9051 Mira Mesa Blvd, #910094
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TNF a Mouse

Tumor Necrosis Factor-Alpha Mouse Recombinant
CYK0552

Product Overview

Name	TNF a Mouse
Description	
Tumor Necrosis Factor-Alpha Mouse Recombinant	
Accession (Primary)	P06804
Synonyms	
TNF-alpha, Tumor necrosis factor ligand superfamily member 2, TNF-a, Cachectin, DIF, TNFA, TNFSF2.	
Introduction	
Tumor necrosis factor is a cytokine involved in systemic inflammation and is a member of a group of cytokines that all stimulate the acute phase reaction. TNF is mainly secreted by macrophages. TNF causes apoptotic cell death, cellular proliferation, differentiation, inflammation, tumorigenesis and viral replication, TNF is also involved in lipid metabolism, and coagulation. TNF's primary role is in the regulation of immune cells. Dysregulation and, in particular, overproduction of TNF have been implicated in a variety of human diseases- autoimmune diseases, insulin resistance, and cancer.	
Source	
Escherichia Coli.	
Physical Appearance	
Sterile Filtered White lyophilized (freeze-dried) powder.	
Formulation	
Lyophilized from a 0.2 μm filtered concentrated solution in PBS, pH 7.2.	
Stability	
Lyophilized Tumor Necrosis Factor-a although stable at room temperature for 3 weeks, should be stored desiccated below -18°C. Upon reconstitution TNF-a should be stored at 4°C between 2-7 days and for future use below -18°C. For long term storage it is recommended to add a carrier protein (0.1% HSA or BSA). Please prevent freeze-thaw cycles.	
Purity	
Greater than 97.0% as determined by: (a) Analysis by RP-HPLC. (c) Analysis by SDS-PAGE.	
Amino acid sequence	
MLRSSSQNSS DKPVAHVVAN HQVEEQLEWL SQRANALLAN GMDLKDNQLV VPADGLYLVY SQVLFKGQGC	

PDYVLLTHTV SRFAISYQEK VNLLSAVKSP CPKDTPEGAE LKPWYEPIYL GGVFQLEKGD QLSAEVNLPK YLDFAESGQV YFGVIAL

Biological Activity

The ED50 as determined by the cytolysis of murine L929 cells in the presence of Actinomycin D is < 0.1ng/ml, corresponding to a Specific Activity of 10,000,000 Units/mg.

Solubility

It is recommended to reconstitute the lyophilized Tumor Necrosis Factor-alpha in sterile 18M-cm H2O not less than 100 µg/ml, which can then be further diluted to other aqueous solutions.

Background

Tumor Necrosis Factor-alpha (TNF-α) is a pro-inflammatory cytokine that plays a critical role in the regulation of immune responses, inflammation, and cell survival. It is primarily produced by activated macrophages, but can also be secreted by other immune cells, such as T cells, natural killer cells, and mast cells. TNF-α is involved in a wide range of physiological and pathological processes, including the defense against infections, the development of autoimmune diseases, and the progression of cancer. TNF-α exerts its effects by binding to two distinct cell surface receptors, TNF receptor 1 (TNFR1) and TNF receptor 2 (TNFR2). Upon binding to its receptors, TNF-α activates multiple signaling pathways, including the nuclear factor-kappa B (NF-κB) pathway, the mitogen-activated protein kinase (MAPK) pathway, and the apoptotic pathway. These signaling pathways regulate various cellular processes, such as inflammation, cell proliferation, differentiation, and apoptosis. In the context of infections, TNF-α plays a crucial role in the body's defense against pathogens. It promotes the recruitment and activation of immune cells, enhances the production of other pro-inflammatory cytokines, and stimulates the expression of adhesion molecules on endothelial cells, facilitating the migration of immune cells to the site of infection. TNF-α also helps to induce fever, which is an important component of the body's immune response to infections. However, excessive or prolonged production of TNF-α can contribute to the development of chronic inflammatory diseases, such as rheumatoid arthritis, inflammatory bowel disease, and psoriasis. In these conditions, elevated levels of TNF-α promote the infiltration of immune cells into the affected tissues, leading to tissue damage and the perpetuation of inflammation. The central role of TNF-α in the pathogenesis of these diseases has led to the development of anti-TNF-α therapies, which have revolutionized the treatment of chronic inflammatory diseases. These therapies include monoclonal antibodies, such as infliximab and adalimumab, and soluble TNF receptor fusion proteins, such as etanercept. Anti-TNF-α therapies have been shown to be effective in reducing inflammation, improving symptoms, and slowing disease progression in patients with chronic inflammatory diseases. In the context of cancer, TNF-α has complex and context-dependent effects on tumor development and progression. On one hand, TNF-α can promote anti-tumor immunity by activating immune cells and stimulating the production of other pro-inflammatory cytokines. On the other hand, chronic inflammation driven by TNF-α can promote tumor growth, angiogenesis, and metastasis. Therefore, the role of TNF-α in cancer is still an area of active research, and the development of TNF-α-targeted therapies for cancer remains a challenge. In conclusion, TNF-α is a pro-inflammatory cytokine that plays a critical role in the regulation of immune responses, inflammation, and cell survival. Its involvement in various physiological and pathological processes has

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made it an important target for the development of therapies for chronic inflammatory diseases and cancer. Anti-TNF-α therapies have revolutionized the treatment of chronic inflammatory diseases, but the complex role of TNF-α in cancer remains an area of ongoing research. Understanding the precise mechanisms by which TNF-α contributes to disease pathogenesis will be crucial for the development of more effective and targeted therapies.

References

Title :PUMA is directly activated by NF- ? B and contributes to TNF- ? -induced apoptosis. Publication :Received 13 October 2008; Revised 3 April 2009; Accepted 3 April 2009; Published online 15 May 2009. Link : TNF a prospec publication

Protein content

Protein quantitation was carried out by two independent methods1. UV spectroscopy at 280 nm using the absorbency value of 1.24 as the extinction coefficient for a 0.1% (1mg/ml) solution. This value is calculated by the PC GENE computer analysis program of protein sequences (IntelliGenetics). 2. Analysis by RP-HPLC, using a calibrated solution of TNF-a as a Reference Standard.

Precautions

TNF a Mouse is for research use only and not for use in diagnostic or therapeutic procedures.

Target Information: (P06804)

Background

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important component of the body's immune response to infections. However, excessive or prolonged production of TNF-α can contribute to the development of chronic inflammatory diseases, such as rheumatoid arthritis, inflammatory bowel disease, and psoriasis. In these conditions, elevated levels of TNF-α promote the infiltration of immune cells into the affected tissues, leading to tissue damage and the perpetuation of inflammation. The central role of TNF-α in the pathogenesis of these diseases has led to the development of anti-TNF-α therapies, which have revolutionized the treatment of chronic inflammatory diseases. These therapies include monoclonal antibodies, such as infliximab and adalimumab, and soluble TNF receptor fusion proteins, such as etanercept. Anti-TNF-α therapies have been shown to be effective in reducing inflammation, improving symptoms, and slowing disease progression in patients with chronic inflammatory diseases. In the context of cancer, TNF-α has complex and context-dependent effects on tumor development and progression. On one hand, TNF-α can promote anti-tumor immunity by activating immune cells and stimulating the production of other pro-inflammatory cytokines. On the other hand, chronic inflammation driven by TNF-α can promote tumor growth, angiogenesis, and metastasis. Therefore, the role of TNF-α in cancer is still an area of active research, and the development of TNF-α-targeted therapies for cancer remains a challenge. In conclusion, TNF-α is a pro-inflammatory cytokine that plays a critical role in the regulation of immune responses, inflammation, and cell survival. Its involvement in various physiological and pathological processes has made it an important target for the development of therapies for chronic inflammatory diseases and cancer. Anti-TNF-α therapies have revolutionized the treatment of chronic inflammatory diseases, but the complex role of TNF-α in cancer remains an area of ongoing research. Understanding the precise mechanisms by which TNF-α contributes to disease pathogenesis will be crucial for the development of more effective and targeted therapies.

References for protein:

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