Recombinant Human KIR2DL3/CD158b2 Fc Chimera
Catalog Number: 2014-KR

DESCRIPTION

Source
Mouse myeloma cell line, NS0-derived human KIR2DL3/CD158b2 protein

<table>
<thead>
<tr>
<th>Human KIR2DL3</th>
<th>IEGRMDFc</th>
<th>Human IgG1</th>
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<tbody>
<tr>
<td>(His22-His245)</td>
<td>(Pro100-Lys330)</td>
<td>(Pro100-Lys330)</td>
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N-terminal Sequence Analysis
His22

Structure / Form
Disulfide-linked homodimer

Predicted Molecular Mass
51.2 kDa (monomer)

SPECIFICATIONS

SDS-PAGE
66-75 kDa, reducing conditions

Activity
Measured by its ability to bind HEK293T human embryonic kidney cells in a flow cytometry assay.
When 10 μg of Recombinant Human KIR2DL3/CD158b2 Fc Chimera is added to 5 x 10⁵ HEK293T cells, >20% of the cells will bind to the protein.

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

Purity
>90%, 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.

BACKGROUND

KIR2DL3 (2DL3, formerly NKAT2, designated CD158b2) is a 341 amino acid (aa) type I transmembrane glycoprotein that belongs to the human killer cell Ig-like receptor (KIR) family of molecules (1, 2). KIRs are expressed on human CD56dim 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, KIR2DL3 has two ITIM domains within its long tail that block activating receptor clustering (2). Within the ECD, KIR2DL3 shares very high aa sequence identity (98%) with KIR2DL2. The two segregate as alleles of the same gene; both recognize Asn80-containing HLA-C1 and, more weakly, Lys80-containing C2 allotypes (1, 5). Extracellular aa identity is also high for KIR2DL1 (93%). The three molecules together recognize and inhibit NK cytotoxicity against cells expressing any HLA-C allotype, allowing for self-recognition (1-3). Compared with KIR2DL2, KIR2DL3 shows lower avidity for HLA-C1 ligands; when compared to KIR2DL1, KIR2DL3 has lower avidity but broader specificity for HLA-C1 ligands (1, 5, 6). Configurations of inhibiting and activating KIR can alter an individual’s susceptibility to viral and autoimmune diseases and leukemia (1-3). For example, rheumatoid arthritis patients that are positive for KIR2DL3 and negative for KIR2DS3 have an earlier disease diagnosis (7).

References: