

DESCRIPTION

Source	Chinese Hamster Ovary cell line, CHO-derived		
	Mouse HVEM (Gln29-Val207) Accession # NP_849262	IEGRMDP	Mouse IgG _{2A} (Glu98-Lys330)
	N-terminus		C-terminus
N-terminal Sequence Analysis	Gln29		
Structure / Form	Disulfide-linked homodimer		
Predicted Molecular Mass	45.6 kDa		

SPECIFICATIONS

SDS-PAGE	60-65 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. When rmHVEM/ Fc chimera is immobilized at 100 ng/ mL, 100 µL/ well, the concentration of biotinylated rmBTLA/ Fc chimera that produces 50% of the optimal binding response is found to be approximately 30-120 ng/mL.
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 1 mg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <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.

BACKGROUND

HVEM (herpesvirus entry mediator) is a type I membrane protein that is TNF receptor superfamily member 14 (TNFRSF14) (1). The mouse HVEM cDNA encodes a 275 amino acid (aa) protein. It contains a 36 aa signal peptide, a 170 aa extracellular domain with three cysteine rich domains (CRD), a 24 aa transmembrane region and a 45 aa cytoplasmic tail with a TRAF interaction domain (1). HVEM expression is highest on naïve, memory and regulatory T cells, but declines during T cell activation (2, 3). It is present at low levels on most resting leukocytes (4). HVEM is a receptor for the IGSF member BTLA (B and T lymphocyte attenuator), CD160, and the TNF family ligand LIGHT (lymphotoxins, exhibits inducible expression, and competes with HSV glycoprotein D for HVEM, a receptor expressed by T lymphocytes) (2, 9). HVEM and BTLA are constitutively expressed on T cells, while LIGHT is generally considered to be inducible upon TCR activation. In the absence of activation, HVEM and BTLA interact monomerically, either in *cis*, or in *trans*. A same cell (or *cis*) interaction likely promotes general cell survival, while a between cell (or *trans*) interaction promotes a state of lymphocyte inactivity through the BTLA cytoplasmic domain. Following T cell activation, LIGHT appears and disrupts existing HVEM-BTLA bonds. A LIGHT-HVEM trimer now forms in *trans*, initiating HVEM-mediated NFκB signaling and a proinflammatory response (10). BTLA and LIGHT interactions are not mutually exclusive, but BTLA appears dominant (4, 6, 7). The herpesvirus envelope glycoprotein gD, which binds HVEM CRD1 to initiate membrane fusion, can antagonize both BTLA and LIGHT binding (1, 6, 7, 9). Human, but not mouse, HVEM can also bind lymphotoxin within CRD2 (9, 11). Graft-versus-host disease and Th1 type intestinal inflammation can be ameliorated by interrupting T cell LIGHT/HVEM interactions, while disruption of BTLA/HVEM interaction promotes intestinal inflammation (12 - 14). Mouse HVEM ECD shares 89% and 53% aa identity with rat and human HVEM, respectively. Mouse HVEM can recognize human BTLA and LIGHT, but human HVEM does not recognize mouse ligands (2, 11).

References:

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