



Recombinant Protein Technical Manual

**Recombinant Rhesus macaque
HVEM/TNFRSF14/CD270 Protein (His Tag)
RPES3684**

Product Data:

Product SKU: RPES3684

Size: 10µg

Species: Rhesus macaque

Expression host: Human Cells

Uniprot: F7GSW4

Protein Information:

Molecular Mass: 18.3 kDa

AP Molecular Mass: 25-40 kDa

Tag: C-His

Bio-activity:

Purity: > 95% as determined by reducing SDS-PAGE.

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

Storage: Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.

Shipping: This product is provided as liquid. It is shipped at frozen temperature with blue ice. Upon receipt, store it immediately at < -20°C.

Formulation: Supplied as a 0.2 µm filtered solution of PBS, pH 7.4.

Reconstitution: Please refer to the printed manual for detailed information.

Application:

Synonyms: 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

Immunogen Information:

Sequence: Leu39-Val203

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 na?ve 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.