Mouse HVEM/TNFRSF14
Alexa Fluor® 750-conjugated Antibody

Monoclonal Rabbit IgG Clone # 2024B
Catalog Number: FAB2516S
100 µg

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse HVEM/TNFRSF14 in direct ELISAs.
Source	Monoclonal Rabbit IgG Clone # 2024B
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse HVEM/TNFRSF14 Gln39-Val207 Accession # NP_849262
Conjugate	Alexa Fluor 750 Excitation Wavelength: 749 nm Emission Wavelength: 775 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	Mouse splenocytes and CHO Chinese hamster ovary cell line transfected with mouse HVEM

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.** ● 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

HVEM (herpesvirus entry mediator) is a type I membrane protein that is TNF receptor superfamily member 14 (TNFRSF14) (1). The mouse HVEM cDNA encodes a 275 amino acid (aa) protein. It contains a 36 aa signal peptide, a 170 aa extracellular domain with three cysteine rich domains (CRD), a 24 aa transmembrane region and a 45 aa cytoplasmic tail with a TRAF interaction domain (1). HVEM expression is highest on naïve, memory and regulatory T cells, but declines during T cell activation (2, 3). It is present at low levels on most resting leukocytes (4). HVEM is a receptor for the IGSF member BTLA (B and T lymphocyte attenuator), CD160, and the TNF family ligand LIGHT (lymphotoxins, exhibits inducible expression, and competes with HSV glycoprotein D for HVEM, a receptor expressed by T lymphocytes) (2, 9). HVEM and BTLA are constitutively expressed on T cells, while LIGHT is generally considered to be inducible upon TCR activation. In the absence of activation, HVEM and BTLA interact monomerically, either in cis, or in trans. A same cell (or cis) interaction likely promotes general cell survival, while a between cell (or trans) interaction promotes a state of lymphocyte inactivity through the BTLA cytoplasmic domain. Following T cell activation, LIGHT appears and disrupts existing HVEM-BTLA bonds. A LIGHT-HVEM trimer now forms in trans, initiating HVEM-mediated NFκB signaling and a proinflammatory response (10). BTLA and LIGHT interactions are not mutually exclusive, but BTLA appears dominant (4, 6, 7). The herpesvirus envelope glycoprotein gD, which binds HVEM CRD1 to initiate membrane fusion, can antagonize both BTLA and LIGHT binding (1, 6, 7, 9). Human, but not mouse, HVEM can also bind lymphotoxin a within CRD2 3 (9, 11). Graft-v s-host disease and Th1 type intestinal inflammation can be ameliorated by interrupting T cell LIGHT/HVEM interactions, while disruption of BTLA/HVEM interaction promotes intestinal inflammation (12-14). Mouse HVEM ECD shares 89% and 53% aa identity with rat and human HVEM, respectively. Mouse HVEM can recognize human BTLA and LIGHT, but human HVEM does not recognize mouse ligands (2, 11).

References:

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