BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com

Anti-MMP3 Rabbit Monoclonal Antibody

Catalog Number: M00775

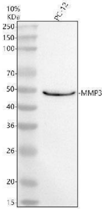
Overview

Product Name	Anti-MMP3 Rabbit Monoclonal Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-MMP3 Rabbit Monoclonal Antibody catalog # M00775. Tested in WB, IHC, ICC/IF applications. This antibody reacts with Human, Mouse, Rat.
Application	IF, IHC, ICC, WB
Clonality	Monoclonal BBO-13
Formulation	Rabbit IgG in stabilizing components, phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P08254

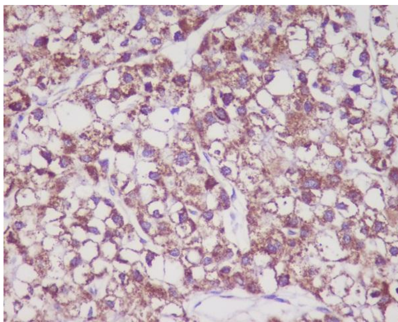
Technical Details

Immunogen	A synthesized peptide derived from human MMP3
Isotype	Rabbit IgG
Form	Liquid
Concentration	0.5mg/ml
Purification	Affinity-chromatography
Suggested Dilutions	WB 1:500-2000 IHC 1:50-200 ICC/IF 1:50-200

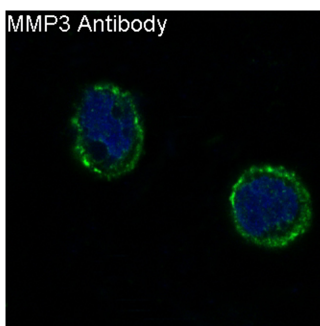
Anti-MMP3 Rabbit Monoclonal Antibody (M00775) Images



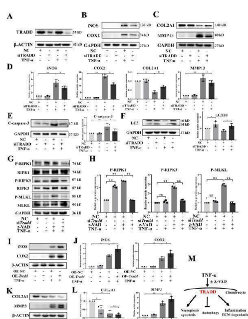
Western blot analysis of MMP3 using anti-MMP3 antibody (M00775). Electrophoresis was performed on a 10% SDS-PAGE gel at 80V (Stacking gel) / 120V (Resolving gel) for 2 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: rat PC-12 whole cell lysates.After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA 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-MMP3 antigen affinity purified monoclonal antibody (M00775) at 1:500 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 1:5000 for 1.5 hour at RT. The signal is developed using an ECL Plus Western Blotting Substrate (Catalog # AR1196-200) with Tanon 5200 system. A specific band was detected for MMP3 at approximately 50 kDa. The expected band size for MMP3 is at 50 kDa.



Immunohistochemical analysis of paraffin-embedded human liver cancer, using MMP3 Antibody.

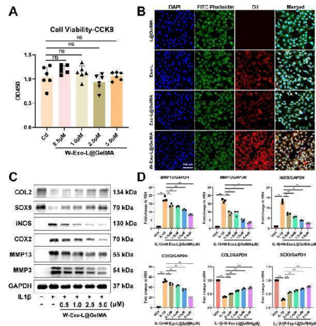


Immunofluorescent analysis of HeLa cells, using MMP3 Antibody.

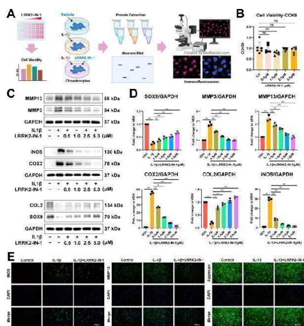


The role of TRADD in TNF-alpha-mediated cellular events. The cells were transfected with siRNA and lipofectamine 3000 for 48 h. After 48 h transfection, the medium was replaced by refresh medium and chondrocytes were treated with TNF-alpha (5 ng/ml) for 24 h, the protein expression of TRADD, iNOS, COX2, COL2A1, MMP13, cleaved-caspase-3, and LC3 was examined (A – C , E , F). After 48 h transfection with siRNA and lipofectamine 3000, the medium was replaced by refresh medium and chondrocytes were pre-treated with z-VAD for 2 h and then exposed to TNF-alpha for 24 h, the phosphorylation of RIPK1, RIPK3, and MLKL as

well as protein expression of these three markers were detected using western blot (G). I , K The protein expression of iNOS, COX2, COL2A1, and MMP3 was examined in chondrocytes overexpressing TRADD after TNF-alpha intervention. D , H , J , L Semi-quantitative analysis of protein bands by image J and data in figures were expressed as means ± SD. * p

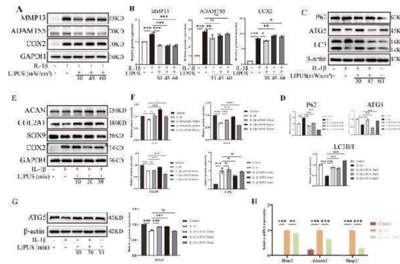


W-Exo-L@GelMA exhibits a strong chondrocyte-targeting effect and a pronounced action on promoting anabolism and suppressing catabolism and inflammation without causing the inhibition of chondrocyte viability. A Cell viability assessed by CCK8 assay. No obvious cytotoxicity on chondrocytes was observed when treated with W-Exo-L@GelMA loaded with 0.5, 1.0, 2.5, and 5.0 μM LRRK2-IN-1 for 48 h. Data represent mean ± SD; N = 6/group; one-way ANOVA; ns, not significant. B Immunofluorescence of Dil-labeled exosomes. The uptake of exosomes was observed in the chondrocytes when treated with Exo-L, Exo-L@GelMA or W-Exo-L@GelMA for 48 h. Dil was used for labeling exosomes (red), DAPI to label nuclei (blue), and Phalloidin to label the cytoskeleton (green). Scar bar: 200 um. C Western blot analyses of the protein levels of anabolic, catabolic, and inflammatory factors in the IL-1beta-induced chondrocytes treated with W-Exo-L@GelMA loaded with 0.5, 1.0, 2.5, and 5.0 μM LRRK2-IN-1 for 48 h. W-Exo-L@GelMA promoted COL2 and SOX9 and inhibited iNOS, COX2, MMP3, and MMP13 protein levels in a dose-dependent manner. D Quantitative analysis of the western blot results. Data represent mean ± SD; N = 3/group; *P<0.05; **P<0.01 by one-way ANOVA Index in PubMed under a CC BY license. PMID: 37605203

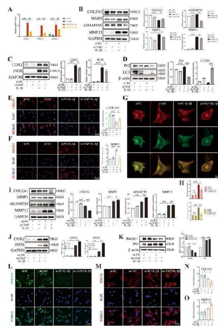


LRRK2-IN-1 suppresses the IL-1beta-induced inflammation and catabolism and induces anabolism without causing the inhibition of chondrocyte viability. A Schematic diagram of cell treatment and experimental procedures. B Cell viability assessed by CCK8 assay. No obvious inhibition of chondrocyte proliferation was observed when treated with 0.5, 1.0, 2.5, and 5.0 μM LRRK2-IN-1 for 24 h. Data represent mean ± SD; N = 6/group; one-way ANOVA; ns, not significant. C Western blot analyses of the protein levels of anabolic, catabolic, inflammatory factors in the IL-1beta-induced chondrocytes treated with 0.5, 1.0, 2.5, and 5.0 μM LRRK2-IN-1 for 24 h. LRRK2-IN-1 suppressed MMP3, MMP13, iNOS, and COX2 and induced COL2 and SOX9 in a dose-dependent manner. D Quantitative analyses of the western blot results. Data represent mean ± SD; N = 3/group; *P<0.05; **P<0.01 by one-way ANOVA. E Immunofluorescence of iNOS, MMP13, and aggrecan expression in the IL-1beta-induced chondrocytes treated with 5.0 μM LRRK2-IN-1 for 24 h. Scar bar: 400 um Index in PubMed under a CC BY license. PMID: 37605203

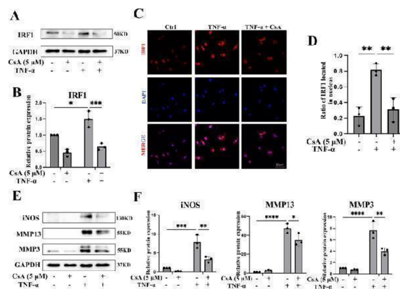
LIPUS inhibits the progression of OA. A , B Western blot and quantitative analysis of MMP13, ADAMTS5 and COX2 expression level with LIPUS application for 20 min. C , D



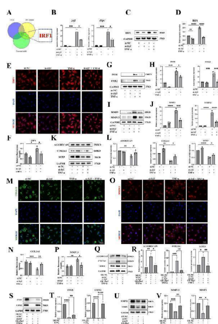
Western blot and quantitative analysis of P62, ATG5 and LC3 expression level with LIPUS application for 20 min. E , F Western blot and quantitative analysis of ACAN, COL2A1, SOX9 and COX2 expression level with 30 mW/cm 2 LIPUS application. G Western blot and quantitative analysis of ATG5 expression level with 30 mW/cm 2 LIPUS application. H qPCR of MMP3, ADAMTS5 and MMP13 relative expression level. Data are shown as mean ± SD. *P<0.05, **P<0.01, ***P<0.001 Index in PubMed under a CC BY license. PMID: 38493143



YAP exacerbated chondrocyte damage induced by IL-1beta. Chondrocytes transfected with si-NC or YAP siRNA following IL-1beta induction for 24 h. A qPCR of MMP3, MMP13 and ADAMTS5 relative expression level. B Western blot and quantitative analysis of COL2A1, MMP3, ADAMTS5 and MMP13 expression level. C , D Western blot and quantitative analysis of COX2, iNOS, P62 and LC3 expression level. E , F Immunofluorescence staining and fluorescence intensity analysis of COL2A1 and MMP13 relative expression level. G , H Chondrocytes were transfected with si-NC or si-YAP and then transfected with mRFP-GFP-LC3 adenovirus following IL-1beta treatment for 24 h. The representative images of fluorescence and quantitative analysis of red dots and yellow dots were shown (scale bar: 10 um). Subsequently, Chondrocytes transfected with oe-NC or oe-YAP following IL-1beta induction for 24 h. I Western blot and quantitative analysis of COL2A1, MMP3, ADAMTS5 and MMP13 expression level. J , K Western blot and quantitative analysis of COX2, iNOS, Beclin-1 and P62 expression level. L – O Immunofluorescence staining and fluorescence intensity analysis of COL2A1 and MMP13 relative expression level. Data are shown as mean ± SD. *P<0.05, **P<0.01, ***P<0.001 Index in PubMed under a CC BY license. PMID: 38493143

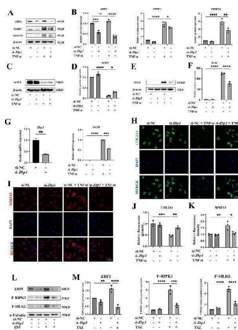


The effect of mtDNA on IRF1 and chondrocyte damage. Chondrocytes were treated with TNF-alpha alone or CsA alone or TNF-alpha combined with CsA for 12 h. (A, B) Western blots and quantitative analysis of expression level of IRF1 (n = 3). (C, D) immunofluorescence staining and fluorescence intensity analysis of IRF1 nuclear location (n = 3). (E, F) Western blots and quantitative analysis of expression level of iNOS, MMP13, and MMP3 (n = 3). Data are shown as the means ± SDs. * P

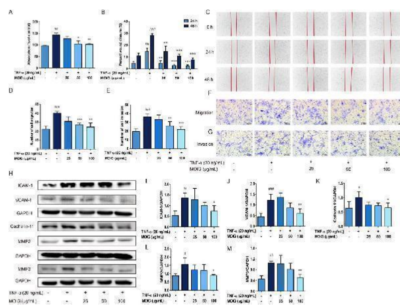


ZBP1 overexpression depends on IRF1. Chondrocytes were transfected with siNC or IRF1 siRNA for 24 h following TNF-alpha induction for 12 h. (A) Prediction of transcription factors using the UCSC Genome Browser database, SPP-ominer database, and Cistrome Data Browser. (B) qPCR results showing the relative mRNA expression levels of Irf1 and Zbp1 (n = 3). (C, D) Western blots and quantitative analysis of the relative protein expression level of IRF1 (n = 3). (E, F) Immunofluorescence staining and fluorescence intensity analysis of ZBP1 expression in chondrocytes transfected with siNC or IRF1 siRNA following TNF-alpha

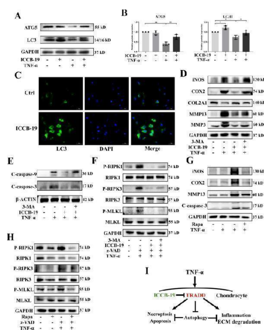
induction for 12 h (n = 3; scale bar: 50 um). (G-L) Western blots and quantitative analysis of the relative protein expression levels of iNOS, COX2, MMP3, MMP13, AGGRECAN, COL2A1, and SOX9 (n = 3). (M-P) Immunofluorescence staining and fluorescence intensity analysis of COL2A1 and MMP13 expression in chondrocytes transfected with siNC or IRF1 siRNA following TNF-alpha induction for 12 h (n = 3; scale bar: 50 um). Chondrocytes were transfected with negative control siRNA(siNC) or IRF1 siRNA (si- Irf1) or ZBP1-plasmid (OE- Zbp1) or empty plasmid (OE-NC) for 24 h following TNF-alpha induction for 24 h. (Q-V) Western blots and quantitative analysis of the relative protein expression of AGGRECAN, COL2A1, SOX9, iNOS, COX2 MMP13, and MMP3 in chondrocytes (n = 3). The data are shown as the means ± SDs. * P



ZBP1 is essential for chondrocyte damage. Chondrocytes were transfected with siNC or ZBP1 siRNA for 24 h following TNF-alpha induction for 24 h. (A, B) Western blots and quantitative analysis of ZBP1, MMP13, and MMP3 expression levels (n = 3). (C, D) Western blots and quantitative analysis of SOX9 expression levels. (E, F) Western blot and quantitative analysis of iNOS expression levels (n = 3). (G) qPCR results showing the relative expression levels of Zbp1 and Inos (n = 3). (H-K) Immunofluorescence staining and fluorescence intensity analysis of the relative expression levels of COL2A1 and MMP13 (n = 3; scale bar: 50 um). Chondrocytes were transfected with siNC or ZBP1 siRNA following TSZ (20 ng/ml TNF-alpha, 100 nM Smac mimetic, and 20 mM Z-VAD) induction for 12 h. (L, M) Western blots and quantitative analysis of ZBP1, P-RIPK3, and P-MLKL expression levels (n = 3). The data are shown as the means ± SDs. * P

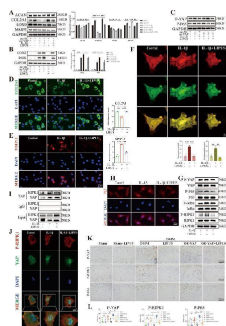


Effects of MOIG on adhesion, migration, invasion and the expression of associated proteins of TNF-alpha-stimulated FLSs cells. (A) Adhesion of FLSs cells. (B) and (C) The wound healing of FLSs at 24 and 48 h; (D) and (F) The migration of FLSs at 6 h; (E) and (G) The invasion of FLSs at 24 h; (H–M) the expression of ICAM-1, VCAM-1, cadherin 11, MMP2 and MMP3 of FLSs. The data were expressed as mean ± SD (n = 3). # $p < 0.05$, ## $p < 0.01$, ### $p < 0.001$ vs. normal ctrl group; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ vs. TNF-alpha model group.Index in PubMed under a CC BY license. PMID: 39444614



ICCB-19 protects against TNF-alpha-induced detrimental events via autophagy mechanism. After a pre-treatment of ICCB-19 for 2 h, chondrocytes were stimulated by TNF-alpha for 24 h. A the protein expression of ATG5 and LC3 was examined. B The semi-quantitative analysis of protein bands by image J. C Representative images of immunofluorescence staining of LC3 in chondrocytes, scale bar: 50 um. D – F After a pre-treatment of ICCB-19 and 3-MA for 2 h, chondrocytes were stimulated by TNF-alpha for 24 h with or without z-VAD, the protein expression of iNOS, COX2, COL2A1, MMP13, MMP3, C-caspase-9, and C-caspase-3 were

examined, and the phosphorylation of RIPK1, RIPK3, and MLKL as well as protein expression of these three markers were detected by western blot. G – H After a pre-treatment of Rapa for 2 h, chondrocytes were stimulated by TNF-alpha for 24 h with or without z-VAD, the protein expression of iNOS, COX2, MMP13, C-caspase-3 and the phosphorylation of RIPK1, RIPK3, and MLKL as well as protein expression of these three markers were detected by western blot. I The graphical scheme summarized the findings of protective role of ICCB-19 in TNF-alpha-caused detrimental events. Data are expressed as means ± SD. * p



LIPUS inhibits the progression of OA via YAP/RIPK1 axis. A Western blot and quantitative analysis of ACAN COL2A1, SOX9 and MMP3 expression level. B Western blot and quantitative analysis of COX2 and iNOS expression level. C Western blot of P-YAP and P-P65 expression level. D , E Immunofluorescence staining and fluorescence intensity analysis of COL2A1 and MMP13 relative expression level. F Chondrocytes were transfected with mRFP-GFP-LC3 adenovirus following IL-1beta treatment for 24 h. The representative images of fluorescence and quantitative analysis of red dots and yellow dots were revealed (scale bar: 10 um). G Western blot of P-YAP, YAP, P-P65, P65, P-IkappaB, IkappaB, P-RIPK1 and RIPK1 relative protein expression. H Images of IF staining for P65 relative expression and the distribution in chondrocytes with IL-1beta induction for 15 min (scale bar: 25 um). I Co-IP experiment of the binding between YAP and RIPK1 after LIPUS intervention. J YAP and RIPK1 colocalization in chondrocytes were detected by IF after LIPUS intervention (scale bar: 10 um). K , L Immunohistochemistry staining to show the P-YAP-positive cell, P-RIPK1-positive cell and P-P65-positive cell in cartilage of the six groups (scale bar: 100 um). Data are shown as mean ± SD. *P<0.05, **P<0.01, ***P<0.001 Index in PubMed under a CC BY license. PMID: 38493143

7 Publications Citing This Product

1. PubMed ID: -, Xiong W,Wu L,Tang R,Zhang Q,Guo Q,Song S.Grape Seed Proanthocyanidins (GSPs) Inhibit the Development of Cutaneous Squamous Cell Carcinoma by Regulating the hsa_circ_0070934/miR-136-5p/PRAF2 Axis.Cancer Manag Res.2021;13:4359-4371https://doi.org/10.2147/CMAR.S302084

2. PubMed ID: ,

3. PubMed ID: 33130473, Sun K,Luo J,Jing X,Xiang W,Guo J,Yao X,Liang S,Guo F,Xu T.Hyperoside ameliorates the progression of osteoarthritis: An in vitro and in vivo study.Phytomedicine.2020 Oct 14;80:153387.doi:10.1016/j.phymed.2020. 153387.Epub ahead of print.PMID:33130473.

Visit bosterbio.com/anti-mmp3-rabbit-monoclonal-antibody-m00775-boster.html to see all 7 publications.

Submit a product review to Biocompare.com

Submit a review of this product to Biocompare.com to receive a $20 Amazon.com giftcard! Your

BOSTER BIOLOGICAL TECHNOLOGY
3942 B Valley Ave, Pleasanton, CA 94566
888-466-3604 | support@bosterbio.com | www.bosterbio.com

reviews help your fellow scientists make the right decisions. Thank you for your contribution.

Anti-MMP3 Rabbit Monoclonal Antibody

For Research Use Only. Not for use in diagnostic procedures.