NKG2A Protein, Human (HEK293, His-Avi)

Cat. No.:	HY-P78499
Synonyms:	NKG2A; NKG2-A; CD94; KLRD1; KP43; KLRC1; CD159A
Species:	Human
Source:	HEK293
Accession:	P26715 (R100-L233)
Gene ID:	3821
Molecular Weight:	40-50 kDa

PROPERTIES

Biological Activity	Immobilized Human NKG2A, His Tag at 5μg/ml (100μl/Well) on the plate. Dose response curve for Anti-NKG2A Antibody, hFc Tag with the EC_{50} of 0.79μg/ml determined by ELISA.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.22 μm filtered solution of PBS, pH 7.4. Normally 5% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	NKG2A Protein, an immune inhibitory receptor crucial for self-nonself discrimination, forms a complex with KLRD1 on cytotoxic and regulatory lymphocyte subsets, recognizing the non-classical major histocompatibility (MHC) class Ib molecule HLA-E loaded with self-peptides from the signal sequence of classical MHC class Ia molecules. This recognition allows cytotoxic cells to monitor MHC class I expression in healthy cells and promotes self-tolerance. Upon binding to HLA-E-peptide complexes, NKG2A transmits intracellular signals through two immunoreceptor tyrosine-based inhibition motifs (ITIMs), recruiting INPP5D/SHP-1 and INPPL1/SHP-2 tyrosine phosphatases to oppose signals from activating receptors. As a key inhibitory receptor on natural killer (NK) cells, NKG2A regulates their activation and effector functions, countering T cell receptor signaling on a subset of memory/effector CD8-positive T cells and distinguishing harmless from pathogenic antigens. In the HLA-E-rich tumor microenvironment, NKG2A acts as an immune inhibitory checkpoint, contributing to the progressive loss of effector functions in NK cells and tumor-specific T cells, a phenomenon known as cell exhaustion. Notably, during viral infection, NKG2A recognizes HLA-E in complex with human cytomegalovirus-derived peptides, inhibiting NK cell cytotoxicity and facilitating viral immune escape.

Caution: Product has not been fully validated for medical applications. For research use only.

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