PRODUCT INFORMATION

TIGIT Extracellular Domain (human, recombinant)

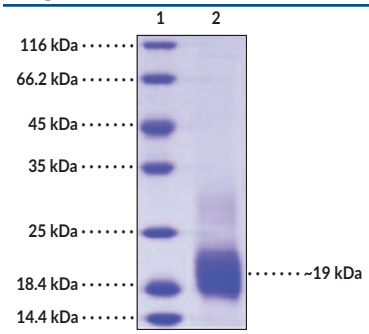
Item No. 32081

Overview and Properties

Synonyms:	T Cell Immunoreceptor with Ig and ITIM Domains, V-set and Immunoglobulin Domain-Containing Protein 9, V-set and Transmembrane Domain-Containing Protein 3, VSig9, Vstm3, Washington University Cell Adhesion Molecule, WUCAM
Source:	Active recombinant human C-terminal His-tagged TIGIT expressed in HEK293 cells
Amino Acids:	22-138
Uniprot No.:	Q495A1
Molecular Weight:	14.2 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
Bioactivity:	See figures for details

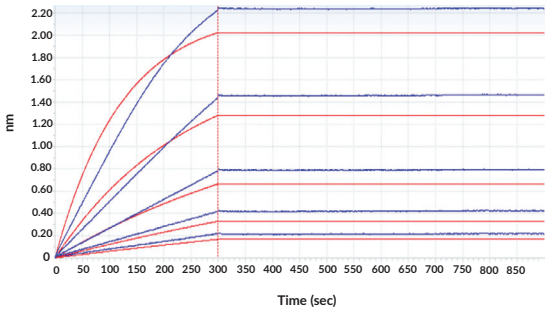
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images

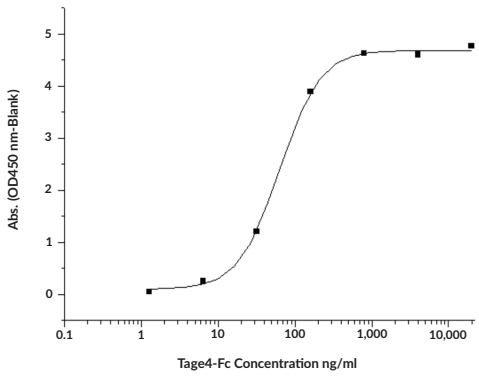


Lane 1: MW Markers
Lane 2: TIGIT Extracellular Domain

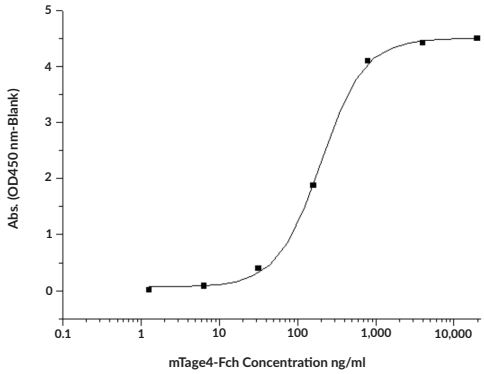
SDS-PAGE Analysis of TIGIT Extracellular Domain. This protein has a calculated molecular weight of 14.2 kDa. It has an apparent molecular weight of approximately 19 kDa in SDS-PAGE under reducing conditions due to glycosylation.



Octet RED System on TIGIT Extracellular Domain. Using the Octet RED System, the affinity constant (K_d) of TIGIT Extracellular Domain, bound to MITG7192A was 0.1 nM.



TIGIT Extracellular Domain Binding in a Functional ELISA. Measured by its ability to bind with human Tage4-Fc in a functional ELISA.



TIGIT Extracellular Domain Binding in a Functional ELISA. Measured by its ability to bind with mouse Tage4-Fch in a functional ELISA.

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Description

T cell immunoreceptor with Ig and ITIM domains (TIGIT) is a type I transmembrane immune receptor.[1] It is composed of an N-terminal extracellular immunoglobulin variable (IgV) domain, a transmembrane domain, and a C-terminal cytoplasmic tail that contains an immunoglobulin tail tyrosine-like (ITT-like) phosphorylation motif and an immunoreceptor tyrosine-based inhibition motif (ITIM).[2] The ITT-like and ITIM motifs are important for the signaling and inhibitory functions of TIGIT.[1] It is expressed primarily on natural killer (NK) cells but also expressed on activated, memory, and follicular T helper cells, as well as certain regulatory T cells and exhausted T cells. TIGIT is a co-inhibitory molecule that acts in opposition to the co-stimulatory receptor CD226 (Item Nos. 32071 | 32072), preventing its interaction with their shared ligands, CD155/Tage4 and CD112/nectin-2, on antigen-presenting cells (APCs), infected cells, and tumor cells. TIGIT on NK cells inhibits NK cell-mediated APC cell killing and lysis, as well as APC-induced cytokine release that, in turn, inhibits IFN-γ secretion from NK cells.[3] On T cells, TIGIT inhibits T cell cytokine production, proliferation, and T cell receptor signaling.[1] It also associates with the adapter proteins Grb2 and β-arrestin 2 to recruit SHIP1 for inhibition of NK or T cell activation. In addition to its cell extrinsic activities, TIGIT has cell intrinsic activity, binding directly to CD226 to disrupt its function. Anti-TIGIT antibodies delay the growth of tumors, protect against metastasis, and decrease tumor burden in mouse models of cancer with an increased anticancer effect when used in combination with PD-1 inhibitors.[3] *TIGIT*$^{-/-}$ mice have reduced tumor growth and increased survival compared to wild-type mice. The expression of *TIGIT* on immune cells in melanoma and gastric cancer patients is associated with metastasis and shorter overall survival. Cayman's TIGIT Extracellular Domain (human, recombinant) protein can be used for binding assay and ELISA applications. This protein consists of 128 amino acids, has a calculated molecular weight of 14.2 kDa, and a predicted N-terminus of Met22 after signal peptide cleavage. By SDS-PAGE, the apparent molecular mass of the protein is approximately 19 kDa due to glycosylation.

References

1. Joller, N. and Kuchroo, V.K. Tim-3, Lag-3, and TIGIT. *Emerging concepts targeting immune checkpoints in cancer and autoimmunity.* Yoshimura, A., editor, 1st edition, *Springer International Publishing* (2017).
2. Liu, S., Zhang, H., Li, M., *et al.* Recruitment of Grb2 and SHIP1 by the ITT-like motif of TIGIT suppresses granule polarization and cytotoxicity of NK cells. *Cell Death Differ.* **20(3)**, 456-464 (2013).
3. Harjunpää, H. and Guillerey, C. TIGIT as an emerging immune checkpoint. *Clin. Exp. Immunol.* **200(2)**, 108-119 (2020).

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