AITRL/TNFSF18 Protein, Human

Cat. No.:	HY-P7318
Synonyms:	rHuActivation-inducible TNF-related Ligand/AITRL; TNFSF18; GITRL; TL-6; rHuAITRL
Species:	Human
Source:	E. coli
Accession:	Q9UNG2 (E52-I176)
Gene ID:	8995
Molecular Weight:	Approximately 14.3 kDa

PROPERTIES

AA Sequence	M E T A K E P C M A K F G P L P S K W Q M A S S E P P C V N K V S D W K L E I L Q N G L Y L I Y G Q V A P N A N Y N D V A P F E V R L Y K N K D M I Q T L T N K S K I Q N V G G T Y E L H V G D T I D L I F N S E H Q V L K N N T Y W G I I L L A N P Q F I
Biological Activity	The ED_{50} is <5 ng/mL as measured by PMBC, corresponding to a specific activity of >2 × 10^5 units/mg.
Appearance	Lyophilized powder.
Formulation	Lyophilized after extensive dialysis against 50 mM Tris, pH 8.0.
Endotoxin Level	<0.2 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
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. And GITRL is mainly expressed on antigen presenting cells (B cells, dendritic cells), macrophages and endothelial cells (ECs)[1]. 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].

Human GITRL shares < 55% common aa identity with mouse. Human GITRL consists of cytoplasmic domain (M1-W27), helical domain (L28-F48), and extracellular domain (L49-S177). Human GITRL is a trimer, but can also be a monomer or assemble in other multimeric structures[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]. Park MS, et al. The association of the activation-inducible tumor necrosis factor receptor and ligand with lumbar disc herniation. Yonsei Med J. 2007 Oct 31;48(5):839-46.

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