

PE/Cyanine7 Anti-Mouse CD272/BTLA Antibody[PK18.6]

Catalog Number: E-AB-F1024H

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	PE/Cyanine7 Rat IgG1, κ Isotype Control[HRPN] [Product E-AB-F09822H]
Conjugation	PE/Cyanine 7
Conjugation Information	PE/Cyanine7 is designed to be excited by the Blue (488 nm), Green (532 nm) and yellow-green (561 nm) lasers and detected using an optical filter centered near 775 nm (e.g., a 780/60 nm bandpass filter).
Storage Buffer	Phosphate buffered solution, pH 7.2, containing 0.09% stabilizer and 1% protein protectant.

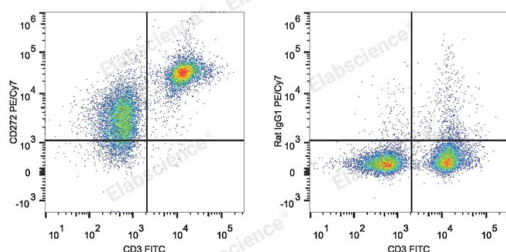
Applications

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 μ L of antibody per test (million cells in 100 μ L staining volume or per 100 μ L of whole blood).** Please check your vial before the experiment. Since applications vary, the appropriate dilutions must be determined for individual use.

Data



C57BL/6 murine splenocytes are stained with PE/Cyanine7 Anti-Mouse CD272 Antibody and FITC Anti-Mouse CD3 Antibody (Left). Splenocytes stained with FITC Anti-Mouse CD3 Antibody and PE/Cyanine7 Rat IgG1 Isotype Control (Right) are used as control.

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
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Uniprot ID

Q7TSA3

Gene ID

208154

Background

CD272, also known as B and T lymphocyte attenuator (BTLA), is an Ig superfamily co-inhibitory 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.

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