Biotinylated Recombinant Human LAG-3 Fc Chimera

Catalog Number: BT2319

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived human LAG-3 protein

Human LAG-3 (Leu23-Leu450) Accession # P18627	IEGRMD	Human IgG_1 (Pro100-Lys330)

N-terminus C-terminus

N-terminal Sequence Analysis	Leu23
Structure / Form	Disulfide-linked homodimer. Biotinylated via amines.
Predicted Molecular Mass	72.7 kDa

SPECIFICATIONS

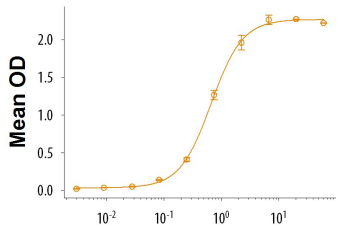
SDS-PAGE	74-91 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Human LSECtin/CLEC4G (Catalog # 2947-CL) is coated at 1 µg/mL, 100 uL/well, biotinylated recombinant LAG-3 Fc Chimera (Catalog# BT2319) binds with an ED_{50} of 0.2-2 µg/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
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 200 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

Binding Activity



Biotinylated Recombinant Human LAG-3 Fc Chimera (µg/mL)

When Recombinant Human LSECtin/CLEC4G (Catalog # 2947-CL) is coated at 1 µg/mL, 100 µL/well, Biotinylated Recombinant Human LAG-3 Fc Chimera Biotinylated (Catalog # BT2319) binds with an ED_{50} of 0.2-2 µg/mL.

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BACKGROUND

LAG-3 (Lymphocyte activation gene-3), designated CD223, is a 70 kDa type I transmembrane protein that is a member of the immunoglobulin superfamily (IgSF) (1, 2). LAG-3 shares approximately 20% amino acid sequence homology with CD4, but has similar structure and binds to MHC class II with higher affinity, providing negative regulation of T cell receptor signaling (1, 2). Human LAG-3 cDNA encodes 525 amino acids (aa) that include a 28 aa signal sequence, a 422 aa extracellular domain (ECD) with four Ig-like domains, a transmembrane region and a highly charged cytoplasmic region. Within the ECD, human LAG-3 shares 70%, 67%, 76%, and 73% aa sequence identity with mouse, rat, porcine, and bovine LAG-3, respectively. LAG-3 is expressed on activated $CD4^+$ and $CD8^+$ T cells, NK cells, and plasmacytoid dendritic cells (pDC), but not on resting T cells (1-3). LAG-3 on activated $CD4^+CD25^+$ T_{reg} cells plays a role in their suppressive activity (4). LAG-3 limits the expansion of activated T cells and pDC in response to selected stimuli (3-5). A soluble 54 kDa form, sLAG-3, can be shed by metalloproteinases ADAM10 and TACE/ADAM17 (6, 7). While monomeric sLAG-3 itself may be inactive, shedding allows for normal T cell activation by removing negative regulation (7). Binding of a homodimerized sLAG-3/Ig fusion protein to MHC class II molecules induces maturation of immature DC, and secretion of cytokines such as IFN-γ and TNF-α by type 1 cytotoxic $CD8^+$ T cells and NK cells (8, 9). sLAG-3/Ig has been used as a potential adjuvant to stimulate a cytotoxic anti-cancer immune response (9, 10). In mice, deletion of LAG-3 and another negative regulator, PD-1, facilitates anti-cancer response but also blocks self-tolerance and increases susceptibility to autoimmune diseases (11, 12). In humans, antibody-mediated down-regulation of LAG-3 and PD-1 allows more effective control of chronic malaria, while in NOD (non-obese diabetic) mice, deletion of LAG-3 alone accelerates diabetes (12-14).

References:

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