AITRL/TNFSF18 Protein, Mouse (HEK293, Fc)

Cat. No.:	HY-P78299
Synonyms:	TNFSF18; AITRL; TL6; GITRL; GITR Ligand
Species:	Mouse
Source:	HEK293
Accession:	Q7TS55 (T47-S173)
Gene ID:	240873
Molecular Weight:	50-60 kDa

PROPERTIES

Appearance	Lyophilized powder
Formulation	Lyophilized from 0.22 μm filtered solution in PBS (pH 7.4). Normally 8% trehalose is added as protectant before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

GITRL (AITRL), a type II transmembrane protein, is a ligand for glucocorticoid-induced TNFR-related protein (GITR). GITR, a member of the TNFR superfamily, is expressed in T cells, natural killer cells and some myeloid cells[1]. And murine GITRL has been detected on dendritic cells (DCs), monocytes, macrophages, B cells, endothelial cells, osteoclasts, and microglia cells [4].

When GITRL binds to GITR, GITR can produce costimulatory signals that regulate T-cell proliferation and effector functions. The interaction stimulates proliferation and cytokine production of both $CD4^+$ Teff and Treg cells, and drives antitumor activity of $CD8^+$ T cells[3]. Besides, GITRL plays a role in EC-activation and promotes adhesion in both mice and humans, which increases STAT-1 phosphorylation and the augmented expression of adhesion molecules such as VCAM-1 and ICAM-1 [2]. Mouse GITRL can activate signal transduction, including inducing a tolerogenic effect in DCs and pro-inflammatory stimuli in macrophages[7].

Mouse GITRL shares < 55% common aa identity with human. Murine GITRL exists as a dimer[4]GITR/GITRL interaction plays a role in the pathogenesis of tumor, inflammation, as well as autoimmune diseases[1]

REFERENCES

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

[2]. Lacal PM, et al. Glucocorticoid-induced tumor necrosis factor receptor family-related ligand triggering upregulates vascular cell adhesion molecule-1 and intercellular adhesion molecule-1 and promotes leukocyte adhesion. J Pharmacol Exp Ther. 2013 Oct;347(1):164-72.

[3]. Wang F, et al. Structures of mouse and human GITR-GITRL complexes reveal unique TNF superfamily interactions. Nat Commun. 2021 Mar 2;12(1):1378.

[4]. Placke T, et al. Glucocorticoid-induced TNFR-related (GITR) protein and its ligand in antitumor immunity: functional role and therapeutic modulation. Clin Dev Immunol. 2010;2010:239083.

[5]. Tian J, et al. Increased GITRL Impairs the Function of Myeloid-Derived Suppressor Cells and Exacerbates Primary Sjögren Syndrome. J Immunol. 2019 Mar 15;202(6):1693-1703.

[6]. Chen M, et al. IFN-β induces the proliferation of CD4+CD25+Foxp3+ regulatory T cells through upregulation of GITRL on dendritic cells in the treatment of multiple sclerosis. J Neuroimmunol. 2012 Jan 18;242(1-2):39-46.

[7]. Nocentini G, et al. Pharmacological modulation of GITRL/GITR system: therapeutic perspectives. Br J Pharmacol. 2012 Apr;165(7):2089-99.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA