BOSTER BIOLOGICAL TECHNOLOGY
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Anti-TNF alpha Antibody Picoband®

Catalog Number: PB9010

About Tnf

TNF alpha (Tumor Necrosis Factor alpha) 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/TNFBR. 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.

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Overview

Product Name	Anti-TNF alpha Antibody Picoband®
Reactive Species	Rat
Description	Boster Bio Anti-TNF alpha Antibody Picoband® catalog # PB9010. Tested in WB applications. This antibody reacts with Rat. The brand Picoband indicates this is a premium antibody that guarantees superior quality, high affinity, and strong signals with minimal background in Western blot applications. Only our best-performing antibodies are designated as Picoband, ensuring unmatched performance.
Application	WB
Clonality	Polyclonal
Formulation	Each vial contains 4 mg Trehalose, 0.9 mg NaCl and 0.2 mg Na2HPO4.
Storage Instructions	Store at -20˚C for one year from date of receipt. After reconstitution, at 4˚C for one month. It can also be aliquotted and stored frozen at -20˚C for six months. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P16599

Technical Details

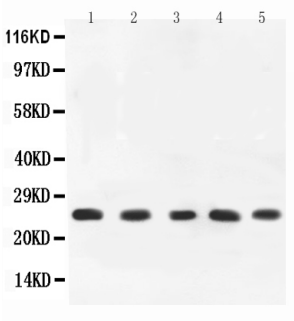
Immunogen	E.coli-derived rat TNF alpha recombinant protein (Position: D89-L235). Rat TNF alpha shares 95% amino acid (aa) sequence identity with mouse TNF alpha.

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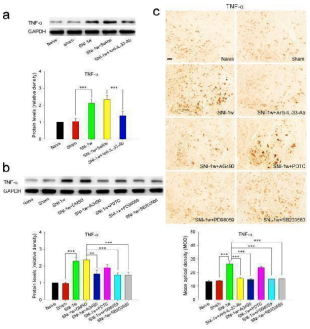
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot.
Cross Reactivity	No cross-reactivity with other proteins
Isotype	Rabbit IgG
Form	Lyophilized
Concentration	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml.
Purification	Immunogen affinity purified.
Suggested Dilutions	Western blot, 0.1-0.5ug/ml, Rat

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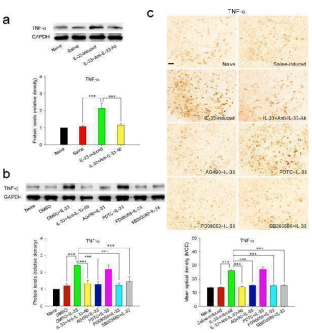
Anti-TNF alpha Antibody Picoband® (PB9010) Images



Western blot analysis of TNF alpha using anti-TNF alpha antibody (PB9010). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 50ug of sample under reducing conditions. Lane 1: PC-12 Whole Cell Lysate, Lane 2: Rat Spleen Tissue Lysate, Lane 3: Rat Brain Tissue Lysate, Lane 4: Rat Kidney Tissue Lysate, Lane 5: Rat Liver Tissue Lysate. After Electrophoresis, proteins were transferred to a Nitrocellulose membrane at 150mA for 50-90 minutes. Blocked the membrane with 5% Non-fat Milk/ TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-TNF alpha antigen affinity purified polyclonal antibody (Catalog # PB9010) at 0.5 ug/mL overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:10000 for 1.5 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for TNF alpha at approximately 26KD. The expected band size for TNF alpha is at 26KD.



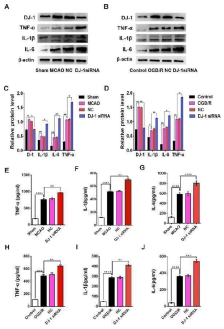
Red nucleus IL-33 facilitates the early development of mononeuropathic pain by inducing TNF-alpha through activating ERK, p38 MAPK and JAK2/STAT3 signaling pathways. A Western blotting showed that red nucleus TNF-alpha was increased at 1 week post-SNI, intrarubral injection of anti-IL-33 antibody at 1 week post-SNI suppressed the overexpression of TNF-alpha (n = 6 per group, F = 14.302, P < 0.001). B Western blotting displayed that intrarubral administration of PD98059, SB203580, or AG490 at 1 week post-SNI inhibited the production of TNF-alpha (n = 6 per group, F = 13.157, P < 0.001). C Immunohistochemistry showed that red nucleus TNF-alpha was upregulated at 1 week post-SNI, intrarubral injection of anti-IL-33 antibody, PD98059, SB203580, or AG490 at 1 week post-SNI suppressed the production of TNF-alpha (n = 4 per group, F = 33.029, P < 0.001). ** P < 0.01 and *** P < 0.001. Scale bars = 50 um Index in PubMed under a CC BY license. PMID: 34225736



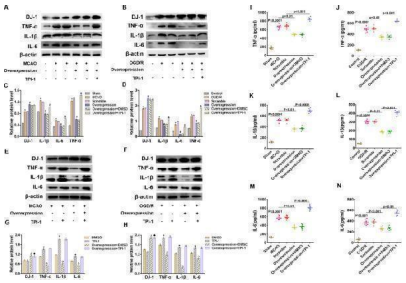
Red nucleus IL-33 evokes mechanical hypersensitivity by inducing TNF-alpha through activating ERK, p38 MAPK, and JAK2/STAT3 signaling pathways. A Western blotting indicated that intrarubral administration of IL-33 stimulated the secretion of TNF-alpha in naive rats (n = 6 per group, F = 15.143, P < 0.001). B Western blotting showed that intrarubral pre-injection of PD98059, SB203580, or AG490, 30 min before IL-33 administration, restrained IL-33-induced overexpression of TNF-alpha in naive rats (n = 6 per group, F = 9.812, P < 0.001). C Immunohistochemistry showed that intrarubral injection of IL-33 potentiated the secretion of TNF-alpha in naive rats, intrarubral pre-injection of PD98059,

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SB203580, or AG490, 30 min prior to IL-33 administration, inhibited IL-33-induced overexpression of TNF-alpha in naive rats (n = 4 per group, F = 44.310, P < 0.001). *** P < 0.001. Scale bars = 50 um Index in PubMed under a CC BY license. PMID: 34225736



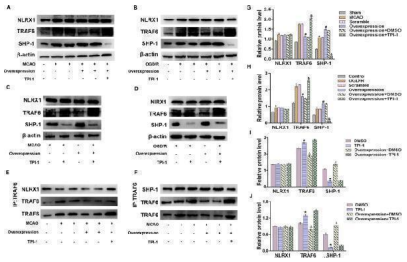
DJ-1 interference increased the expression of TNF-alpha, IL-1beta, and IL-6 after cerebral I/R injury. a and c Western blot detecting DJ-1 and the cytokines TNF-alpha, IL-1beta, and IL-6 in rats. b , d Western blot detecting DJ-1 and the cytokines TNF-alpha, IL-1beta, and IL-6 in astrocytes. e–g Quantification of TNF-alpha, IL-1beta, and IL-6 in rats by ELISA. h-j Quantification of TNF-alpha, IL-1beta, and IL-6 in astrocytes by ELISA. The data are expressed as the mean ± SEM. * p < 0.05, ** p < 0.01, *** p < 0.001, **** p < 0.0001. n = 6 per group Index in PubMed under a CC BY license. PMID: 32151250



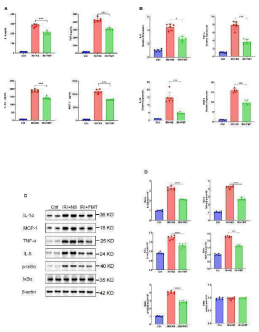
DJ-1 inhibited the expression of TNF-alpha, IL-1beta, and IL-6 after cerebral I/R injury via SHP-1. a , c After virus and TPI-1 were used to overexpress DJ-1 and inhibit SHP-1, respectively, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in rats. b , d After virus and TPI-1 were used to overexpress DJ-1 and inhibit SHP-1, respectively, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in astrocytes. e , g After treatment with an SHP-1 inhibitor, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in rats. f , h After treatment with an SHP-1 inhibitor, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in astrocytes. i , k , m Quantification of TNF-alpha, IL-1beta, and IL-6 in rats by ELISA. j , l , n Quantification of TNF-alpha, IL-1beta, and IL-6 in astrocytes by ELISA. The data are expressed as the mean ± SEM. * p < 0.05 vs. the sham group; # p < 0.05 vs. the MCAO group; & p < 0.05 vs. the overexpression group; ★ p < 0.05 vs. the DMSO group; △ p < 0.05 vs. the DMSO group; ◆ p < 0.05 vs. the TPI-1 group. n = 6 per group. The data are expressed as the mean ± SEM. * p < 0.05 vs. the control group; # p < 0.05 vs. the OGD/R group; & p < 0.05 vs. the overexpression group; ★ p < 0.05 vs. the DMSO group; △ p < 0.05 vs. the DMSO group; ◆ p < 0.05 vs. the TPI-1 group. n = 6 per group. The groups in a and b and their corresponding groups in the statistical graphs are as follows: sham (−−−), MCAO or OGD/R (+−−), scramble (+−−), overexpression (++−), overexpression + DMSO (++−), and overexpression + TPI-1 (+++). The groups in e and f and the corresponding groups in the statistical graphs are as follows: DMSO (+−−), TPI-1 (+−+), overexpression + DMSO (++−), overexpression + TPI-1 (+++) Index in PubMed under a CC BY license. PMID: 32151250

DJ-1 regulated the disassociation of NLRX1 from TRAF6 after cerebral I/R injury via SHP-1. a , g After virus and TPI-1 were used to overexpress DJ-1 and inhibit SHP-1, respectively, Western blotting was used to detect NLRX1, TRAF6, and SHP-1 in rats. b , h After virus and TPI-1 were used to

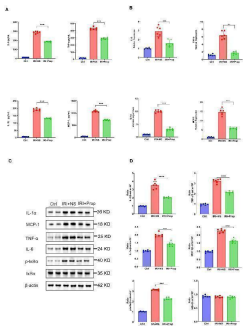
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overexpress DJ-1 and inhibit SHP-1, respectively, Western blotting was used to detect NLRX1, TRAF6, and SHP-1 in astrocytes. c , i After treatment with an SHP-1 inhibitor, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in rats. d , j After treatment with an SHP-1 inhibitor, Western blotting was used to detect the cytokines IL-1beta, IL-6, and TNF-alpha in astrocytes. e Immunoprecipitation and immunoblot analyses of NLRX1-TRAF6 in rats. f Immunoprecipitation and immunoblot analyses of SHP-1-TRAF6 in rats. The data are expressed as the mean ± SEM. * $p < 0.05$ vs. the sham group; # $p < 0.05$ vs. the MCAO group; & $p < 0.05$ vs. the overexpression group; ★ $p < 0.05$ vs. the DMSO group; △ $p < 0.05$ vs. the DMSO group. n = 6 per group. The data are expressed as the mean ± SEM. * $p < 0.05$ vs. the control group; # $p < 0.05$ vs. the OGD/R group; & $p < 0.05$ vs. the overexpression group; ★ $p < 0.05$ vs. the DMSO group; △ $p < 0.05$ vs. the DMSO group. n = 6 per group Index in PubMed under a CC BY license. PMID: 32151250



Fecal microbiota transplantation (FMT) reduced systemic or local inflammatory response. (A) Protein levels of the pro-inflammatory cytokines and chemokines in the blood. (B) mRNA levels of the pro-inflammatory cytokines and chemokines in kidney tissue using real-time PCR. (C) Western blot analysis of the pro-inflammatory cytokines in kidney tissue. (D) Protein band intensities quantified using optical densitometry and normalized to beta-actin levels within the respective experimental groups. n = 6. Representative images from six independent experiments. The p -values above the data denote statistical comparisons on the groups—the control (Ctrl), ischemia–reperfusion injury plus normal saline (IRI+NS), and IRI+FMT groups—which were calculated using ANOVA with Tukey's post-hoc test. * $p < 0.05$; *** $p < 0.001$.Index in PubMed under a CC BY license. PMID: 41078364



Propionic acid reduced the systemic or local inflammatory response. (A) Protein levels of the pro-inflammatory cytokines and chemokines in the blood. (B) mRNA levels of the pro-inflammatory cytokines and chemokines in kidney tissue using real-time PCR. (C) Western blot analysis of the pro-inflammatory cytokines in kidney tissue. (D) Protein band intensities quantified using optical densitometry and normalized to beta-actin levels within the respective experimental groups. n = 6. Representative images from six independent experiments. The p -values above the data denote statistical comparisons of the groups—the control (Ctrl), ischemia–reperfusion injury plus normal saline (IRI+NS), and IRI plus propionic acid (IRI+Prop) groups—which were calculated using ANOVA with Tukey's post-hoc test. ** $p < 0.01$; *** $p < 0.001$.Index in PubMed under a CC BY license. PMID: 41078364

80 Publications Citing This Product

1. PubMed ID: 34225736, Li HN,Yang QQ,Wang WT,Tian X,Feng F,Zhang ST,Xia YT,Wang JX,Zou YW,Wang JY,Zeng XY.Red nucleus IL-33

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facilitates the early development of mononeuropathic pain in male rats by inducing TNF-alpha through activating ERK, p38 MAPK, and JAK2/STAT3.J Neuroinflammation.2021 Jul 5;18(1):150.doi:10.1186/s12974-021-02198-9.PMID:34225736;PMCID:PMC8258957.

2. PubMed ID: 33823825, Wang B,Gao C,Zhang P,Sun W,Zhang J,Gao J.The increased motion of lumbar induces ligamentum flavum hypertrophy in a rat model. BMC Musculoskelet Disord.2021 Apr 6;22(1):334.doi:10.1186/s12891-021-04203-x.PMID:33823825.

3. PubMed ID: -, Yang Ping,Yingpeng Li,Shaowa Lü,Yali Sun,Wanmeng Zhang,Jialin Wu,Ting Liu,Yongji Li,A study of nanometre aggregates formation mechanism and antipyretic effect in Bai-Hu-Tang, an ancient Chinese herbal decoction,Biomedicine & Pharmacotherapy,Volume 124,202

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