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Recombinant Human SLAMF7/CD319 Protein (His Tag)

Catalog Number: PKSH031026

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

Species Human

Source HEK293 Cells-derived Human SLAMF7/CD319 protein Met 1-Met 226, with an C-

terminal His

 Mol_Mass
 23.8 kDa

 Accession
 NP_067004.3

Bio-activity Measured by its ability to bind biotinylated human SH2D1A-His in a functional

ELISA.

Properties

Purity > 93 % as determined by reducing SDS-PAGE.

Endotoxin < 1.0 EU per µg of the protein as determined by the LAL method.

Storage Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80

°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of

reconstituted samples are stable at < -20°C for 3 months.

Shipping This product is provided as lyophilized powder which is shipped with ice packs.

Formulation Lyophilized from sterile PBS, pH 7.4

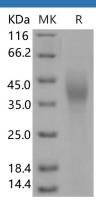
Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants

before lyophilization.

Please refer to the specific buffer information in the printed manual.

Reconstitution Please refer to the printed manual for detailed information.

Data



> 93 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

Fax: 1-832-243-6017

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SLAM family member 7 (SLAMF7), also known as CRACC, CD319, CD2-like receptor-activating cytotoxic cells, and CS1, is a single-pass type I membrane protein and a member of the CD2 family of cell surface receptors. SLAMF7 is expressed in NK cells, activated B-cells, NK-cell line but not in promyelocytic, B-cell lines, or T-cell lines. Although the cytoplasmic domain of CS1 contains immunoreceptor tyrosine-based switch motifs (ITSM), which enables to recruite signaling lymphocyte activation molecule (SLAM)-associated protein (SAP/SH2D1A), it activates NK cells in the absence of a functional SAP. CS1 is a self ligand and homophilic interaction of CS1 regulates NK cell cytolytic activity. CRACC positively regulated natural killer cell functions by a mechanism dependent on the adaptor EAT-2 but not the related adaptor SAP. However, in the absence of EAT-2, CRACC potently inhibited natural killer cell function. It was also inhibitory in T cells, which are typically devoid of EAT-2. Thus, CRACC can exert activating or inhibitory influences on cells of the immune system depending on cellular context and the availability of effector proteins.

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