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# Product Data Sheet

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## PerCP/Cy5.5 anti-human CD272 (BTLA)

**Catalog # /** 2322570 / 100 tests  
**Size:** 2322565 / 25 tests

**Clone:** MIH26

**Isotype:** Mouse IgG2a,  $\kappa$

**Immunogen:** Human BTLA transfected cells

**Reactivity:** Human

**Preparation:** The antibody was purified by affinity chromatography and conjugated with PerCP/Cyanine5.5 under optimal conditions. The solution is free of unconjugated PerCP/Cyanine5.5 and unconjugated antibody.

**Formulation:** Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide and 0.2% (w/v) BSA (origin USA).

**Concentration:** Lot-specific

□ Human peripheral blood lymphocytes stained with CD19 Brilliant Violet 421™ and CD272 (Clone MIH26) PerCP/Cy5.5 (top) or mouse IgG2a,  $\kappa$  PerCP/Cy5.5 isotype control (bottom).

## Applications:

**Applications:** Flow Cytometry

**Recommended Usage:** Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is 5  $\mu$ l per million cells or 5  $\mu$ l per 100  $\mu$ l of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

\* PerCP/Cyanine5.5 has a maximum absorption of 482 nm and a maximum emission of 690 nm.

**Application Notes:** Additional reported applications (for the relevant formats) include: inhibition of T cell proliferation and cytokine production<sup>1</sup>. Clone MIH26 has agonistic activity on BTLA, resulting in the inhibition of activation.

**Application References:**

1. Watanabe N, *et al.* 2003. *Nat. Immunol.* 4:670.
2. Sun Y, *et al.* 2009. *J. Immunol.* 183:1946.
3. Gonzalez LC, *et al.* 2005. *P. Natl. Acad. Sci. USA* 102:1116.

**Description:** 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<sup>+</sup> 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<sup>+</sup> and CD8<sup>+</sup> 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.

**Antigen**  
**References:**

1. Watanabe N, *et al.* 2003. *Nat. Immunol.* 4:670.
2. Sun Y, *et al.* 2009. *J. Immunol.* 183:1946.
3. Gonzalez LC, *et al.* 2005. *P. Natl. Acad. Sci. USA* 102:1116.