

## APC Anti-Mouse CD272/BTLA Antibody[PK18.6]

**Catalog Number:** E-AB-F1024UE

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

### Description

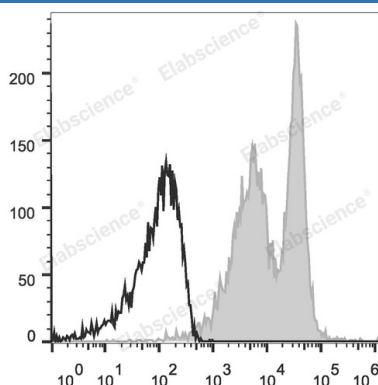
<b>Reactivity</b>	Mouse
<b>Host</b>	Rat
<b>Isotype</b>	Rat IgG1, $\kappa$
<b>Clone No.</b>	PK18.6
<b>Isotype Control</b>	APC Rat IgG1, $\kappa$ Isotype Control[HRPN] [Product E-AB-F09823E]
<b>Conjugation</b>	APC
<b>Conjugation Information</b>	APC is designed to be excited by the Red (627-640 nm) laser and detected using an optical filter centered near 660 nm (e.g., a 660/20 nm bandpass filter).
<b>Storage Buffer</b>	Phosphate buffered solution, pH 7.2, containing 0.09% stabilizer and 1% protein protectant.

### Applications

### Recommended usage

<b>FCM</b>	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 $\mu\text{g}/10^6$ cells in 100 $\mu\text{L}$ volume].
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### Data



C57BL/6 murine splenocytes are stained with APC Anti-Mouse CD272 Antibody (filled gray histogram). Unstained splenocytes (empty black histogram) are used as control.

### Preparation & Storage

<b>Storage</b>	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.
<b>Shipping</b>	Ice bag

### Antigen Information

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

### For Research Use Only

## 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.