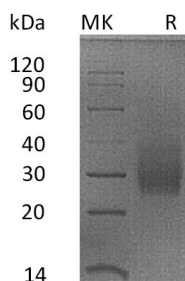


## Summary

<b>Name</b>	HVEM/TNFRSF14/CD270
<b>Purity</b>	Greater than 95% as determined by reducing SDS-PAGE
<b>Endotoxin level</b>	<1 EU/μg as determined by LAL test.
<b>Construction</b>	Recombinant Rhesus Macaque TNF Receptor Superfamily Member 14 is produced by our Mammalian expression system and the target gene encoding Leu39-Val203 is expressed with a 6His tag at the C-terminus.
<b>Accession #</b>	F7GSW4
<b>Host</b>	Human Cells
<b>Species</b>	Rhesus macaque
<b>Predicted Molecular Mass</b>	18.3 KDa
<b>Formulation</b>	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.
<b>Stability&amp;Storage</b>	Store at ≤-70°C, stable for 6 months after receipt. Store at ≤-70°C, stable for 3 months under sterile conditions after opening. Please minimize freeze-thaw cycles.
<b>Reconstitution</b>	Always centrifuge tubes before opening. Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100μg/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.

## SDS-PAGE image



## Background

**Product Name: Recombinant Rhesus Macaque HVEM (C-6His)**  
**Catalog #: PHV1975**



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**Alternative Names**

Tumor Necrosis Factor Receptor Superfamily Member 14; Herpes Virus Entry Mediator A; Herpesvirus Entry Mediator A; HveA; Tumor Necrosis Factor Receptor-Like 2; TR2; CD270; TNFRSF14; HVEA; HVEM

**Background**

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<sup>+</sup> T cells, CD8<sup>+</sup> 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<sup>+</sup> 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.

**Note**

For Research Use Only , Not for Diagnostic Use.