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Biotin Human GITR (TNFRSF18) Protein (C-His-Avi)

Catalog Number:	811303, 811304
Size:	25 ug, 100 ug
Target Name:	TNFRSF18, AITR, GITR, CD357
Regulatory Status:	RUO

PRODUCT DETAILS

Application:	ELISA, BLI
Format:	Liquid, Biotinylated
Expression Host:	CHO
Species:	Human
Sources:	Recombinant Human Human GITR/TNFRSF18 (Gln26-Glu161) with C-terminus His-Avi-tag is expressed in CHO cell. This protein was site-specifically labeled with Biotin by BirA ligase.
Accession Number:	Q9Y5U5
Molecular Weight:	The protein has a predicted molecular weight of 18.1 kDa. Under DTT-reducing conditions, it migrates at approximately 25 kDa on SDS-PAGE.
Affinity Tag:	C-His-Avi
Purity:	>95% based on SDS-PAGE under reducing condition
Formulation:	1xPBS buffer, pH7.4, 0.22 μm filtered
Endotoxin level:	Less than 0.1 EU/μg protein as determined by the LAL method
Protein Concentration:	25μg size is bottled at 0.2mg/mL concentration. 100 μg size is supplied at a lot-specific concentration.
Storage and Handling:	Briefly centrifuge the vial upon receipt. An unopened vial can be stored at 4°C for up to 2 weeks, or at -20°C or below for up to six months. The protein may be further diluted to 0.1 mg/mL using 0.22 μm-filtered PBS buffer (pH 7.4). For long-term storage, the diluted stock solution should be aliquoted and stored at ≤ –70°C to minimize freeze-thaw cycles. If additional dilution is required, carrier proteins such as FBS or BSA should be added to maintain protein stability.
Recommended Usage:	For detection, use a secondary reagent with this product.

BACKGROUND INFORMATION

Glucocorticoid-induced tumor necrosis factor receptor–related protein (GITR), also known as CD357 or TNFRSF18, is a co-stimulatory receptor that plays an important role in regulating T cell activation, survival, and immune balance. GITR is constitutively expressed at high levels on regulatory T cells (Tregs) and at lower levels on naïve conventional T cells, with expression rapidly upregulated following T cell activation. It is also expressed on natural killer (NK) cells and some myeloid populations. Through its signaling, GITR influences both effector and regulatory arms of the immune system.

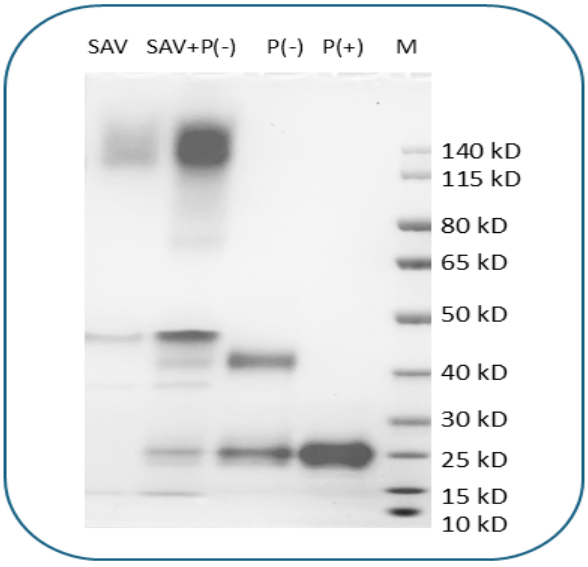
Structurally, GITR is a type I transmembrane glycoprotein and a member of the tumor necrosis factor receptor (TNFR) superfamily. Its extracellular region contains cysteine-rich domains typical of TNFR family members that mediate ligand binding. GITR has a single transmembrane segment and a cytoplasmic tail that lacks intrinsic enzymatic activity but recruits TNF receptor–associated factors (TRAFs), particularly TRAF2 and TRAF5. These adaptor proteins initiate downstream signaling cascades, including activation of NF-κB and MAPK pathways, which promote cell survival, proliferation, and cytokine production.

The primary ligand for GITR is GITR ligand (GITRL), also known as TNFSF18, which is expressed on activated antigen-presenting cells such as dendritic cells, macrophages, and B cells, as well as on endothelial cells in inflamed tissues. Engagement of GITR by GITRL provides a co-stimulatory signal that enhances effector T cell activation and resistance to suppression. In regulatory T cells, GITR signaling can transiently reduce suppressive function, thereby shifting the immune balance toward activation in certain contexts.

GITR has been implicated in a variety of disease settings. In autoimmune and inflammatory diseases, heightened GITR signaling may contribute to excessive T cell activation and tissue damage by weakening regulatory control. In allergic disease, GITR can promote Th2 responses and inflammation. In cancer, GITR is highly expressed on tumor-infiltrating Tregs, where it contributes to immune suppression within the tumor microenvironment. At the same time, GITR expression on effector T cells provides an opportunity to enhance anti-tumor immunity.

Therapeutically, GITR is an active target in immuno-oncology. Agonistic antibodies targeting GITR aim to simultaneously stimulate effector T cells and attenuate Treg-mediated suppression within tumors, thereby enhancing anti-tumor immune responses. These agents are being evaluated alone and in combination with immune checkpoint inhibitors. Conversely, strategies to inhibit GITR–GITRL interactions are being explored for the treatment of autoimmune and inflammatory diseases, highlighting GITR's dual relevance as both a driver of immunity and a contributor to immune-mediated pathology.

PRODUCT DATA



Human GITR (TNFRSF18) Protein (C-His-Avi) was biotinylated in vitro using BirA ligase. SDS-PAGE analysis under reducing (P+) and non-reducing (P-) conditions shows the protein has a purity greater than 95%. A gel shift assay using co-incubation with streptavidin indicates that the biotinylation efficiency of the GITR protein exceeds 80%.

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