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GITRL Antibody

Catalog # ASC10348

Product Information

Application	WB, ICC, E
Primary Accession	Q9UNG2
Other Accession	Q9UNG2, 13124621
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	20308
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	GITRL antibody can be used for the detection of GITRL by Western blot at 1 g/mL. Antibody can also be used for immunocytochemistry starting at 10 g/mL.

Additional Information

Gene ID	8995
Other Names	GITRL Antibody: TL6, AITRL, GITRL, hGITRL, TL6, UNQ149/PRO175, Tumor necrosis factor ligand superfamily member 18, Activation-inducible TNF-related ligand, tumor necrosis factor (ligand) superfamily, member 18
Target/Specificity	TNFSF18;
Reconstitution & Storage	GITRL antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	GITRL Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	TNFSF18 (HGNC:11932)
Synonyms	AITRL, GITRL, TL6
Function	Cytokine that binds to TNFRSF18/AITR/GITR. Regulates T-cell responses. Can function as costimulator and lower the threshold for T- cell activation and T-cell proliferation. Important for interactions between activated T-lymphocytes and endothelial cells. Mediates activation of NF-kappa-B. Triggers increased phosphorylation of STAT1 and up-regulates expression of VCAM1 and ICAM1 (PubMed:23892569). Promotes leukocyte adhesion to

	endothelial cells (PubMed:23892569). Regulates migration of monocytes from the splenic reservoir to sites of inflammation (By similarity).
Cellular Location	Cell membrane; Single-pass type II membrane protein
Tissue Location	Expressed at high levels in the small intestine, ovary, testis, kidney and endothelial cells

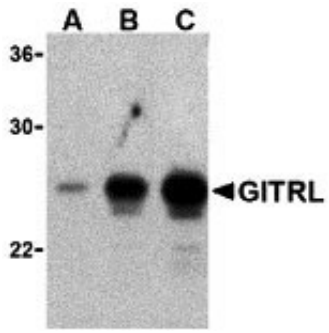
Background

GITRL Antibody: The tumor necrosis factor (TNF) and TNF receptor (TNFR) gene superfamilies regulate numerous biological functions including cell proliferation, differentiation, and survival through regulating the activation of the transcription factor NF-κB and various mitogen-activated protein kinases. The glucocorticoid-induced tumor necrosis factor receptor (GITR) is an emerging member of this family that is expressed on CD4+ CD25+ regulatory T cells and appears to have crucial immune regulation functions. Its ligand GITRL is expressed in endothelial and antigen-presenting cells and can activate NF-κB, induce both pro- and anti-apoptotic effects, inhibit the suppressive activity of regulatory T cells, and co-stimulate responder T cells through GITR. Dominant negative forms of NIK and TRAF2 expressed in transfected 293 cells substantially inhibited NF-κB activation, suggesting that the GITRL-GITR pathway involves both NIK and TRAF2.

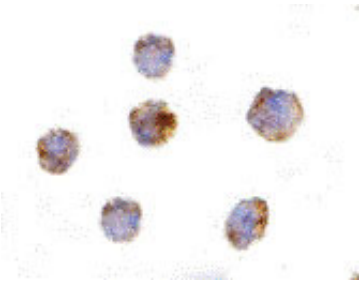
References

Gaur U, Aggarwal BB. Regulation of proliferation, survival and apoptosis by members of the TNF superfamily. Biochem. Pharmacol. 2003; 66:1403-8.
Ronchetti S, Nocentini G, Riccardi C, et al. Role of GITR in activation response of T lymphocytes.Blood 2002; 100:350-2.
Shimizu J, Yamakai S, Takahashi T, et al. Stimulation of CD25(+) CD4(+) regulatory T cells through GITR breaks immunological self- tolerance. Nat. Immunol. 2002;3:135-42.
Gurney AL, Marsters SA, Huang A, et al. Identification of a new member of the tumor necrosis factor family and its receptor, a human ortholog of mouse GITR. Curr. Biol. 1999; 9:215-218.

Images



Western blot analysis of (A) 5 ng, (B) 25 ng, and (C) 50 ng of purified recombinant GITRL with ITRL antibody at 1 µg/mL.



Immunocytochemistry of GITRL in THP-1 cells with GITRL antibody at 10 µg/mL.