

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human HVEM/TNFRSF14 in ELISA.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2742B
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Human embryonic kidney cell HEK293-derived human HVEM/TNFRSF14 Pro37-Val202 Accession # Q92956
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Flow Cytometry	Titration recommended for optimal concentration with starting range of 0.1-1 µg/1 million cells. Sample used for this experiment was Human PBMCs
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PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

HVEM (herpesvirus entry mediator), also known as TNFRSF14 and CD270, is a type I membrane protein in the TNF receptor superfamily, and it can both promote and inhibit T cell activity (1). Mature human HVEM consists of a 164 amino acid (aa) extracellular domain (ECD) with three cysteine-rich domains (CRD), a 21 aa transmembrane segment, and a 60 aa cytoplasmic tail with a TRAF interaction domain (2, 3). Within the ECD, human HVEM shares 55% aa sequence identity with mouse and rat HVEM. Alternative splicing generates an additional isoform with a substitution of the N-terminal 10 amino acids including the signal peptide. HVEM is highly expressed on naïve CD4⁺ T cells, CD8⁺ T memory cells, regulatory T cells, dendritic cells, monocytes, and neutrophils (4-8). Its expression declines during effector T cell activation but is up-regulated during Treg activation (4, 5). HVEM functions as a receptor for BTLA, CD160, LIGHT/TNFSF14, and Lymphotoxin-α (4, 9-12). Ligand of HVEM by LIGHT triggers T cell, monocyte, and neutrophil activation (8, 10) and contributes to Th1 inflammation and cardiac allograft rejection (13, 14). In contrast, HVEM binding to CD160 or BTLA suppresses T cell and dendritic cell activation (4, 7, 9, 10) and dampens intestinal inflammation (15). HVEM enhances the development of CD8⁺ T cell memory and Treg function (5, 6). It is additionally expressed on intestinal epithelial cells, where its binding by intraepithelial lymphocyte (IEL) expressed CD160 promotes epithelial integrity and host defense (16). The herpesvirus envelope glycoprotein gD, which binds HVEM to initiate membrane fusion, can antagonize both BTLA and LIGHT binding (2, 9, 11).

References:

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