Recombinant Human HVEM/TNFRSF14 Protein (His & Fc Tag)(Active)

Catalog Number:PKSH031674

Note: Centrifuge before opening to ensure complete recovery of vial contents.

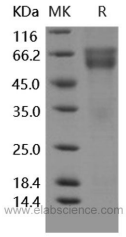
Description

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
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Val 202
Accession	NP_003811.2
Calculated Molecular Weight	45.4 kDa
Observed molecular weight	60-65 kDa
Tag	C-His & Fc
Bioactivity	Measured by its ability to bind biotinylated mouse BTLA-Fc in functional ELISA.

Properties

Purity	> 85 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg as determined by the LAL method.
Storage	Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 100mM Glycine, 10mM NaCl, 50mM Tris, pH 7.5
Reconstitution	Please refer to the printed manual for detailed information.

Data



Background

Herpesvirus entry mediator (HVEM), also referred to as TNFRSF14, TR2 (TNF receptor-like molecule) and ATAR (another TRAF-associated receptor), is a member of type I transmembrane protein belonging to the TNF-receptor superfamily. It is expressed on many immune cells, including T and B cells, NK cells, monocytes, and neutrophils. Two TNF superfamily ligands lymphotoxin α (TNF-β) and LIGHT (TNFSF14) are identified as cellular ligands for HVEM and initiate the positive signaling. However, recent studies have revealed that HVEM is also involved in the unique inhibitory signaling pathway for T cells through activating tyrosine phosphorylation of the immunoreceptor tyrosine-based inhibitory motif (ITIM) in B and T lymphocyte attenuator (BTLA). HVEM provides a stimulatory signal following engagement with LIGHT (TNFSF14) on T cells. In contrast, it can also provide an inhibitory signal to T cells when it

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binds the B and T lymphocyte attenuator (BTLA), a ligand member of the Immunoglobulin (Ig) superfamily. Thus, HVEM may be viewed as a molecular switch, capable of facilitating both stimulatory and inhibitory cosignaling in T cells. Substantial evidence from both human disease and from experimental mouse models has indicated that dysregulation of the LIGHT-HVEM-BTLA cosignaling pathway can cause inflammation in the lung and in mucosal tissues.

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