SONY

# Product Data Sheet

## Alexa Fluor® 647 anti-human CD272 (BTLA)

**Catalog # / Size:** 2322595 / 25 tests
2322600 / 100 tests

**Clone:** MIH26

**Isotype:** Mouse IgG2a, κ

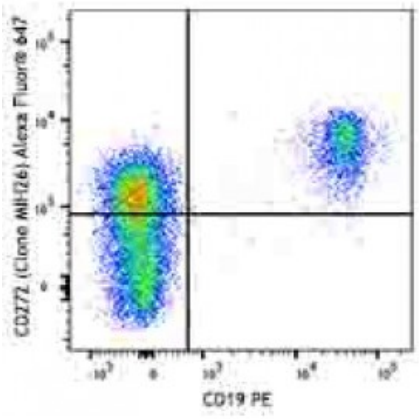
**Immunogen:** Human BTLA transfected cells

**Reactivity:** Human

**Preparation:** The antibody was purified by affinity chromatography and conjugated with Alexa Fluor® 647 under optimal conditions.

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

**Concentration:** 0.5



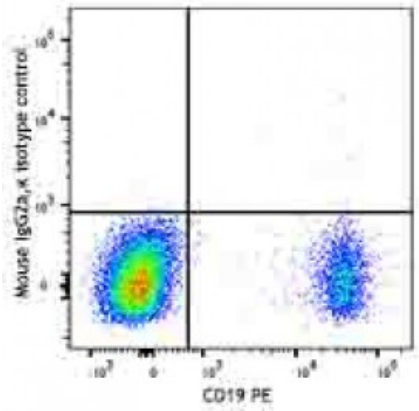

Human peripheral blood lymphocytes were stained with CD19 PE (Clone HIB19) and CD272 Alexa Fluor® 647 (clone MIH26) (top) or Mouse IgG2a, κ Alexa Fluor® 647 isotype control (bottom).

## 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 1.0 microL per million cells or 1.0 microL per 100 microL of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

* Alexa Fluor® 647 has a maximum emission of 668 nm when it is excited at 633 nm / 635 nm.




**Application Notes:** Additional reported applications (for the relevant formats) include: inhibition of T cell proliferation and cytokine production1. Clone MIH26 has agonistic activity on BTLA, resulting in the inhibition of activation.

**Application References:**
1. Otsuki N, *et al.* 2006. *Biochem. Bioph. Res. Co.* 344:1121.
2. Okano M, *et al.* 2008. *Clin. Exp. Allergy* 38:1891.

**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 $CD8^+$ 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.

**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.