

## Recombinant Mouse SLAMF7/CD319 Protein (His Tag)

Catalog Number: PKSM040807

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

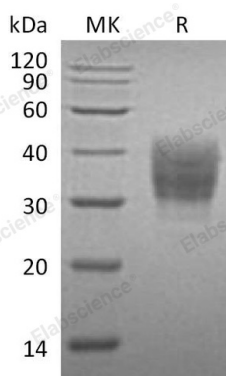
### Description

<b>Species</b>	Mouse
<b>Source</b>	HEK293 Cells-derived Mouse SLAMF7/CD319 protein Met 1-Gly 224, with an C-terminal His
<b>Calculated MW</b>	23.7 kDa
<b>Accession</b>	NP_653122.2
<b>Bio-activity</b>	Measured by its ability to bind biotinylated mouse SLAMF7 in a functional ELISA.

### Properties

<b>Purity</b>	> 95 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU per µg of the protein as determined by the LAL method.
<b>Storage</b>	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.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	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.
<b>Reconstitution</b>	Please refer to the printed manual for detailed information.

### Data



> 95 % as determined by reducing SDS-PAGE.

### Background

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 recruit 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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