SONY

Product Data Sheet

Brilliant Violet 421™ anti-human CD272 (BTLA)

Catalog # / Size: 2322555 / 25 tests
2322560 / 100 tests

Clone: MIH26

Isotype: Mouse IgG2a, κ

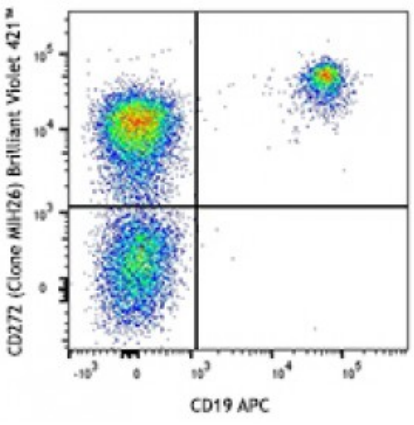
Immunogen: Human BTLA transfected cells

Reactivity: Human

Preparation: The antibody was purified by affinity chromatography and conjugated with Brilliant Violet 421™ under optimal conditions. The solution is free of unconjugated Brilliant Violet 421™ and unconjugated antibody.

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

Concentration: Lot-specific



Human peripheral blood lymphocytes stained with CD19 APC and CD272 (Clone MIH26) Brilliant Violet 421™ (top) or mouse IgG2a, κ Brilliant Violet 421™ isotype control (bottom).

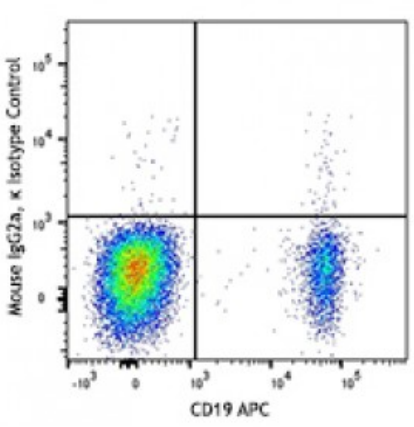
Applications:

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 microL per million cells or 5 microL per 100 microL of whole blood. It is recommended that the reagent be titrated for optimal performance for each application.

Brilliant Violet 421™ excites at 405 nm and emits at 421 nm. The standard bandpass filter 450/50 nm is recommended for detection. Brilliant Violet 421™ is a trademark of Sirigen Group Ltd.

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.