

Recombinant Rhesus macaque HVEM/TNFRSF14/CD270 Protein (His Tag)

Catalog Number: PKSQ050069

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

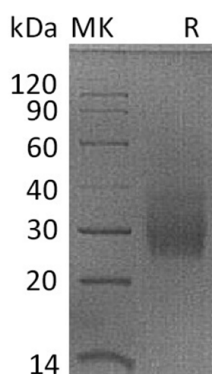
Description

Species	Rhesus macaque
Source	HEK293 Cells-derived Rhesus macaque HVEM/TNFRSF14/CD270 protein Leu39-Val203, with an C-terminal His
Calculated MW	18.3 kDa
Observed MW	22-40 kDa
Accession	F7GSW4
Bio-activity	Not validated for activity

Properties

Purity	> 95 % 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 a 0.2 µm filtered solution of 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



> 95 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

Herpesvirus entry mediator (HVEM) is a type I membrane protein in the TNF receptor superfamily, and it can both promote and inhibit T cell activity. HVEM is highly expressed on naïve CD4⁺ T cells, CD8⁺ T memory cells, regulatory T cells, dendritic cells, monocytes, and neutrophils. It functions as a receptor for BTLA, CD160, LIGHT/TNFSF14, and Lymphotoxin-alpha. Ligation of HVEM by LIGHT triggers T cell, monocyte, and neutrophil activation and contributes to Th1 inflammation and cardiac allograft rejection. In contrast, HVEM binding to CD160 or BTLA suppresses T cell and dendritic cell activation and dampens intestinal inflammation. HVEM enhances the development of CD8⁺ T cell memory and Treg function. It is additionally expressed on intestinal epithelial cells, where its binding by intraepithelial lymphocyte (IEL) expressed CD160 promotes epithelial integrity and host defense. The herpesvirus envelope glycoprotein gD, which binds HVEM to initiate membrane fusion, can antagonize both BTLA and LIGHT binding.