

**PE anti-human CD357 (GITR)**

**Catalog # / Size:** 2456015 / 25 tests  
2456020 / 100 tests

**Clone:** 108-17

**Isotype:** Mouse IgG2a, κ

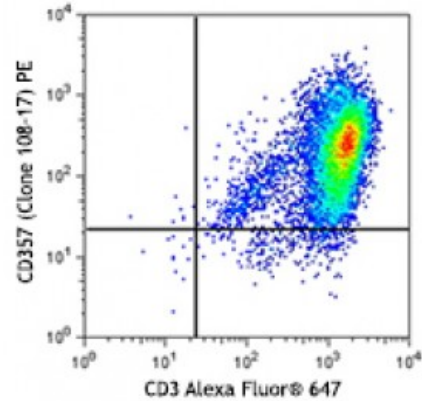
**Immunogen:** Recombinant human GITR-Fc chimera

**Reactivity:** Human

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

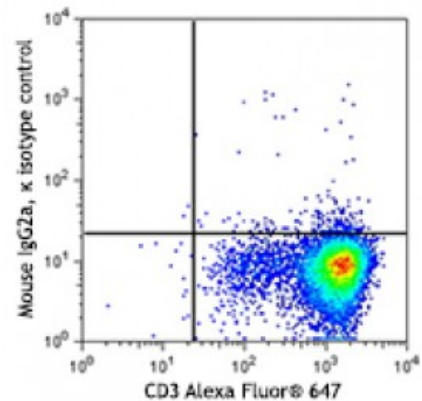


Human peripheral blood lymphocytes were activated for 3 days with PHA, and then stained with CD3 Alexa Fluor® 647 and CD357 (GITR) (clone 108-17) PE (top) or mouse IgG2a, κ PE isotype control (bottom).

**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.



**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.