Elabscience®

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 $< -20^{\circ}C$ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μ m 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

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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.