PRODUCT INFORMATION

GITRL Extracellular Domain (human, recombinant)

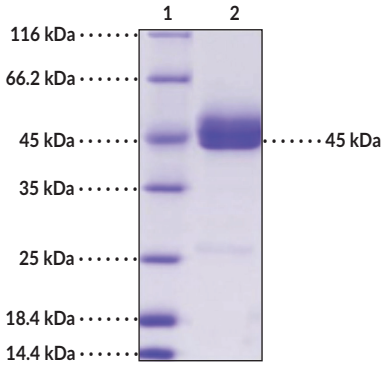
Item No. 32018

Overview and Properties

Synonyms:	AITRL, Glucocorticoid-induced TNF-Related Ligand, TNFSF18, Tumor Necrosis Factor Ligand Superfamily Member 18
Source:	Active 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.

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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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