

Mouse HVEM / TNFRSF14 Protein (His Tag)

Catalog Number: 10567-M08H

General Information

Gene Name Synonym:

Atar; HveA; Hvem; Tnfrs14

Protein Construction:

A DNA sequence encoding the mouse TNFRSF14 (NP_849262.1) (Met1-Gln206) was expressed with a polyhistidine tag at the C-terminus.

Source: Mouse

Expression Host: HEK293 Cells

QC Testing

Purity: > 96 % as determined by SDS-PAGE.

Endotoxin:

<1.0 EU per µg 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: Gln 39

Molecular Mass:

The recombinant mouse TNFRSF14 consists of 179 amino acids and predicts a molecular mass of 19.8 kDa.

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:

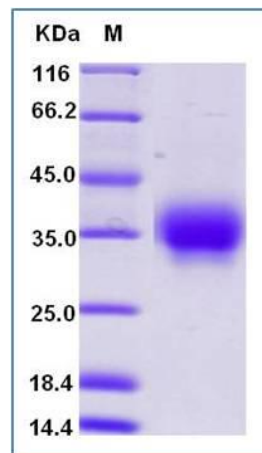
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

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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