

Product Data Sheet

NKG2D/CD314 Protein, Cynomolgus (HEK293, His)

Cat. No.:	HY-P72504
Synonyms:	NKG2-D type II integral membrane protein; CD314; KLRK1; NKG2-D
Species:	Cynomolgus
Source:	HEK293
Accession:	P61252 (F78-V216)
Gene ID:	102120479
Molecular Weight:	22-35 kDa

DDODEDTIEC	
PROPERTIES	
AA Sequence	FLNSLFNQEV QIPLTESYCG PCPKNWICYK NNCYQFFNES KNWYESQASC MSQNASLLKV YSKEDQDLLK LVKSYHWMGL VHIPTNGSWQ WEDGSILSPN LLTIIEMQKG DCALYASSFK GYIENCSIPN TYICMQRTV
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/ μ g, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH ₂ O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
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 proteir 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 crucial for immunosurveillance, binding to diverse 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 triggers signaling cascades involving 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. 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.

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

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