

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived human LILRB4/CD85k/ILT3 protein
Pro17-His257, with a C-terminal 6-His tag
Accession # AAH26309

N-terminal Sequence Analysis Pro17& Ala (N+3 of signal derived sequence)

Predicted Molecular Mass 27 kDa

SPECIFICATIONS

SDS-PAGE 29-43 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Human LILRB4/CD85k/ILT3 is coated at 2 µg/mL, Recombinant Human Angiopoietin-like Protein 7/ANGPTL7 (Catalog # 914-AN) binds with an apparent $K_d < 1$ nM.

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

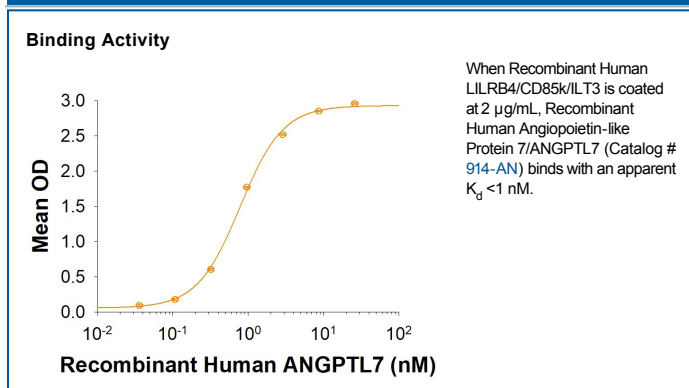
Reconstitution Reconstitute at 100 µg/mL in PBS.

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

ILT3, also known as CD85k and LIR-5, is an approximately 60 kDa transmembrane glycoprotein that negatively regulates immune cell activation (1). Mature human ILT3 consists of a 238 amino acid (aa) extracellular domain with two Ig-like domains, a 21 aa transmembrane segment, and a 168 aa cytoplasmic domain with 3 immunoreceptor tyrosine-based inhibitory motifs (ITIM) (2). Alternative splicing of human ILT3 generates an isoform that lacks the first ITIM and a secreted isoform that circulates in the serum of cancer patients (3, 4). ILT3 is expressed on dendritic cells (DC), monocytes, macrophages, and vascular endothelial cells (EC) (2, 5, 6). Ligand of ILT3 triggers ITIM-mediated inhibition of cell-activating signaling, leading to enhanced immune tolerance and reduced allogeneic graft rejection (2, 4, 7, 8). Soluble ILT3 induces the differentiation of CD8⁺ T suppressor cells (Ts) that can inhibit the effector functions of CD4⁺ Th cells and CD8⁺ CTL (4, 7, 9). In turn, CD8⁺ Ts cells induce ILT3 up-regulation and a tolerogenic phenotype in monocytes, DC, and EC (5, 6, 8, 10, 11).

References:

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