mM Tris-HCl, 100mM Glycine, pH

7.5

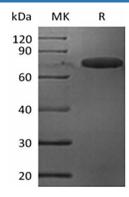
Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants

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

For Research Use Only

Elabscience Bionovation Inc.



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Mouse Leukocyte Immunoglobulin-like Receptor Subfamily B Member 4 (LILRB4/CD85k/ILT3) is an approximately transmembrane glycoprotein that negatively regulates immune cell activation. Mouse LILRB4 consists of a 215 amino acid (aa) extracellular domain with two Ig-like domains, a 22 aa transmembrane segment, and a 75 aa cytoplasmic domain with 3 immunoreceptor tyrosine-based inhibitory motifs (ITIM). Within the ECD, mouse LILRB4 shares 45% and 77% aa sequence identity with human and rat LILRB4, respectively. Alternative splicing of mouse LILRB4 generates a potentially soluble isoform that lacks the transmembrane segment. LILRB4 is expressed on dendritic cells (DC), monocytes, macrophages, and vascular endothelial cells (EC). Ligation of LILRB4 triggers ITIM-mediated inhibition of cellactivating signaling, leading to enhanced immune tolerance and reduced allogeneic graft rejection. Soluble LILRB4 induces the differentiation of CD8+ T suppressor cells (Ts) that can inhibit the effector functions of CD4+ Th cells and CD8+ CTL. In turn, CD8+ Ts cells induce LILRB4 up-regulation and a tolerogenic phenotype in monocytes, DC, and EC.

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