

GITR/TNFRSF18 Protein, Mouse, Recombinant (aa 22-153, His)

General Information

Synonyms:	TNFRSF18;AITR;GITR-D;CD357;GITR
Protein Construction:	Ser22-His153
Species:	Mouse
Expression Host:	HEK293 Cells
Accession:	Q8C4K3
Molecular Weight:	15.2 kDa (predicted). Due to glycosylation, the protein migrates to 30-40 kDa based on Tris-Bis PAGE result.

QC Testing

Biological Activity:	Activity has not been tested. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 95% as determined by Tris-Bis PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm 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 μg/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.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry 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. GIRT 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]]]. European Journal of Cancer, 2016, 67:1-10.

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Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481