Recombinant Mouse HVEM/TNFRSF14 Protein (His & Fc

Tag)

Catalog Number: PKSM040930



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

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Synonyms Tnfrsf14;Herpesvirus entry mediator;HVEM;TR2;TNF receptor-like

molecule; ATAR; another TRAF-associated receptor; Tumor necrosis factor receptor

superfamily member 14;Atar;HveA

Species Mouse

Expression Host HEK293 Cells
Sequence Met 1-Gln 206
Accession NP_849262.1
Calculated Molecular Weight 46.4 kDa
Observed molecular weight 65 kDa
Tag C-His-Fc

Bioactivity Immobilized mouse HVEM-Fch at 10 μ g/mL (100 μ l/well) can bind biotinylated

mouse BTLA-Fc, The EC50 of biotinylated mouse BTLA-Fcis 64-96 ng/mL.

Properties

Purity > 90 % as determined by reducing SDS-PAGE.

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

Storage Generally, 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°C for 2-7 days. Aliquots

of reconstituted samples are stable at < -20°C for 3 months.

Shipping This product is provided as lyophilized powder which is shipped with ice packs.

Formulation Lyophilized from sterile PBS, pH 7.4

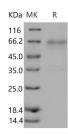
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

protectants before lyophilization.

Please refer to the specific buffer information in the printed manual.

Reconstitution Please refer to the printed manual for detailed information.

Data



> 90 % as determined by reducing SDS-PAGE.

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

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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.

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