

NKG2D/CD314 Protein, Human (Biotinylated, HEK293, His-Avi)

Cat. No.:	HY-P78183
Synonyms:	CD314; D12S2489E; KLR; NKG2-D; NKG2D
Species:	Human
Source:	HEK293
Accession:	P26718 (F78-V216)
Gene ID:	100528032
Molecular Weight:	Approximately 19.0 kDa

PROPERTIES

Biological Activity	Measured by its binding ability in a functional ELISA. When immobilized Biotinylated Human NKG2D His at 1 µg/mL (100µ L/Well), Anti-NKG2D Antibody hFc binds with an EC ₅₀ of 4.4 ng/mL.
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.
Reconstitution	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH ₂ O.
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

NKG2D/CD314 protein operates as an activating and costimulatory receptor essential for immunosurveillance, binding to diverse cellular stress-inducible ligands presented on autologous tumor cells and virus-infected cells. It plays a dual role in innate immune responses, stimulating both activating killer (NK) cells and acting as a costimulatory receptor for T-cell receptors (TCR) in CD8(+) T-cell-mediated adaptive immune responses, enhancing T-cell activation. The receptor facilitates perforin-mediated elimination of ligand-expressing tumor cells, and its signaling cascades involve calcium influx, ultimately leading to TNF-alpha expression. Additionally, NKG2D/CD314 participates in NK cell-mediated bone marrow graft rejection and may regulate the differentiation and survival of NK cells. Its ligand-binding capacity extends to various subfamilies of MHC class I-related glycoproteins, including MICA, MICB, RAET1E, RAET1G, RAET1L/ULBP6, ULBP1, ULBP2, ULBP3 (ULBP2>ULBP1>ULBP3), and ULBP4. The protein forms homodimers through disulfide linkage and heterohexamers with HCST/DAP10 subunits, a crucial interaction for NK cell surface expression and cytotoxicity induction. Furthermore, it can establish disulfide-bonded heterodimers with CD94 and interacts with CEACAM1, recruiting PTPN6 for VAV1 dephosphorylation, while not interacting with TYROBP.

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

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