

**GITR**  
**Catalog # PVGS1565****Specification**

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**GITR - Product Information**

Primary Accession [Q9Y5U5-1](#)  
**Species**  
Human

**Sequence**  
Gln26-Glu161

**Purity**  
> 95% as analyzed by SDS-PAGE

**Endotoxin Level**  
< 0.1 EU/ µg of protein by gel clotting method

**Biological Activity**  
Immobilized GITR, hFc, Human at 5.0 µg/ml (100 µl/well) can bind biotinylated GITR Ligand, hFc, Human (Cat. No.: Z03446) when detected by Streptavidin-HRP.

**Expression System**  
HEK 293

Formulation **Lyophilized from a 0.2 µm filtered solution in PBS, 5% trehalose and mannitol.**

**Reconstitution**  
It is recommended that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. Reconstitute the lyophilized powder in ddH<sub>2</sub>O or PBS up to 100 µg/ml.

**Storage & Stability**  
Upon receiving, this product remains stable for up to 6 months at lower than -70°C. Upon reconstitution, the product should be stable for up to 1 week at 4°C or up to 3 months at -20°C. For long term storage it is recommended that a carrier protein (example 0.1% BSA) be added. Avoid repeated freeze-thaw cycles.

**GITR - Additional Information**

**Target Background**  
GITR (glucocorticoid-induced tumor necrosis factor receptor), also known as AITR and TNFRSF18, is a 40 kDa transmembrane glycoprotein that functions in immune regulation. Mature human GITR consists of a 137 amino acid extracellular domain (ECD) with three tandem TNFR cysteine-rich repeats, a 21 aa transmembrane segment, and a 58 aa cytoplasmic domain. Within the ECD, human GITR shares 55% and 60% aa sequence identity with mouse and rat GITR, respectively. Alternative splicing generates an isoform with a short deletion in the cytoplasmic domain and a potentially secreted isoform that is substituted within the third TNFR repeat and lacks the transmembrane and cytoplasmic regions. GITR is expressed on CD4<sup>+</sup> CD25<sup>+</sup> regulatory T cells (Treg) as well as on subsets of thymocytes, lymph node

cells, and splenocytes, and it is upregulated on antigen-activated conventional CD4<sup>+</sup> and CD8<sup>+</sup> T cells. GITR binding by GITR Ligand/TNFSF18 costimulates the proliferation and activation of CD4<sup>+</sup> or CD8<sup>+</sup> conventional T cells. It also induces the proliferation of Treg but inhibits the ability of Treg to suppress immune responses. This can result in the development of autoimmunity, increased tumor cell killing by effector T cells, and increased inflammation in arthritis, allergic asthma, and inflammatory bowel disease. GITR is also expressed on sympathetic neurons where it enhances NGF-induced neurite outgrowth and branching.

## **GITR - Protein Information**

## **GITR - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## **GITR - Images**