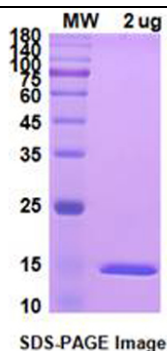


Product Details

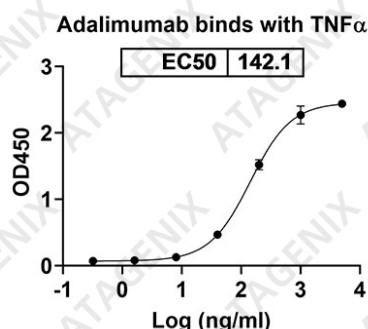
Summary

English name	Recombinant Human TNF- α protein ,N- His Tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	<1.0 EU per μ g of the protein as determined by the LAL method.
Construction	A DNA sequence encoding the human TNF(Val77~Leu233) was fused with the N-terminal His Tag
Accession #	P01375
Host	Mammalian cells
Species	Homo sapiens (Human)
Predicted Molecular Mass	17.32kDa
Formulation	Supplied as solution form in 50 mM Tris-HCl, pH 7.5, 150 mM NaCl or lyophilized from 50 mM Tris-HCl, pH 7.5, 150 mM NaCl.
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution. A copy of datasheet will be provided with the products, please refer to it for details.

SDS-PAGE image



Bioactivity



Background

Background

Tumor necrosis factor alpha (TNF α) is a cytokine produced primarily by monocytes and macrophages. It is found in synovial cells and macrophages in the tissues. The primary role of TNF α is in the regulation of immune cells. TNF α is able to induce apoptotic cell death, to induce inflammation, and to inhibit tumorigenesis and viral replication. Dysregulation of TNF α production has been implicated in a variety of human diseases, including major depression, Alzheimer's disease and cancer. Recombinant TNF α is used as an immunostimulant under the INN tasonermin. TNF α can be produced ectopically in the setting of malignancy and parallels parathyroid hormone both in causing secondary hypercalcemia and in the cancers with which excessive production is associated.

Alternative Names

DIF, TNF-alpha, TNFA, TNFSF2, cachexin, cachectin, TNF α

References

Reddy, Lakshmi, Maruthi Prasad, Varadacharyulu, Kodihela (2020)

Epigallocatechin gallate suppresses inflammation in human coronary artery endothelial cells by inhibiting NF- κ B Life sciences () 118136

Frontier progress

The endothelium is a critical regulator of vascular homeostasis, controlling vascular tone and permeability as well as interactions of leukocytes and platelets with blood vessel walls. Consequently, endothelial dysfunction featuring inflammation and reduced vasodilation are considered central to cardiovascular disease (CVD) pathogenesis and have become a therapeutic area of focus. Type II endothelial cell (EC) activation by stress-related stimuli such as tumor necrosis factor- α (TNF- α) initiates the nuclear factor- κ B (NF- κ B) signaling pathway, a master regulator of inflammatory responses. Because dysregulated NF- κ B signaling has been tightly linked to several CVDs, EC-specific inhibition of NF- κ B represents an attractive pharmacological strategy. As accumulating evidence highlights the clinical benefits of tea catechin for multiple diseases including CVDs, we sought to determine whether the tea catechin epigallocatechin gallate (EGCG) that displays antioxidative, anti-inflammatory,

Recombinant Human TNF- α protein ,N- His Tag

hypolipidemic, anti-thrombogenic, and anti-hypertensive properties offers protection against CVDs by suppressing the canonical NF- κ B pathway. Our findings indicate that EGCG downregulates multiple components of the TNF- α -induced NF- κ B signaling pathway and thereby reduces the consequent increase in inflammatory gene transcription and protein expression. Furthermore, EGCG blocked type II EC activation, evidenced by diminished EC leakage and monocyte adhesion in EGCG-treated cells. In summary, our study advances knowledge of EGCG's anti-inflammatory effects on the NF- κ B pathway and hence its benefits on endothelial health, supporting its therapeutic potential for CVDs.