CD94 Protein, Human (HEK293, His)

Cat. No.:	HY-P72700
Synonyms:	Natural killer cells antigen CD94; KP43; CD94; KLRD1
Species:	Human
Source:	HEK293
Accession:	Q13241 (S34-I179)
Gene ID:	3824
Molecular Weight:	23-28 kDa

PROPERTIES

AA Sequence	S F T K L S I E P A F T P G P N I E L Q K D S D C C S C Q E K W V G Y R C N C Y F I S S E Q K T W N E S R H L C A S Q K S S L L Q L Q N T D E L D F M S S S Q Q F Y W I G L S Y S E E H T A W L W E N G S A L S Q Y L F P S F E T F N T K N C I A Y N P N G N A L D E S C E D K N R Y I C K Q Q L I
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
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. 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 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	CD94 Protein, an immune receptor crucial for self-nonself discrimination, forms a complex with KLRC1 or KLRC2 on cytotoxic and regulatory lymphocyte subsets, recognizing the non-classical major histocompatibility (MHC) class Ib molecule HLA-E loaded with self-peptides derived from the signal sequence of classical MHC class Ia and other non-classical MHC class Ib molecules. This interaction enables cytotoxic cells to monitor MHC class I expression in healthy cells, fostering self-tolerance. Primarily serving as a ligand-binding subunit without the capacity to signal, the KLRD1-KLRC1 complex acts as an immune inhibitory receptor, with CD94 playing a key inhibitory role on natural killer (NK) cells. CD94 dominantly counteracts T cell receptor signaling on a subset of memory/effector CD8-positive T cells to prevent autoimmunity. On intraepithelial CD8-positive gamma-delta regulatory T cells, CD94 triggers TGFB1 secretion, limiting the cytotoxic

programming of intraepithelial CD8-positive alpha-beta T cells and distinguishing harmless from pathogenic antigens. In the HLA-E-rich tumor microenvironment, CD94 acts as an immune inhibitory checkpoint, potentially contributing to the progressive loss of effector functions in NK cells and tumor-specific T cells, a state known as cell exhaustion. Upon HLA-E-peptide binding, CD94 transmits intracellular signals through KLRC1 immunoreceptor tyrosine-based inhibition motifs (ITIMs), recruiting INPP5D/SHIP-1 and INPPL1/SHIP-2 tyrosine phosphatases to ITIMs, ultimately opposing signals from activating receptors by dephosphorylating proximal signaling molecules.

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