AITRL/TNFSF18 Protein, Human (HEK293, Fc)

Cat. No.:	HY-P76110
Synonyms:	Tumor necrosis factor ligand superfamily member 18; AITRL; hGITRL; TNFSF18; TL6
Species:	Human
Source:	HEK293
Accession:	Q9UNG2/NP_005083.3 (Q50-S177)
Gene ID:	8995
Molecular Weight:	Approximately 40.9 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized a 0.2 μm filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants 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. 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.

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