

Elab Fluor® 647 Anti-Mouse CD272/BTLA Antibody[PJ196]

Catalog Number: AN00633M

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

Description

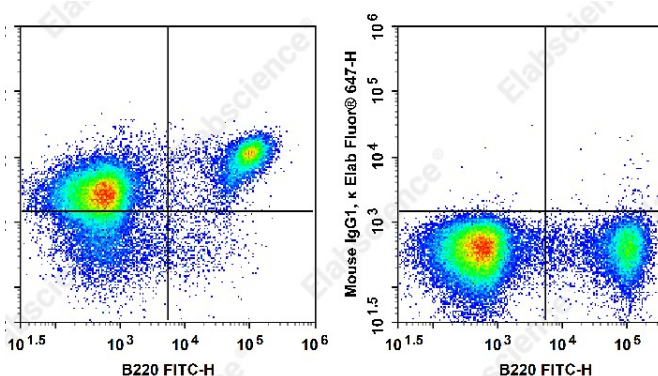
Reactivity	Mouse
Host	Mouse
Isotype	Mouse IgG1, κ
Clone No.	PJ196
Isotype Control	Elab Fluor® 647 Mouse IgG1, κ Isotype Control[MOPC-21] [Product E-AB-F09792M]
Conjugation	Elab Fluor® 647
Conjugation Information	Elab Fluor® 647 is designed to be excited by the Red laser (627-640 nm) and detected using an optical filter centered near 670 nm (e.g., a 660/20 nm bandpass filter).
Storage Buffer	Phosphate buffered solution, pH 7.2, containing 0.09% stabilizer.

Applications

Recommended usage

FCM	Each lot of this antibody is quality control tested by flow cytometric analysis. The amount of the reagent is suggested to be used 5 μL of antibody per test (million cells in 100 μL staining volume or per 100 μL of whole blood). Please check your vial before the experiment. Since applications vary, the appropriate dilutions must be determined for individual use.
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Data



Staining of C57BL/6 murine splenocytes cells with FITC Anti-

Mouse CD45R/B220 Antibody and Elab Fluor® 647 Anti-Mouse CD272(BTLA) Antibody[PJ196] (left) or Elab Fluor® 647 Mouse 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 24 months. Please protected from prolonged exposure to light and do not freeze.
Shipping	Ice bag

Antigen Information

Alternate Names	BTLA;B and T lymphocyte attenuator
Uniprot ID	Q7Z6A9
Gene ID	151888

For Research Use Only

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Rev. V1.2

Background

B and T lymphocyte attenuator (BTLA) is an Ig superfamily coinhibitory 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 significant up-regulated expression of BTLA compared with effector and naïve T cells. BTLA may cooperate with CTLA-4 and PD-1 to control T cell tolerance and autoimmunity. It has been reported that BTLA may regulate T cell function through binding to B7-H4.