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GITR

Catalog # PVGS1565

Product Information

Primary Accession	Q9Y5U5-1
Species	Human
Sequence	Gln26-Glu161
Purity	> 95% as analyzed by SDS-PAGE
Endotoxin Level	
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_2O 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.

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 $CD4^+$ $CD25^+$ regulatory T cells (Treg) as well as on subsets of thymocytes, lymph node cells, and splenocytes, and it is upregulated on antigen-activated conventional $CD4^+$ and $CD8^+$ T cells. GITR binding by GITR Ligand/TNFSF18 costimulates the proliferation and activation of $CD4^+$ or $CD8^+$ 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.

Protein Information