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Product Data Sheet

Recombinant Rat TNF-α (carrier-free)

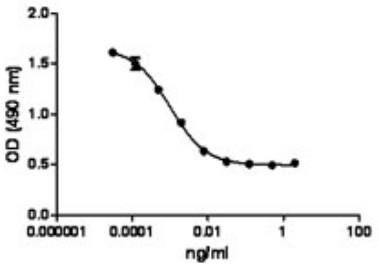
Catalog # / Size: 580102 / 10 µg
580104 / 25 µg
580106 / 100 µg
580108 / 500 µg

Source: Rat TNF-α, amino acids Leu80-Leu235 (Accession # NM_012675) was expressed in *E. coli.*

Molecular Mass: The 157 amino acid N-terminal methionylated recombinant protein has a predicted molecular mass of approximately 17,361 Da. The DTT-reduced protein migrates at approximately 17kDa and the non-reduced protein migrates at approximately 16kDa by SDS-PAGE.

Purity: Purity is >98%, as determined by Coomassie stained SDS-PAGE.

Endotoxin Level: Endotoxin level is <0.1 EU/µg (<0.01 ng/µg) protein as determined by the LAL method.



Rat TNF-α cytotoxicity on L929 cells

Activity: The ED_{50} is 5- 15 pg/ml, corresponding to a specific activity of 2 - 0.66 x 10^8units/mg, as determined by a dose dependent cytotoxicity assay using L929 cells treated with actinomycin D.

Preparation: 10-100µg sizes are bottled at 200µg/mL. 500µg sizes and larger are bottled at the concentration indicated on the vial.

Formulation: 0.22µm filtered protein solution is in 10mM NaH_2PO_4, 150mM NaCl, pH 7.2.

Storage: Unopened vial can be stored at 4°C for three months, at -20°C for six months, or at -70°C for one year. For maximum results, quick spin vial prior to opening. Stock solutions should be prepared at no less than 10µg/mL in buffer containing carrier protein such as 1% BSA or HSA or 10% FBS. For long term-storage, aliquot into polypropylene vials and store in a manual defrost freezer. **Avoid repeated freeze/thaw cycles.**

Applications:

Applications: Bioassay

Description: TNF-α was originally described as an endotoxin-induced, macrophage-derived factor that promotes hemorrhagic necrosis of solid tumors and the cachexia of chronic infections. TNF-α has also been implicated in a range of inflammatory, infectious, and malignant disorders. At the cellular level, TNF-α modulates a broad spectrum of responses including inflammation, immunoregulation, proliferation, apoptosis, and antiviral activity. In bone, the cytokine inhibits extracellular matrix deposition, stimulates matrix metalloprotease synthesis, and enhances production of osteoclastogenic cytokines such as M-CSF and RANKL. Chronic exposure to TNFa in vivo increases osteoclastogenesis through two distinct mechanisms. TNFa first affects osteoclastogenesis at the osteoclast precursor stage in the bone marrow by priming these cells to differentiate into cFms+/CD11b+/RANK+/- osteoclast progenitors via a RANKL/RANK independent mechanism. These osteoclast precursors then enter the blood and peripheral tissues where they differentiate into mature osteoclasts in the presence of RANKL, and this process is accelerated by TNF. The role of TNF at this later stage of osteoclast differentiation is RANKL/RANK dependent. Importantly, TNF-α promotes bone resorption both in vitro and in vivo by enhancing the proliferation and differentiation of osteoclast precursors.

Antigen References:
1. Boyce BF, *et al. J Med* 54:127-131 2005.
2. Lam J, *et al. J Clin Invest* 106:1481-1488 2000.
3. Udagawa N, *et al. Arthritis Res*4:281-289 2002.
4. Kwon J, *et al. Gene* 132:227-236 1993.

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