

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived human LILRB4/CD85k/ILT3 protein		
	Human LILRB4 (Gly24-Glu259) Accession # NP_001265355.2	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Gly24		
Structure / Form	Biotinylation via amines		
Predicted Molecular Mass	53 kDa		

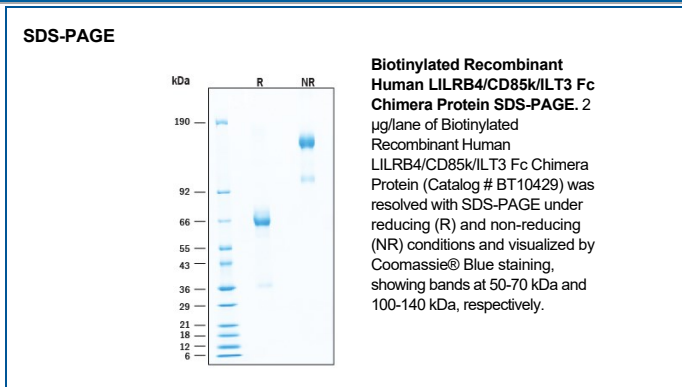
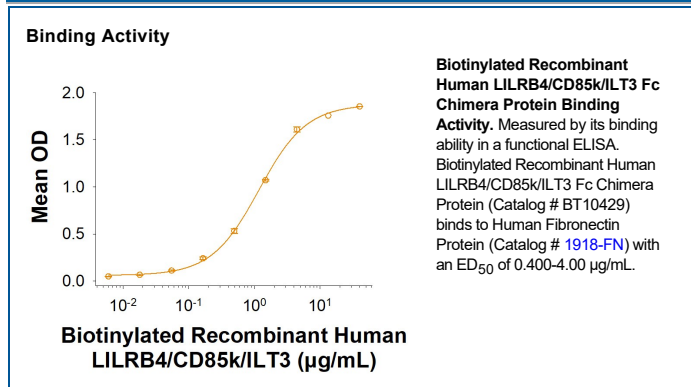
SPECIFICATIONS

SDS-PAGE	50-70 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Biotinylated Recombinant Human LILRB4/CD85k/ILT3 Fc Chimera binds to Human Fibronectin Protein (Catalog # 1918-FN) with an ED ₅₀ of 0.400-4.00 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>85%, 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 with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in water.
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. <ul style="list-style-type: none"> • 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 LILRB4, CD85k, and LIR5, is an approximately 60 kDa transmembrane glycoprotein that negatively regulates immune cell activation (1). Mature human ILT3 consists of an extracellular domain (ECD) with two Ig-like domains, a transmembrane segment, and a cytoplasmic domain with 3 immunoreceptor tyrosine-based inhibitory motifs (ITIM) (2). The mature ECD of human ILT3 shares 44% amino acid identity with mouse ILT3. 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). Ligation 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). Recently, a novel anti-LILRB4 CAR-T Cell was been used to treat monocytic acute myeloid leukemia in humanized hematopoietic-reconstituted mice models (12).

References:

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