Recombinant Human TNF alpha (N-6His)

Catalog No.: RP0080

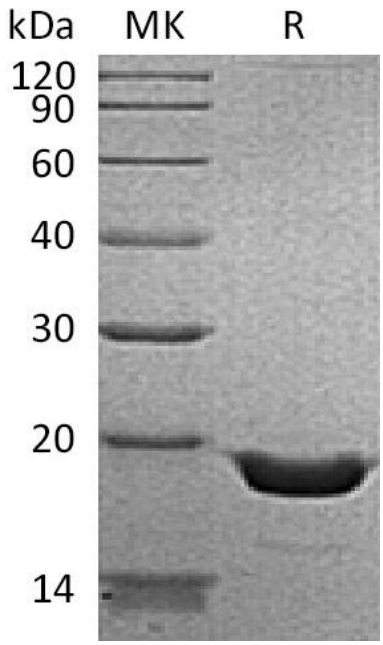
Basic Information

Information	
Source	*Human Cells*
Description	Recombinant Human Tumor Necrosis Factor Alpha is produced by our E.coli expression system and the target gene encoding Gly57-Leu233 is expressed with a 6His tag at the N-terminus.
Accession	P01375
Known As	Tumor Necrosis Factor; Cachectin; TNF-Alpha; Tumor Necrosis Factor Ligand Superfamily Member 2; TNF-a; TNF; TNFA; TNFSF2
Predicted Mol Mass	21.8 KDa
Apparent Mol Mass	18 KDa, reducing conditions

Properties	
Formulation	Lyophilized from a 0.2 μm filtered solution of 20mM Histidine, 8 %Trehalose, 0.05%Tween80, pH5.0.
Storage	Lyophilized protein should be stored at ≤ -20°C, stable for one year after receipt. Reconstituted protein solution can be stored at 2-8°C for 2-7 days. Aliquots of reconstituted samples are stable at ≤ -20°C for 3 months.
Endotoxin	< 1 EU/μg as determined by LAL test.
Reconstitution	Always centrifuge tubes before opening.Do not mix by vortex or pipetting. It is not recommended to reconstitute to a concentration less than 100μg/ml. Dissolve the lyophilized protein in distilled water. Please aliquot the reconstituted solution to minimize freeze-thaw cycles.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.

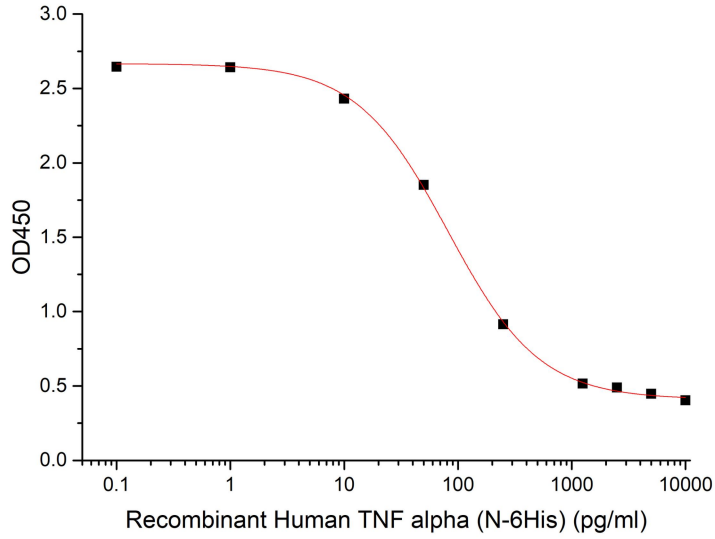
Experimental Data

Purity-SDS-PAGE



Greater than 95% as determined by reducing SDS-PAGE. (QC verified)

Bioactivity-Cell Based Assay



Measured in a cytotoxicity assay using L-929 mouse fibroblast cells in the presence of the metabolic inhibitor actinomycin D. The ED50 for this effect is 30-150 pg/ml. (QC verified)

Background

Tumor Necrosis Factor-α (TNF-α) is secreted by macrophages, monocytes, neutrophils, T-cells, and NK-cells following stimulation by bacterial LPS. Cells expressing CD4 secrete TNF-α while cells that express CD8 secrete little or no TNF-α. Synthesis of TNF-α can be induced by many different stimuli including interferons, IL2, and GM-CSF. The clinical use of the potent anti-tumor activity of TNF-α has been limited by the proinflammatory side effects such as fever, dose-limiting hypotension, hepatotoxicity, intravascular thrombosis, and hemorrhage. Designing clinically applicable TNF-α mutants with low systemic toxicity has been of intense pharmacological interest. Human TNF-α that binds to murine TNF-R55 but not murine TNF-R7, exhibits retained anti-tumor activity and reduced systemic toxicity in mice compared with murine TNF-α, which binds to both murine TNF receptors. Based on these results, many TNF-α mutants that selectively bind to TNF-R55 have been designed. These mutants displayed cytotoxic activities on tumor cell lines in vitro and have exhibited lower systemic toxicity in vivo. Recombinant Human TNF-α High Active Mutant differs from the wild-type by amino acid subsitution of amino acids 1-7 with Arg8, Lys9, Arg10 and Phe157. This mutant form has been shown to have increased activity with less inflammatory side effects in vivo.