

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived
Gly24-Lys238, with a C-terminal 6-His tag
Accession # Q64281

N-terminal Sequence Analysis Gly24 & Ser37 (minor)

Predicted Molecular Mass 25 kDa

SPECIFICATIONS

SDS-PAGE 34-42 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Mouse LILRB4/CD85k/ILT3 is coated onto a microplate at 2 µg/mL, Recombinant Human Angiotensin-like Protein 7/ANGPTL7 (Catalog # 914-AN) binds with a typical ED₅₀ = 20-120 ng/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

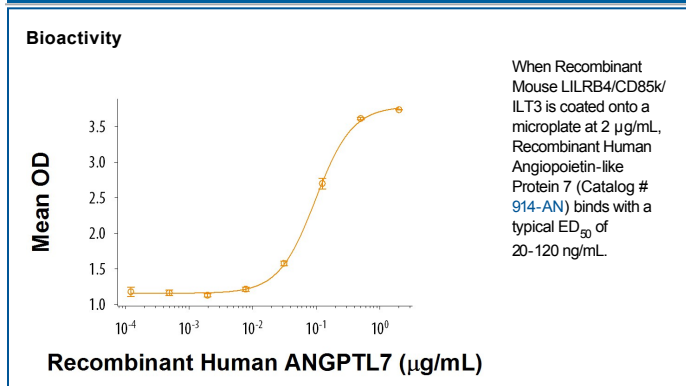
Reconstitution Reconstitute at 500 µg/mL in PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

LILRB4, also known as ILT3, CD85k, and LIR-5, is an approximately 60 kDa transmembrane glycoprotein that negatively regulates immune cell activation (1). Mature 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) (2). 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 (2). LILRB4 is expressed on dendritic cells (DC), monocytes, macrophages, and vascular endothelial cells (EC) (3, 6, 7). Ligation of LILRB4 triggers ITIM-mediated inhibition of cell-activating signaling, leading to enhanced immune tolerance and reduced allogeneic graft rejection (3, 5, 8, 9). Soluble LILRB4 induces the differentiation of CD8⁺ T suppressor cells (Ts) that can inhibit the effector functions of CD4⁺ Th cells and CD8⁺ CTL (5, 8, 10). In turn, CD8⁺ Ts cells induce LILRB4 up-regulation and a tolerogenic phenotype in monocytes, DC, and EC (6, 7, 9, 11, 12).

References:

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