Canine GITR / TNFRSF18 Protein, His Tag

Catalog # GIR-C52H6

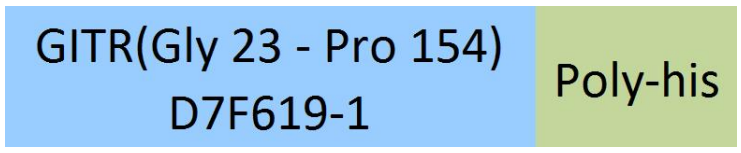
Synonym

AITR,GITR,TNFRSF18,CD357

Source

Canine GITR, His Tag(GIR-C52H6) is expressed from human 293 cells (HEK293). It contains AA Gly 23 - Pro 154 (Accession # D7F619-1).
Predicted N-terminus: Gly 23

Molecular Characterization



This protein carries a polyhistidine tag at the C-terminus.

The protein has a calculated MW of 15.8 kDa. The protein migrates as 20-25 kDa under reducing (R) condition (SDS-PAGE) due to glycosylation.

Endotoxin

Less than 1.0 EU per μg by the LAL method / rFC method.

Purity

>90% as determined by SDS-PAGE.

Formulation

Lyophilized from 0.22 μm filtered solution in PBS, pH7.4 with trehalose as protectant.

Contact us for customized product form or formulation.

Reconstitution

Please see Certificate of Analysis for specific instructions.

For best performance, we strongly recommend you to follow the reconstitution protocol provided in the CoA.

Storage

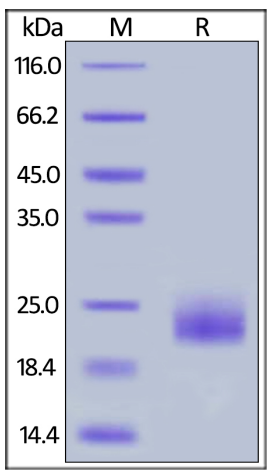
For long term storage, the product should be stored at lyophilized state at -20°C or lower.

Please avoid repeated freeze-thaw cycles.

This product is stable after storage at:

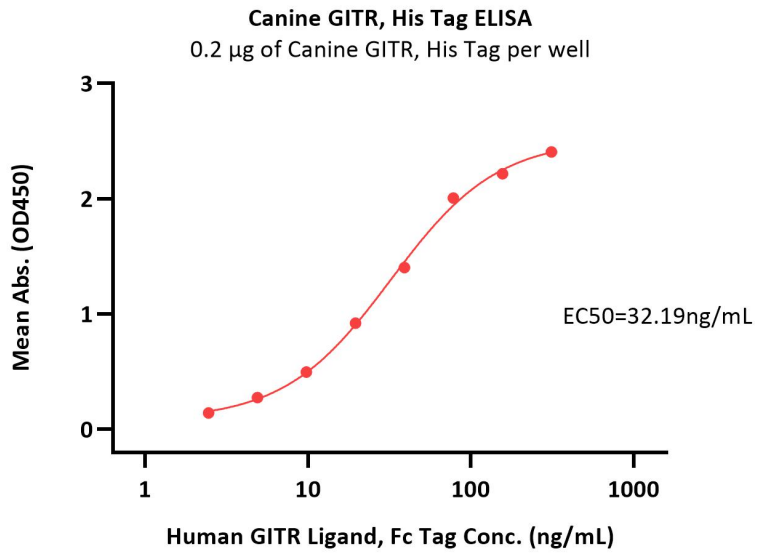
- -20°C to -70°C for 12 months in lyophilized state;
- -70°C for 3 months under sterile conditions after reconstitution.

SDS-PAGE



Canine GITR, His Tag on SDS-PAGE under reducing (R) condition. The gel was stained with Coomassie Blue. The purity of the protein is greater than 90%.

Bioactivity-ELISA



Immobilized Canine GITR, His Tag (Cat. No. GIR-C52H6) at 2 μg/mL (100 μL/well) can bind Human GITR Ligand, Fc Tag (Cat. No. GIL-H526a) with a linear range of 2-40 ng/mL (QC tested).

Background

Glucocorticoid-induced TNFR-related protein (GITR) is also known as Tumor necrosis factor receptor superfamily member 18 (TNFRSF18), activation-inducible TNFR family receptor (AITR), CD antigen CD357, which is a member of the tumor necrosis factor receptor (TNF-R) superfamily. GITR is receptor for TNFSF18, which seems to be involved in interactions between activated T-lymphocytes and endothelial cells and in the regulation of T-cell receptor-mediated cell death. GITR also mediated NF-kappa-B activation via the TRAF2/NIK pathway.