

Anti-GITR [DTA-1] VivopureX 5 mg, 5 mg, Ab01060-2.0-VXM View online

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This chimeric mouse antibody was made using the variable domain sequences of the original Rat IgG2b format, for improved compatibility with existing reagents, assays and techniques.

Isotype and Format: Mouse IgG2a, Lambda

Clone Number: DTA-1

Alternative Name(s) of Target: CD357; AITR; GITR-D; Glucocorticoid-induced TNFR-related protein; TNFRSF18; Tumor necrosis factor receptor superfamily member 18

UniProt Accession Number of Target Protein: 035714

Published Application(s): agonist, IP, WB, FC

Published Species Reactivity: Mouse

Immunogen: This antibody was raised by immunising Wistar rats with the CD25+CD4+ T cell line. Hybridoma cells were generated by fusing spleen cells from the immunized rat with P3X63Ag8.653 myeloma cells, assessed for secreting antibodies capable of neutralizing in vitro CD25+CD4+ T cellmediated suppression of anti-CD3-stimulated T cell proliferation9, and then cloned. This DTA-1 clone secreted a monoclonal rat immunoglobulin G2a (IgG2a) that showed high neutralizing activity. **Specificity:** This antibody is specific for murine GITR, a member of the tumor necrosis factor receptor (TNFR) family that is expressed at low levels on unstimulated T cells, B cells, and macrophages. Upon activation, CD4+ and CD8+ T cells up-regulate GITR expression, whereas immunoregulatory T cells constitutively express high levels of GITR.

Application Notes: This antibody has been used in various FACS analyses for diverse immuno-oncological applications, such as to delineate how membrane-organizing protein moesin controls Treg differentiation and antitumor immunity via TGF- β signaling (Ansa-Addo et al, 2017), to suggest the therapeutic potential of TGF- β -induced Tregs (iTregs) in treating autoimmune gastritis (Nguyen et al, 2014), and to demonstrate that costimulation with the ligand of GITR elicites dose-dependent enrichment for cells of lower TCR affinity in the Treg cell repertoire (Mahmud et al, 2014). This antibody has also been used in Western Blots to elucidate the mechanism of human T-cell leukemia virus type 1 (HTLV-1) in the development of neoplastic and inflammatory diseases (Satou et al, 2011), and in immunoprecipitation analysis to aid the investigation of the role of GITR in dominant immunological self-tolerance maintained by CD25+CD4+ regulatory T cells (Shimizu et al, 2002). In addition, the anti-tumour effects of this agonistic anti-GITR antibody have been demonstrated in various murine models (Scirka et al, 2017; Coe et al, 2010; Cohen et al. 2010; Hu et al,

2008; Ko et al, 2005).

Antibody First Published in: Shimizu Jun et al. Stimulation of CD25+CD4+ regulatory T cells through GITR breaks immunological self-tolerance. Nat Immunol. 2002 Feb;3(2):135-42. PMID:11812990
Note on publication: Describe the original generation of this antibody and its subsequent applications in FACS, immunoprecipitation, in vitro and in vivo functional assays to show that GITR plays a key role in dominant immunological self-tolerance maintained by CD25+CD4+ regulatory T cells.

Product Form

Size: 5 mg VivopureX products are produced at high purity (>98%), low endotoxin (<0.5 EU/mg) and are formulated without preservatives. These antibodies are chimerized to have an Fc domain matching their target species to reduce immunogenicity and give you the optimal effector function for your experiment. As a result VivopureX products are the ideal choice for in vivo research applications.

- Purification: Protein A affinity purified
- Supplied In: PBS only.

Storage Recommendation: All VivopureX products are formulated in PBS only without addition of preservatives. To ensure optimal storage and prevent microbial contamination, only open and dispense under sterile conditions. Store at 4°C for up to 3 months. For longer storage, aliquot and store at -20°C. **Concentration:** >=1mg (see vial label for exact conc)

Important note – This product is for research use only. It is not intended for use in therapeutic or diagnostic procedures for humans or animals.