

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived		
	Cynomolgus Monkey HVEM/TNFRSF14 (Pro37-Val203) Accession # XP_005545061	IEGRMD	Human IgG ₁ (Pro100-Lys330)
	N-terminus		C-terminus

N-terminal Sequence Analysis	Pro37
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	44 kDa

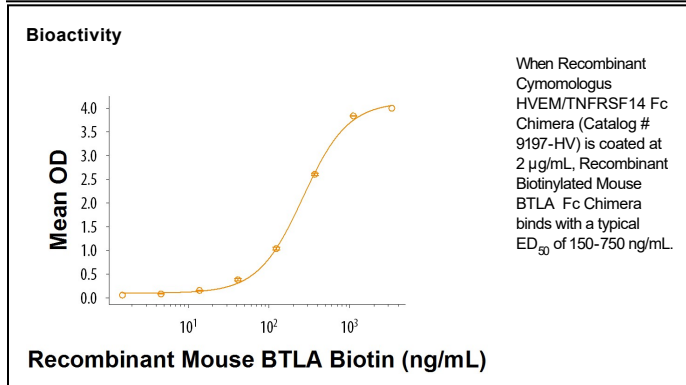
SPECIFICATIONS

SDS-PAGE	56-63 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When Recombinant Cynomolgus Monkey HVEM/TNFRSF14 Fc Chimera is coated at 2 µg/mL (100 µL/well), biotinylated recombinant mouse BTLA Fc Chimera binds with a typical ED ₅₀ of 150-750 ng/mL.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

HVEM (herpesvirus entry mediator), also known as TNFRSF14 and CD270, is a type I membrane protein in the TNF receptor superfamily, and it can both promote and inhibit T cell activity (1). Mature cynomolgous HVEM consists of a 171 amino acid (aa) extracellular domain (ECD) with three cysteine-rich domains (CRD), a 24 aa transmembrane segment, and a 42 aa cytoplasmic tail with a TRAF interaction domain (2, 3). Within the ECD, cynomolgous HVEM shares 88%, 54%, and 54% aa sequence identity with human, mouse, and rat HVEM, respectively. HVEM is highly expressed on naïve CD4⁺ T cells, CD8⁺ T memory cells, regulatory T cells, dendritic cells, monocytes, and neutrophils (4-8). Its expression declines during effector T cell activation but is up-regulated during Treg activation (4, 5). HVEM functions as a receptor for BTLA, CD160, LIGHT/TNFSF14, and Lymphotoxin- α (4, 9-12). Ligation of HVEM by LIGHT triggers T cell, monocyte, and neutrophil activation (8, 10) and contributes to Th1 inflammation and cardiac allograft rejection (13, 14). In contrast, HVEM binding to CD160 or BTLA suppresses T cell and dendritic cell activation (4, 7, 9, 10) and dampens intestinal inflammation (15). HVEM enhances the development of CD8⁺ T cell memory and Treg function (5, 6). It is additionally expressed on intestinal epithelial cells, where its binding by intraepithelial lymphocyte (IEL) expressed CD160 promotes epithelial integrity and host defense (16). The herpesvirus envelope glycoprotein gD, which binds HVEM to initiate membrane fusion, can antagonize both BTLA and LIGHT binding (2, 9, 11).

References:

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