GITR Protein, Human (Biotinylated, HEK293, His-Avi)

Cat. No.:	HY-P78137
Synonyms:	CD357; GITR; GITR-D; TNFRSF18; AITR
Species:	Human
Source:	HEK293
Accession:	Q9Y5U5 (Q26-E161)
Gene ID:	8784
Molecular Weight:	26-28 kDa

PROPERTIES

Biological Activity	Immobilized Human GITR Ligand Trimer, hFc Tag at 5μg/ml (100μl/well) on the plate. Dose response curve for Biotinylated Human GITR, His Tag with the EC_{50} of 0.14μg/ml determined by ELISA.
Appearance	Solution.
Formulation	Supplied as a 0.22 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background	GITR is expressed on regulatory T cells (Tregs) and some activated immune cells, including effector T lymphocytes, nature killer (NK) cells, and neutrophils[1]. The amino acid sequence of human GITR protein has low homology for mouse GITR protein. GITR does not have any enzymatic activity and signaling is propagated via recruiting TRAF-family members, specifically TRAF1, TRAF2 and TRAF5, to the GITR-signaling complex. The signaling is then mediated through NF-kB and MAPK pathways. GITR does not have any enzymatic activity and signaling is propagated via recruiting TRAF-family members, specifically TRAF1, TRAF2 and TRAF5, to the GITR-signaling complex. The signaling is then mediated through NF-kB and MAPK pathways, protecting T cells from TCR activation-induced cell death[2]. GITR (Glucocorticoid-induced TNFR-related protein, also known as TNFRSF18) is a type I transmembrane protein. GITR stimulates the proliferation of effector T-lymphocytes and partially reverses the immunosuppressive function of CD4+CD25+ Tregs[1]. GITR is activated by its ligand GITRL (TNFSF18). GITR induces NOS in murine macrophage in a time and dose-dependent manner[3]. GITR inhibits Multiple Myeloma (MM) cell proliferation in vitro and in vivo and induces apoptosis[4].

REFERENCES

[1]. Tian J, et al. The Role of GITR/GITRL Interaction in Autoimmune Diseases. Front Immunol. 2020 Oct 9;11:588682.

[2]. Krausz LT, et al. GITR-GITRL system, a novel player in shock and inflammation. ScientificWorldJournal. 2007 May 1;7:533-66.

[3]. Shin HH, et al. Recombinant glucocorticoid induced tumor necrosis factor receptor (rGITR) induces NOS in murine macrophage. FEBS Lett. 2002 Mar 13;514(2-3):275-80.

[4]. Liu Y, et al. Novel tumor suppressor function of glucocorticoid-induced TNF receptor GITR in multiple myeloma. PLoS One. 2013 Jun 13;8(6):e66982.

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