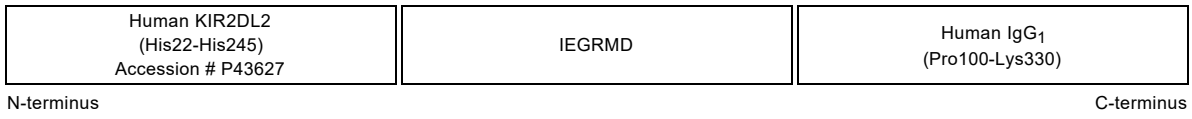
Recombinant Human KIR2DL2/CD158b1 Fc Chimera

Catalog Number: 3015-KR

DESCRIPTION

Source

Mouse myeloma cell line, NS0-derived human KIR2DL2/CD158b1 protein



N-terminal Sequence Analysis	His22
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	51.2 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	65-75 kDa, reducing conditions
Activity	Bioassay data are not available.
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
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.

BACKGROUND

KIR2DL2 (2DL2, formerly NKAT6, designated CD158b) is a 348 amino acid (aa) type I transmembrane glycoprotein that belongs to the human killer cell Ig-like receptor (KIR) family (1, 2). KIRs are expressed on human $CD56^{dim}$ NK cells and T cell subsets, and regulate effector functions in the innate immune system (1-3). KIRs are named for the number of Ig-like domains (2D or 3D) in the extracellular domain (ECD), and whether they have long or short (L, S) cytoplasmic tails (1-3). Individuals will express varying subsets of inhibiting and activating KIRs with varying polymorphisms (1, 4). Like other inhibiting KIRs, KIR2DL2 has two ITIM domains within its long tail that block activating receptor clustering (2, 5). Within the ECD, KIR2DL2 shares very high aa sequence identity (98%) with KIR2DL3. The two segregate as alleles of the same gene, although KIR2DL2 shows higher avidity for HLA-C1 ligands (1, 6). Extracellular aa identity is also high for KIR2DL1 (92%). The three together recognize and inhibit NK cytotoxicity against cells expressing any HLA-C allotype, allowing self-recognition, but also conferring susceptibility to leukemia (1-3). KIR2DL2 recognizes Asn80-containing HLA-C1 and, more weakly, Lys80-containing C2 allotypes (1, 6-8). KIR2DL2 can impact both innate and adaptive immunity, contributing to either viral persistence or antiviral immunity, depending on the HLA class I molecules expressed by the individual (9).

References:

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