

## Recombinant Human LILRB4/CD85k/ILT3 Protein (aa 22-259, Fc Tag)

**Catalog Number:** PKSH033604

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

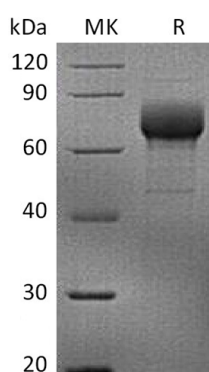
### Description

<b>Species</b>	Human
<b>Source</b>	HEK293 Cells-derived Human LILRB4/CD85k/ILT3 protein Gln22-Glu259, with an C-terminal Fc
<b>Calculated MW</b>	53.2 kDa
<b>Observed MW</b>	65-85 kDa
<b>Accession</b>	Q8NHJ6
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 90 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU per µg of the protein as determined by the LAL method.
<b>Storage</b>	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.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
<b>Reconstitution</b>	Please refer to the specific buffer information in the printed manual. Please refer to the printed manual for detailed information.

### Data



> 90 % as determined by reducing SDS-PAGE.

### Background

### For Research Use Only

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.