Recombinant Human Tumor Necrosis Factor alpha is produced by our E.coli expression system and the target gene encoding is expressed.

**DESCRIPTION**

Accession #: P01375
Known as: Tumor Necrosis Factor; Cachectin; TNF-Alpha; Tumor Necrosis Factor Ligand Superfamily Member 2; TNF-α; TNF; TNFA; TNFSF2

**FORMULATION**

Lyophilized from a 0.2 μm filtered solution of 20mM PB, 150mM NaCl, pH 7.4.

**SHIPPING**

The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature listed below.

**STORAGE**

Lyophilized protein should be stored at < -20°C, though stable at room temperature for 3 weeks. Reconstituted protein solution can be stored at 4-7°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.

**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.

**QUALITY CONTROL**

Bioactivity* Measured by the cytolysis of murine L929 cells in the presence of Actinomycin D. ED50 is less than 0.01 ng/ml. Specific Activity of 1.0 x 10^8 IU/mg.

Purity: Greater than 95% as determined by reducing SDS-PAGE.

Endotoxin: Less than 0.1 ng/μg (1 IEU/μg).

**AMINO ACID SEQUENCE**

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 substitution 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.

**BACKGROUND**

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