

GITR/TNFRSF18 Protein, Human, Recombinant (aa 26-161, His & Avi), Biotinylated

General Information

Synonyms: GITR-D;CD357;AITR;GITR;TNFRSF18

Protein Construction: Gln26-Glu161

Species: Human

Expression Host: HEK293 Cells

Accession: Q9Y5U5-1

Molecular Weight: 17.2 kDa (predicted). Due to glycosylation, the protein migrates to 26-28 kDa based on Tris-Bis PAGE result.

QC Testing

Biological Activity: Immobilized Human GITR Ligand Trimer, hFc Tag at 5 μ g/ml (100 μ l/well) on the plate. Dose response curve for Biotinylated Human GITR, His Tag with the EC50 of 77.9ng/ml determined by ELISA.

Purity: > 95% as determined by Tris-Bis PAGE; > 95% as determined by HPLC

Endotoxin: < 1 EU/ μ g by the LAL method.

Formulation: Lyophilized from a solution filtered through a 0.22 μ m filter, containing PBS (pH 7.4). Typically, 8% trehalose is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in distilled water. The product concentration should not be less than 100 μ g/ml. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Shipping:

In general, Lyophilized powders are shipping with blue ice.

Protein 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. GITR is a receptor for TNFSF18. Seems to be involved in interactions between activated T-lymphocytes and endothelial cells and in the regulation of T-cell receptor-mediated cell death. Mediated NF-kappa-B activation via the TRAF2/NIK pathway.

Reference

Knee D A, et al. Rationale for anti-GITR cancer immunotherapy[J]. European Journal of Cancer, 2016, 67:1-10.

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