Human HVEM / TNFRSF14 Protein (His & Fc Tag)

Catalog Number: **10334-H03H**

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Biological Solution Specialist

General Information

Gene Name Synonym:

ATAR; CD270; HVEA; HVEM; LIGHTR; TR2; Atar; HveA; Hvem; Tnfrs14

Protein Construction:

A DNA sequence encoding the extracellular domain (Met 1-Val 202) of human HVEM (NP_003811.2) precursor was fused with the C-terminal polyhistidine-tagged Fc region of human IgG1 at the C-terminus.

Source: Human

Expression Host: Human Cells

QC Testing

Purity: > 85 % as determined by SDS-PAGE

Endotoxin:

< 1.0 EU per μg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Pro 37

Molecular Mass:

The recombinant human HVEM/Fc is a disulfide-linked homodimeric protein after removal of the signal peptide. The reduced monomer consists of 413 amino acids and predicts a molecular mass of 45.4 kDa. By SDS-PAGE under reducing conditions, the apparent molecular mass of rh HVEM/Fc monomer is approximately 60-65 kDa due to glycosylation.

Formulation:

Lyophilized from sterile 100mM Glycine, 10mM NaCl, 50mM Tris, pH 7.5

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

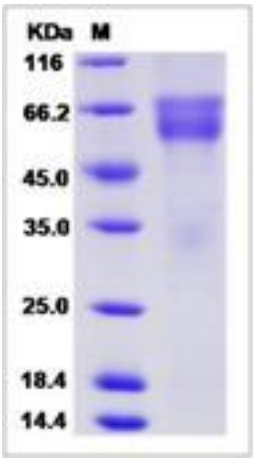
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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 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.

References

1.Murphy KM, *et al.* (2006) Balancing co-stimulation and inhibition with BTLA and HVEM. Nat Rev Immunol. 6(9): 671-81.
2.Heo SK, *et al.* (2007) HVEM signaling in monocytes is mediated by intracellular calcium mobilization. J Immunol. 179(9): 6305-10.
3.Steinberg MW, *et al.* (2008) A crucial role for HVEM and BTLA in preventing intestinal inflammation. J Exp Med. 205(6): 1463-76.