Recombinant Mouse NKG2A/CD159a Fc Chimera

Catalog Number: 9198-NK

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived

MDP	Mouse IgG_{2A} (Glu98-Lys330)	IEGR	Mouse NKG2A (Ala94-Ile244) Accession # Q9Z202

N-terminus C-terminus

N-terminal Sequence Analysis Met

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 44 kDa

SPECIFICATIONS

SDS-PAGE 58-67 kDa, reducing conditions

Activity Measured by its binding ability in a functional ELISA.
When Recombinant Mouse NKG2A/CD159a Fc Chimera is coated at 0.5 µg/mL, recombinant human CD94 binds with a typical ED_{50} of 0.1-0.6 µg/mL.

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 500 µg/mL in PBS.

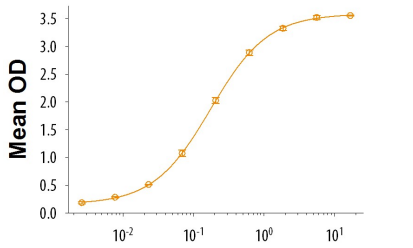
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

DATA

Bioactivity



When Recombinant Mouse NKG2A Fc Chimera (Catalog # 9198-NK) is coated at 0.5 µg/mL, recombinant human CD94 binds with a typical ED_{50} of 0.1-0.6 µg/mL.

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BACKGROUND

NKG2A/CD159a is an approximately 40 kDa transmembrane C-type lectin superfamily protein that inhibits innate immune system activation (1). Mouse NKG2A consists of a 70 amino acid (aa) cytoplasmic domain with two ITIM inhibitory motifs, a 23 aa transmembrane segment, and a 151 aa extracellular domain (ECD) with one C-type lectin domain (2). Within the ECD, mouse NKG2A shares 41% and 71% aa sequence identity with human and rat NKG2A, respectively. Alternative splicing generates additional isoforms with a 17 aa deletion in the extracellular juxtamembrane region or a substitution of that region plus the transmembrane segment. NKG2A is expressed on a subset of NK cells and $CD8^+$ T cells (2-6) where it forms a covalent heterodimer with CD94 (5, 7, 8). NKG2A-CD94 heterodimers bind to the widely expressed nonclassical MHC-I molecule, HLA-E (Qa-1^b in mouse), which presents a peptide derived from the signal peptide of classical MHC-I molecules (2, 7). Triggering the NKG2A-CD94 complex inhibits the cytolytic activity of NK and $CD8^+$ T cells (2, 3, 5, 6, 9). This enables the innate immune system to detect cells that express host MHC-I molecules and to protect them from NK cell mediated lysis. This mechanism is subverted by human cytomegalovirus which encodes a peptide that is homologous to the HLA-E binding peptide (10). HCMV infected cells up-regulate both HLA-E and NKG2A expression and utilize this peptide to escape from immune clearance (3, 10). In contrast, vaccinia virus induces HLA-E down-regulation, thus permitting NK cell lysis of the virally infected cell (11).

References:

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