

Alexa Fluor® 488 anti-human CD357 (GITR)

Catalog # / Size: 2456050 / 100 tests
2456045 / 25 tests

Clone: 108-17

Isotype: Mouse IgG2a, κ

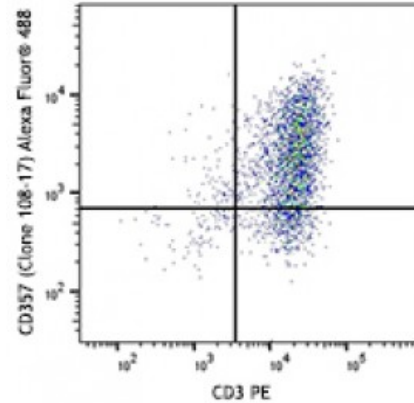
Immunogen: Recombinant human GITR-Fc chimera

Reactivity: Human

Preparation: The antibody was purified by affinity chromatography and conjugated with Alexa Fluor® 488 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

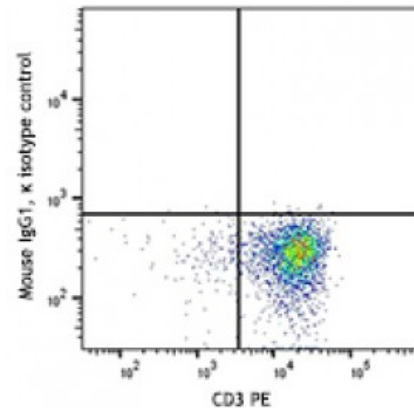


Human peripheral blood lymphocytes were activated for three days with PHA, and then stained with CD3 FITC and CD357 (GITR) (clone 108-17) Alexa Fluor™ 488 (top) or mouse IgG2a, κ Alexa Fluor™ 488 isotype control (bottom).

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.



* Alexa Fluor® 488 has a maximum emission of 519 nm when it is excited at 488 nm.

Description: GITR (glucocorticoid-induced TNF receptor family-regulated gene) is a 25 kD TNF receptor superfamily member (also known as AITR and TNFRSF18). GITR is expressed on activated lymphocytes and is upregulated by T cell receptor engagement. The cytoplasmic domain of GITR is homologous to CD40, 4-1BB and CD27 and has been shown to interact with TRAF 1-3, but not TRAF 5 or 6. GITR signaling has been shown to regulate T cell proliferation and TCR-mediated apoptosis, and to break immunological self-tolerance. GITR binds GITRL and is involved in the development of regulatory T cells and to regulate the activity of Th1 subsets.

Antigen References:

1. van der Werf N, *et al.* 2011. *J. Immunol.* 187:1411.
2. Shimizu J, *et al.* 2002. *Nat. Immunol.* 3:135.
3. McHugh RS, *et al.* 2002. *Immunity* 16:311.

4. Kwon B, *et al.* 1999.