## **Product Data Sheet**

## **APC anti-mouse CD357 (GITR)**

**Catalog # / Size:** 1231555 / 25 μg

1231560 / 100 µg

Clone: DTA-1

**Isotype:** Rat IgG2b,  $\lambda$ 

Immunogen: Mouse CD25+ CD4+ T cells

Reactivity: Mouse

**Preparation:** The antibody was purified by affinity

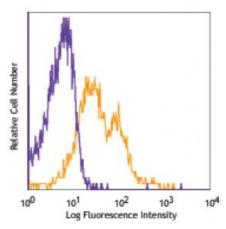
chromatography, and conjugated with APC under optimal conditions. The solution is free of unconjugated APC and

unconjugated antibody.

Formulation: Phosphate-buffered solution, pH 7.2,

containing 0.09% sodium azide.

Concentration: 0.2



C57BL/6 splenocytes stained with

DTA-1 APC

## **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 <0.25 microg per million colls in 100 microl, volume, It is

this reagent is ≤0.25 microg per million cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each

application.

Application References:

1. De Luca A, et al. 2007. J. Immunol. 179:5999. PubMed

**Description:** 

GITR (glucocorticoid-induced TNFR-related gene) is a member of the TNF receptor superfamily, also known as TNFRSF18 and AITR (in humans). It is expressed at low levels on resting T lymphocytes and at high levels on CD25<sup>+</sup> CD4<sup>+</sup> Tregs. The expression of GITR on T cells can be upregulated upon activation. Interaction of GITR with its ligand (GITRL) has been demonstrated to augment T cell activation, proliferation, cytokine production as well as MAPKs and NF-κB activation, and abrogate the inhibitory function of CD25<sup>+</sup> CD4<sup>+</sup> Tregs. *In vivo* activation of GITR causes development of autoimmune diseases and restores the suppressed immune response.

Antigen References:

1. Tone M, et al. 2003. Proc. Natl. Acad. Sci. USA 100:15059.

2. Shimizu J, et al. 2002 Nat. Immunol. 3:135.

3. Stephens GL, et al. 2004. J. Immunol. 173:5008.

4. McHugh RS, et al