Recombinant Human TNF-α

Catalog Number: 210-TA/CF

DESCRIPTION

Source	*E. coli*-derived Val77-Leu233, with and without an N-terminal Met Accession # P01375
N-terminal Sequence Analysis	Met & Val77
Predicted Molecular Mass	17.5 kDa

SPECIFICATIONS

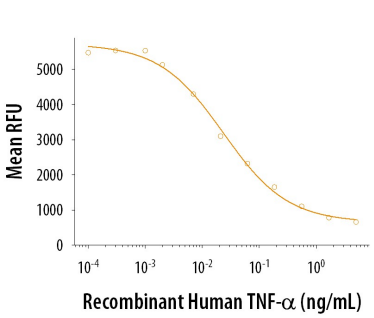
SDS-PAGE	17 kDa, reducing conditions
Activity	Measured in a cytotoxicity assay using L-929 mouse fibroblast cells in the presence of the metabolic inhibitor actinomycin D. Matthews, N. and M.L. Neale (1987) in Lymphokines and Interferons, A Practical Approach. Clemens, M.J. *et al.* (eds): IRL Press. 221. The ED_{50} for this effect is 25-100 pg/mL. The specific activity of Recombinant Human TNF-α is approximately 7.6 x 10^4 IU/µg, which is calibrated against human TNF-α WHO International Standard (NIBSC code: 88/786).
Endotoxin Level	<0.10 EU per 1 µg 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 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

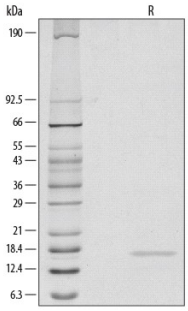
DATA

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 ED_{50} for this effect is 25-100 pg/mL.

SDS-PAGE



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

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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 consisits 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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