

Mouse HVEM / TNFRSF14 Protein (His & Fc Tag)

Catalog Number: 10567-M03H



Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

Atar; HveA; Hvem; Tnfrs14

Protein Construction:

A DNA sequence encoding the extracellular domain (Met 1-Gln 206) of mouse HVEM (NP_849262.1) precursor was fused with C-terminal His-tagged Fc region of human IgG1 at the C-terminus.

Source: Mouse

Expression Host: HEK293 Cells

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Bio Activity:

Measured by its binding ability in a functional ELISA. Immobilized mouse HVEM-Fc (Cat:10567-M03H) at 10 µg/mL (100 µl/well) can bind biotinylated mouse BTLA-Fc (Cat:51060-M02H). The EC₅₀ of biotinylated mouse BTLA-Fc (Cat:51060-M02H) is 64-96 ng/mL.

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 °C

Predicted N terminal: Pro 40

Molecular Mass:

The recombinant mouse HVEM/Fc is a disulfide-linked homodimeric Protein after removal of the signal peptide. The reduced monomer consists of 415 amino acids and predicts a molecular mass of 46.4 kDa. By SDS-PAGE under reducing conditions, the apparent molecular mass of rmHVEM/Fc monomer is approximately 65 kDa due to the glycosylation.

Formulation:

Lyophilized from sterile PBS, pH 7.4

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°C to -80°C 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.

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