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Recombinant Human HVEM/TNFRSF14 Protein (His Tag)

Catalog Number: PKSH033657

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

Description

Species Human

Source HEK293 Cells-derived Human HVEM; TNFRSF14 protein Leu39-Val202, with an C-

terminal His

Calculated MW18.4 kDaObserved MW28 kDaAccessionQ92956

Bio-activity Not validated for activity

Properties

Purity > 95 % 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 a 0.2 µm filtered solution of PBS, pH 7.4.

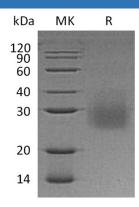
Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants

before lyophilization.

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

Reconstitution Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

Background

Elabscience Bionovation Inc.



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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.

Fax: 1-832-243-6017