

PRODUCT INFORMATION



GITRL Extracellular Domain (human, recombinant)

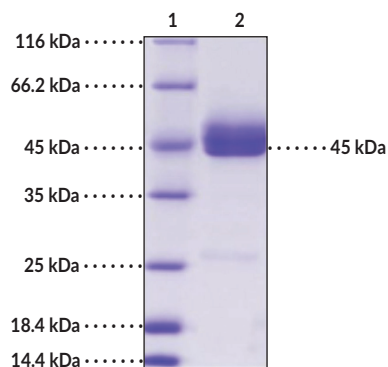
Item No. 32018

Overview and Properties

Synonyms:	AITRL, Glucocorticoid-induced TNF-Related Ligand, TNFSF18, Tumor Necrosis Factor Ligand Superfamily Member 18
Source:	Recombinant C-terminal mouse IgG2a Fc-tagged human GITRL expressed in HEK293 cells
Amino Acids:	72-199
Uniprot No.:	Q9UNG2
Molecular Weight:	40.9 kDa
Storage:	-80°C (as supplied)
Stability:	≥1 year
Purity:	≥90% estimated by SDS-PAGE
Supplied in:	Lyophilized from sterile PBS, pH 7.4
Endotoxin Testing:	<1.0 EU/μg, determined by the LAL endotoxin assay

Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Image



Lane 1: MW Markers
Lane 2: GITRL Extracellular Domain

SDS-PAGE Analysis of GITRL Extracellular Domain.

This protein has a calculated molecular weight of 40.9 kDa. It has an apparent molecular weight of approximately 45 kDa by SDS-PAGE under reducing conditions due to glycosylation.

WARNING
THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA
This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the complete Safety Data Sheet, which has been sent via email to your institution.

WARRANTY AND LIMITATION OF REMEDY
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Description

Glucocorticoid-induced TNF-related ligand (GITRL), also known as TNF superfamily member 18 (TNFSF18), is a type II transmembrane glycoprotein and member of the TNF superfamily with roles in adaptive immunity.¹ It is composed of an N-terminal cytoplasmic domain, a transmembrane domain, and a C-terminal extracellular domain that binds to its receptor GITR (Item No. 32017) on T cells to regulate their activity. GITRL is expressed on professional and non-professional antigen-presenting cells, including B cells, monocytes, unstimulated myeloid dendritic cells, and plasmacytoid dendritic cell precursors, as well as microvascular-derived endothelial cells. GITRL co-stimulation increases activation and proliferation of T cell receptor-triggered effector T cells. It also enhances the activity of effector T cells by binding to regulatory T cells and inhibiting their activity. Upregulation of dendritic cell *Gitrl* levels suppresses regulatory T cell function, enhances effector T cell function, and delays tumor progression in a murine Lewis lung carcinoma model.² Serum and synovial fluid levels of GITRL are increased and positively correlated with autoantibody production in patients with rheumatoid arthritis.³ Serum levels of GITRL are positively correlated with disease progression in patients with Sjögren syndrome.⁴ Cayman's GITRL Extracellular Domain (human, recombinant) protein is a disulfide-linked homodimer. The reduced monomer, comprised of the GITRL extracellular domain (amino acids 72-199) fused to mouse IgG2a Fc at its C-terminus, consists of 361 amino acids and has a calculated molecular weight of 40.9 kDa. As a result of glycosylation, the monomer migrates at approximately 45 kDa by SDS-PAGE under reducing conditions.

References

1. Nocentini, G. and Riccardi, C. GITR: A modulator of immune response and inflammation. *Therapeutic Targets of the TNF Superfamily*. Grewal, I.S., editor, 1st edition, Springer (2009).
2. Tian, J., Ma, J., Ma, K., et al. Up-regulation of GITRL on dendritic cells by WGP improves anti-tumor immunity in murine Lewis lung carcinoma. *PLoS One* **7(10)**, e46936 (2012).
3. Li, L., Wen, W., Jia, R., et al. GITRL is associated with increased autoantibody production in patients with rheumatoid arthritis. *Clin. Rheumatol.* **35(9)**, 2195-2202 (2016).
4. Tian, J., Rui, K., Hong, Y., et al. Increased GITRL impairs the function of myeloid-derived suppressor cells and exacerbates primary Sjögren syndrome. *J. Immunol.* **202(6)**, 1693-1703 (2019).

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