Elab Fluor®700 Anti-Mouse CD272/BTLA Antibody[PK18.6]

Catalog Number: E-AB-F1024UM1

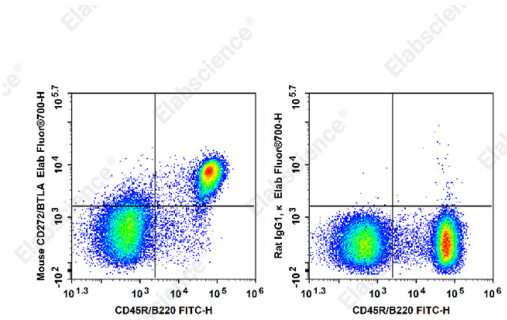
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Mouse
Host	Rat
Isotype	Rat IgG1, κ
Clone No.	PK18.6
Isotype Control	Elab Fluor®700 Rat IgG1, κ Isotype Control[HRPN] [Product E-AB-F09823M1]
Conjugation	Elab Fluor®700
Conjugation Information	Elab Fluor® 700 is designed to be excited by the Red laser (627-640 nm) and detected using an optical filter centered near 719 nm (e.g., a 725/40 nm bandpass filter).
Storage Buffer	Phosphate buffered solution, pH 7.2, containing 0.09% sodium azide and 1% BSA.

Applications	Recommended usage
FCM	Each lot of this antibody is quality control tested by flow cytometric analysis. Please check your vial before the experiment. Since applications vary, the appropriate dilutions must be determined for individual use. We suggest each investigator should titrate the reagent to obtain optimal results [The recommended concentration is 0.1-1 μg/10^6 cells in 100 μL volume].

Data



Staining of C57BL/6 murine splenocytes with FITC Anti-Mouse CD45R/B220 Antibody[RA3.3A 1/6.1] andElab Fluor® 700 Anti-Mouse CD272/BTLA Antibody[PK18.6](left) orElab Fluor®700 Rat IgG1, κ Isotype Control(right). Total viable cells were used for analysis.

Preparation & Storage

Storage	Keep as concentrated solution. This product can be stored at 2-8°C for 12 months. Please protected from prolonged exposure to light and do not freeze.
Shipping	Ice bag

Antigen Information

Alternate Names	B- and T-lymphocyte attenuator;B- and T-lymphocyte-associated protein;Btla;CD272
Uniprot ID	Q7TSA3
Gene ID	208154

For Research Use Only

Background

CD272, also known as B and T lymphocyte attenuator (BTLA), is an Ig superfamily co-inhitory receptor with structural similarity to programmed cell death 1 (PD-1) and CTLA-4. BTLA is expressed on B cells, T cells, macrophages, dendritic cells, NKT cells, and NK cells. Engagement of BTLA by its ligand herpes virus entry mediator (HVEM) is critical for negatively regulating immune response. The absence of BTLA with HVEM inhibitory interactions leads to increased experimental autoimmune encephalomyelitis severity, enhanced rejection of partially mismatched allografts, an increased CD8+ memory T cell population, increased severity of colitis, and reduced effectiveness of T regulatory cells. BTLA plays an important role in the induction of peripheral tolerance of both CD4+ and CD8+ T cells in vivo. Tolerant T cells have significantly higher expression of BTLA compared with effectors and naïve T cells. BTLA may cooperate with CTLA-4 and PD-1 to control T cell tolerance and autoimmunity. It was reported that BTLA may regulate T cell function by binding to B7-H4, but further studies are needed to confirm. The existence of three distinct BTLA alleles has been reported.