CD94 Protein, Human (HEK293, His-Avi)

Cat. No.:	HY-P78421
Synonyms:	CD94; KLRD1; KP43; NK cell receptor
Species:	Human
Source:	HEK293
Accession:	Q13241 (S34-I179)
Gene ID:	3824
Molecular Weight:	38-42 kDa

PROPERTIES

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.
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	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.

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

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA