Recombinant Human HVEM/TNFRSF14 Fc Chimera

Catalog Number: 356-HV/CF

DESCRIPTION

Source Mouse myeloma cell line, NS0-derived

N-terminal Sequence Analysis	Pro37
Structure / Form	Disulfide-linked homodimer
Predicted Molecular Mass	45 kDa (monomer)

SPECIFICATIONS

SDS-PAGE	60 kDa, reducing conditions
Activity	Measured by its ability to inhibit TNF-β-mediated cytotoxicity using L-929 mouse fibroblast cells. The ED_{50} for this effect is 2.5-10 µg/mL in the presence of 50 pg/mL of Recombinant Human TNF-β/TNFSF1 (Catalog # 211-TB).
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>90%, 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 sterile 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.** ● 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), 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 human HVEM consists of a 164 amino acid (aa) extracellular domain (ECD) with three cysteine-rich domains (CRD), a 21 aa transmembrane segment, and a 60 aa cytoplasmic tail with a TRAF interaction domain (2, 3). Within the ECD, human HVEM shares 55% aa sequence identity with mouse and rat HVEM. Alternative splicing generates an additional isoform with a substitution of the N-terminal 10 amino acids including the signal peptide. 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 epitheilal 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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