

TNF Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP13532b**Specification**

TNF Antibody (C-term) Blocking peptide - Product InformationPrimary Accession [P01375](#)**TNF Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 7124**Other Names**

Tumor necrosis factor, Cachectin, TNF-alpha, Tumor necrosis factor ligand superfamily member 2, TNF-a, Tumor necrosis factor, membrane form, N-terminal fragment, NTF, Intracellular domain 1, ICD1, Intracellular domain 2, ICD2, C-domain 1, C-domain 2, Tumor necrosis factor, soluble form, TNF, TNFA, TNFSF2

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP13532b was selected from the C-term region of TNF. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

TNF Antibody (C-term) Blocking peptide - Protein Information**Name** TNF**Synonyms** TNFA, TNFSF2**Function**

Cytokine that binds to TNFRSF1A/TNFR1 and TNFRSF1B/TNFR. It is mainly secreted by macrophages and can induce cell death of certain tumor cell lines. It is potent pyrogen causing fever by direct action or by stimulation of interleukin-1 secretion and is implicated in the induction of cachexia. Under certain conditions it can stimulate cell proliferation and induce cell differentiation. Impairs regulatory T- cells (Treg) function in individuals with rheumatoid arthritis via FOXP3 dephosphorylation. Up-regulates the expression of protein phosphatase 1 (PP1), which dephosphorylates the key 'Ser-418' residue of FOXP3, thereby inactivating FOXP3 and rendering Treg cells functionally defective (PubMed:<a href="http://www.uniprot.org/citations/23396208"

target="_blank">23396208). Key mediator of cell death in the anticancer action of BCG-stimulated neutrophils in combination with DIABLO/SMAC mimetic in the RT4v6 bladder cancer cell line (PubMed:22517918, PubMed:16829952, PubMed:23396208). Induces insulin resistance in adipocytes via inhibition of insulin-induced IRS1 tyrosine phosphorylation and insulin-induced glucose uptake. Induces GKAP42 protein degradation in adipocytes which is partially responsible for TNF-induced insulin resistance (By similarity). Plays a role in angiogenesis by inducing VEGF production synergistically with IL1B and IL6 (PubMed:12794819). Promotes osteoclastogenesis and therefore mediates bone resorption (By similarity).

Cellular Location

Cell membrane; Single-pass type II membrane protein [Tumor necrosis factor, soluble form]: Secreted [C-domain 2]: Secreted.

TNF Antibody (C-term) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

TNF Antibody (C-term) Blocking peptide - Images

TNF Antibody (C-term) Blocking peptide - Background

This gene encodes a multifunctional proinflammatory cytokine that belongs to the tumor necrosis factor (TNF) superfamily. This cytokine is mainly secreted by macrophages. It can bind to, and thus functions through its receptors TNFRSF1A/TNFR1 and TNFRSF1B/TNFR2. This cytokine is involved in the regulation of a wide spectrum of biological processes including cell proliferation, differentiation, apoptosis, lipid metabolism, and coagulation. This cytokine has been implicated in a variety of diseases, including autoimmune diseases, insulin resistance, and cancer. Knockout studies in mice also suggested the neuroprotective function of this cytokine.

TNF Antibody (C-term) Blocking peptide - References

Majewski, P.M., et al. J. Biol. Chem. 285(45):34828-34838(2010) Kwon, H.J., et al. J. Immunol. 185(7):3980-3989(2010) Muller, T., et al. Scand. J. Immunol. 72(4):365-371(2010) Qin, H., et al. J. Int. Med. Res. 38(3):760-768(2010) Acharyya, S., et al. PLoS ONE 5 (8), E12479 (2010) :