

## TNF-alpha/TNFSF2 protein, Rabbit

<b>Cat. No.:</b>	HY-P70800
<b>Synonyms:</b>	Tumor Necrosis Factor; Cachectin; TNF-Alpha; Tumor Necrosis Factor Ligand Superfamily Member 2; TNF-a; TNF; TNFA; TNFSF2
<b>Species:</b>	Rabbit
<b>Source:</b>	E. coli
<b>Accession:</b>	P04924 (V78-L235)
<b>Gene ID:</b>	100009088
<b>Molecular Weight:</b>	Approximately 19.0 kDa

### PROPERTIES

<b>AA Sequence</b>	<p>V T L R S A S R A L      S D K P L A H V V A      N P Q V E G Q L Q W      L S Q R A N A L L A</p> <p>N G M K L T D N Q L      V V P A D G L Y L I      Y S Q V L F S G Q G      C R S Y V L L T H T</p> <p>V S R F A V S Y P N      K V N L L S A I K S      P C H R E T P E E A      E P M A W Y E P I Y</p> <p>L G G V F Q L E K G      D R L S T E V N Q P      E Y L D L A E S G Q      V Y F G I I A L</p>
<b>Appearance</b>	Lyophilized powder.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution of 20 mM PB, 250 mM NaCl, pH 7.4.
<b>Endotoxin Level</b>	<1 EU/µg, determined by LAL method.
<b>Reconstitution</b>	It is not recommended to reconstitute to a concentration less than 100 µg/mL in ddH <sub>2</sub> O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
<b>Storage &amp; Stability</b>	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
<b>Shipping</b>	Room temperature in continental US; may vary elsewhere.

### DESCRIPTION

<b>Background</b>	<p>TNF alpha is produced by various types of cells including macrophages, monocytes, neutrophils, T cells, and NK-cells<sup>[2]</sup>. The amino acid sequence of human TNF alpha protein has low homology between mouse, rat, bovine, cynomolgus TNF alpha protein. While, human TNF alpha shares 94.85% aa sequence identity with cynomolgus TNF alpha protein, mouse TNF alpha shares 94.47% aa sequence identity with rat TNF alpha protein.</p> <p>TNF alpha exists in two forms; a type II transmembrane protein (tmTNF-α) and a mature soluble protein (sTNF-α). TNF-α binds to its receptors, mainly TNFR1 and TNFR2, and then transmits molecular signals for biological functions such as inflammation and cell death. Both sTNF-α and tmTNF-α activate TNFR1, and process a death domain (DD) that interacts with the TNFR1-associated death domain (TRADD) adaptor protein. The TNFR2 signaling pathway is mainly activated by</p>
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tmTNF- $\alpha$ . TNFR1 signaling tends to be pro-inflammatory and apoptotic. TNFR2 results in NF- $\kappa$ B and MAPKs and AKT activation, TNFR2 activation is associated with homeostatic bioactivities such as tissue regeneration, cell proliferation, and cell survival, as well as host defense and inflammation<sup>[1]</sup>.

TNF- $\alpha$  is critical for normal immune response, abnormal secretion TNF  $\alpha$  activates synovial fibroblasts, keratinocytes, osteoclasts, induces rheumatoid arthritis, inflammatory bowel disease, psoriatic arthritis (PsA), and noninfectious uveitis (NIU)<sup>[3]</sup>. TNF  $\alpha$  positively regulates endogenous TNF- $\alpha$  expression levels independently of Pgp efflux activity, induces IHF cells proliferation<sup>[4]</sup>. TNF  $\alpha$  in tissues may promote cancer growth, invasion, and metastasis. Besides, TNF  $\alpha$  stimulates NF- $\kappa$ B pathway via TNFR2 and anti-TNF- $\alpha$  MAb significantly suppresses the tumor development in colitis-associated cancer (CAC) mouse<sup>[5]</sup>. TNF  $\alpha$  as a proneurogenic factor activates the SAPK/JNK pathway and can facilitate neuronal replacement and brain repair in response to brain injury<sup>[6]</sup>.

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## REFERENCES

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- [3]. Jang DI, et al. The Role of Tumor Necrosis Factor Alpha (TNF- $\alpha$ ) in Autoimmune Disease and Current TNF- $\alpha$  Inhibitors in Therapeutics. *Int J Mol Sci*. 2021 Mar 8;22(5):2719.
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- [6]. Bernardino L, et al. Tumor necrosis factor- $\alpha$  modulates survival, proliferation, and neuronal differentiation in neonatal subventricular zone cell cultures. *Stem Cells*. 2008 Sep;26(9):2361-71.
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