NKG2D/CD314 Protein, Human (sf9, His)

Cat. No.:	HY-P73319
Synonyms:	CD314; KLRK1; NKG2-D type II integral membrane protein; NKG2D
Species:	Human
Source:	Sf9 insect cells
Accession:	P26718 (F78-V216)
Gene ID:	22914/100528032
Molecular Weight:	Approximately 22.6 kDa

PROPERTIES

Biological Activity	Immobilized human NKG2D-His (78-216) at 2 μg/mL (100 μL/well) can bind human ULBP1-hFc and the EC_{50} is 8-50 ng/mL.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 20 mM Tris, 300 mM NaCl, 10% Glycerol, pH 8.0. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants 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	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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