

DESCRIPTION

Source *E. coli*-derived human TNF-alpha protein
Val77-Leu233, with and without an N-terminal Met
Accession # P01375

N-terminal Sequence Analysis Met & Val77

Predicted Molecular Mass 17 kDa

SPECIFICATIONS

SDS-PAGE 17 kDa, reducing conditions

Activity Measured in a cytotoxicity assay using L-929 mouse fibroblast cells in the presence of the metabolic inhibitor actinomycin D. Matthews, N. and M.L. Neale (1987) in *Lymphokines and Interferons, A Practical Approach*. Clemens, M.J. *et al.* (eds): IRL Press. 221. The ED₅₀ for this effect is 25-100 μ g/mL.

Endotoxin Level <0.10 EU per 1 μ g of the protein by the LAL method.

Purity >97%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 μ m filtered solution in PBS. *Small pack size (5 & 20 μ g) is supplied as a 0.2 μ m filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 0.1-1 mg/mL in sterile PBS.

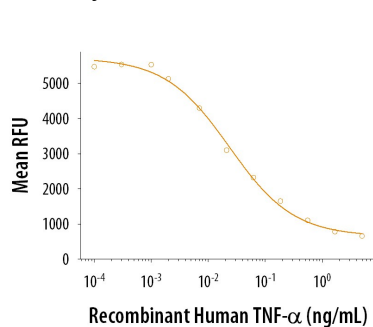
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (5 & 20 μ g) is shipped with dry ice or equivalent. Upon receipt, store it immediately at -20 to -70 $^{\circ}$ C.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 $^{\circ}$ C as supplied.
- 1 month, 2 to 8 $^{\circ}$ C under sterile conditions after reconstitution.
- 3 months, -20 to -70 $^{\circ}$ C under sterile conditions after reconstitution.

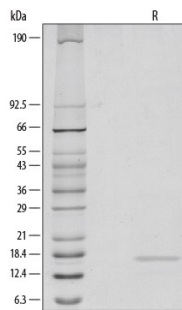
DATA

Bioactivity



Recombinant Human TNF-alpha Protein Bioactivity
Recombinant Human TNF- α (Catalog # 210-TA/CF) induces cytotoxicity in the L-929 mouse fibroblast cell line in the presence of the metabolic inhibitor actinomycin D. The ED₅₀ for this effect is 25-100 μ g/mL.

SDS-PAGE



Recombinant Human TNF-alpha Protein SDS-PAGE 1 μ g/lane of Recombinant Human TNF- α was resolved by SDS-PAGE with silver staining, under reducing (R) conditions, showing a band at 17 kDa.

BACKGROUND

Tumor necrosis factor alpha (TNF-α), also known as cachectin and TNFSF2, is the prototypic ligand of the TNF superfamily. It is a pleiotropic molecule that plays a central role in inflammation, immune system development, apoptosis, and lipid metabolism (1, 2). Human TNF-α consists of a 35 amino acid (aa) cytoplasmic domain, a 21 aa transmembrane segment, and a 177 aa extracellular domain (ECD) (3). Within the ECD, human TNF-α shares 97% aa sequence identity with rhesus and 71%-92% with bovine, canine, cotton rat, equine, feline, mouse, porcine, and rat TNF-α. TNF-α is produced by a wide variety of immune, epithelial, endothelial, and tumor cells (1, 2). TNF-α is assembled intracellularly to form a noncovalently linked homotrimer which is expressed on the cell surface (4). Cell surface TNF-α can induce the lysis of neighboring tumor cells and virus infected cells, and it can generate its own downstream cell signaling following ligation by soluble TNFR I (2, 5). Shedding of membrane bound TNF-α by TACE/ADAM17 releases the bioactive cytokine, a 55 kDa soluble trimer of the TNF-α extracellular domain (6-8). TNF-α binds the ubiquitous 55-60 kDa TNF RI (9, 10) and the hematopoietic cell-restricted 80 kDa TNF RII (11, 12), both of which are also expressed as homotrimers (1, 2, 13). Both type I and type II receptors bind TNF-α with comparable affinity (14), although only TNF RI contains a cytoplasmic death domain which triggers the activation of apoptosis. Soluble forms of both types of receptors are released and can neutralize the biological activity of TNF-α (15).

References:

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