FITC-Labeled NKG2D/CD314 Protein, Human (HEK293, Fc)

Cat. No.:	HY-P701284
Synonyms:	NKG2D; CD314; KLRK1; NK cell receptor D
Species:	Human
Source:	HEK293
Accession:	P26718 (F78-V216)
Gene ID:	22914/100528032
Molecular Weight:	50-70 kDa

PROPERTIES

AA Sequence	F L N S L F N Q E V Q I P L T E S Y C G P C P K N W I C Y K N N C Y Q F F D E S K N W Y E S Q A S C M S Q N A S L L K V Y S K E D Q D L L K L V K S Y H W M G L V H I P T N G S W Q W E D G S I L S P N L L T I I E M Q K G D C A L Y A S S F K G Y I E N C S T P N T Y I C M Q R T V
Appearance	Solution.
Formulation	Supplied as a 0.22 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year, protect from light. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice

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.

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