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Anti-GAPDH Antibody Picoband®

Catalog Number: A00227-1

About GAPDH

Glyceraldehyde 3-phosphate dehydrogenase (abbreviated as GAPDH or less commonly as G3PDH) is an enzyme of ~37kDa that catalyzes the sixth step of glycolysis and thus serves to break down glucose for energy and carbon molecules. This gene encodes a member of the glyceraldehyde-3-phosphate dehydrogenase protein family. GAPDH is mapped to 12p13.31. The encoded protein has been identified as a moonlighting protein based on its ability to perform mechanistically distinct functions. The product of this gene catalyzes an important energy-yielding step in carbohydrate metabolism, the reversible oxidative phosphorylation of glyceraldehyde-3-phosphate in the presence of inorganic phosphate and nicotinamide adenine dinucleotide (NAD). The encoded protein has additionally been identified to have uracil DNA glycosylase activity in the nucleus.

Overview

Product Name	Anti-GAPDH Antibody Picoband®
Reactive Species	Chicken, Human, Monkey, Mouse, Rat, Zebrafish
Description	Boster Bio Anti-GAPDH Antibody Picoband® catalog # A00227-1. Tested in Flow Cytometry, IF, IHC, ICC, WB applications. This antibody reacts with Human, Mouse, Rat, Monkey, Chicken, Zebrafish. 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	Flow Cytometry, IF, IHC, ICC, 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	P04406

Technical Details

Immunogen	E.coli-derived human GAPDH recombinant protein (Position: N136-E335). Human GAPDH shares 95% and 94.5% amino acid (aa) sequence identity with mouse and rat GAPDH, respectively.
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot, and HRP Conjugated anti-Rabbit IgG Super Vision Assay Kit (SV0002-1) for IHC(P) and ICC.
Cross Reactivity	No cross-reactivity with other proteins
Isotype	Rabbit IgG

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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, Human, Monkey, Mouse, Rat, Chicken, Zebrafish Immunohistochemistry (Paraffin-embedded Section), 2-5ug/ml, Human Immunocytochemistry/Immunofluorescence, 5 ug/ml, Human Flow Cytometry(Fixed), 1-3 ug/1x10^6 cells, Human

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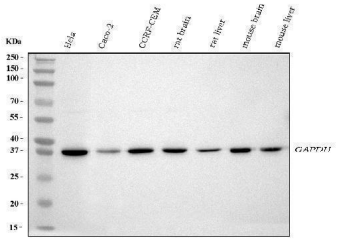
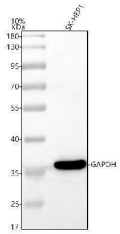
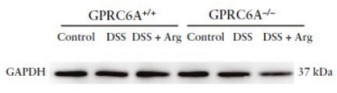
Anti-GAPDH Antibody Picoband® (A00227-1) Images



Western blot analysis of GAPDH using anti-GAPDH antibody (A00227-1). 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 30 ug of sample under reducing conditions. Lane 1: human Hela whole cell lysates, Lane 2: human CACO-2 whole cell lysates, Lane 3: human CCRF-CEM whole cell lysates, Lane 4: rat brain tissue lysates, Lane 5: rat liver tissue lysates, Lane 6: mouse brain tissue lysates, Lane 7: mouse liver tissue 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-GAPDH antigen affinity purified polyclonal antibody (Catalog # A00227-1) 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 1:5000 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 GAPDH at approximately 36 kDa. The expected band size for GAPDH is at 36 kDa.



Western blot analysis of GAPDH using anti-GAPDH antibody (A00227-1). 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: human SK-HEP1 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-GAPDH antigen affinity purified polyclonal antibody (A00227-1) 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 (Catalog # BA1054) 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 GAPDH at approximately 36 kDa. The expected band size for GAPDH is at 36 kDa.



Western blot analysis of GAPDH using anti-GAPDH antibody (A00227-1). 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 30 ug of sample under reducing conditions. Lane 1-6: mouse intestinal tissue 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-GAPDH antigen affinity purified polyclonal

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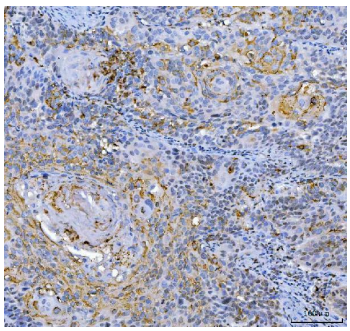
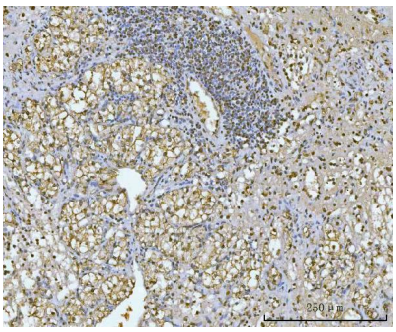
antibody (Catalog # A00227-1) at 1:1000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti rabbit secondary antibody at a dilution of 1:5000 for 1 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with ChemiDoc MP system. A specific band was detected for GAPDH at approximately 37 kDa. The expected band size for GAPDH is at 36 kDa.

Western blot analysis of GAPDH using anti-GAPDH antibody (A00227-1). 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 30 ug of sample under reducing conditions. Lane 1-2: human OCI-LY1whole 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-GAPDH antigen affinity purified polyclonal antibody (Catalog # A00227-1) at 1:5000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a HRP Conjugated AffiniPure Goat Anti-Rabbit IgG (H+L) for 1 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with ChemiDoc MP system. A specific band was detected for GAPDH at approximately 35 kDa. The expected band size for GAPDH is at 36 kDa.



IHC analysis of GAPDH using anti-GAPDH antibody (A00227-1). GAPDH was detected in a paraffin-embedded section of human laryngeal squamous cell carcinoma tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-GAPDH Antibody (A00227-1) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.



IHC analysis of GAPDH using anti-GAPDH antibody (A00227-1). GAPDH was detected in a paraffin-embedded section of human renal clear cell carcinoma tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-GAPDH Antibody (A00227-1) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.

IHC analysis of GAPDH using anti-GAPDH antibody (A00227-1). GAPDH was detected in a paraffin-embedded

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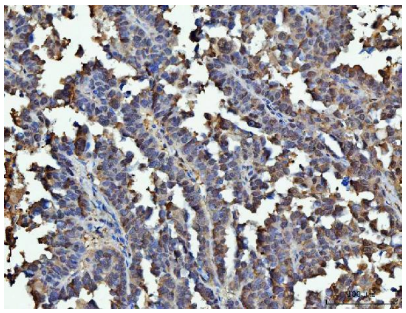
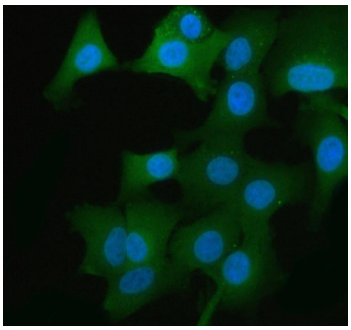
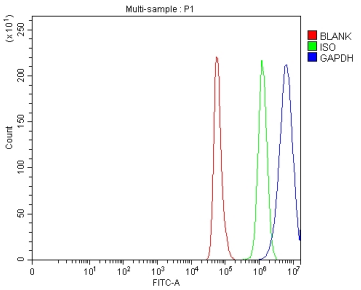


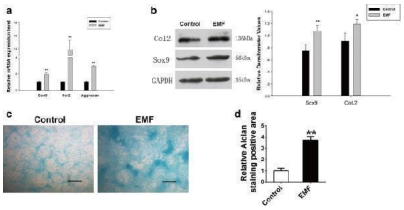
section of human ovarian serous cancer tissue. Heat mediated antigen retrieval was performed in EDTA buffer (pH 8.0, epitope retrieval solution). The tissue section was blocked with 10% goat serum. The tissue section was then incubated with 2 ug/ml rabbit anti-GAPDH Antibody (A00227-1) overnight at 4°C. Peroxidase Conjugated Goat Anti-rabbit IgG was used as secondary antibody and incubated for 30 minutes at 37°C. The tissue section was developed using HRP Conjugated Rabbit IgG Super Vision Assay Kit (Catalog # SV0002) with DAB as the chromogen.



IF analysis of GAPDH using anti-GAPDH antibody (A00227-1). GAPDH was detected in an immunocytochemical section of A549 cells. Enzyme antigen retrieval was performed using IHC enzyme antigen retrieval reagent (AR0022) for 15 mins. The cells were blocked with 10% goat serum. And then incubated with 5 ug/mL rabbit anti-GAPDH Antibody (A00227-1) overnight at 4°C. DyLight®488 Conjugated Goat Anti-Rabbit IgG (BA1127) was used as secondary antibody at 1:500 dilution and incubated for 30 minutes at 37°C. The section was counterstained with DAPI. Visualize using a fluorescence microscope and filter sets appropriate for the label used.

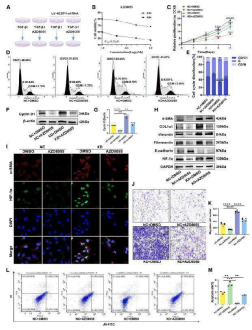


Flow Cytometry analysis of Hela cells using anti-GAPDH antibody (A00227-1). Overlay histogram showing Hela cells stained with A00227-1 (Blue line). To facilitate intracellular staining, cells were fixed with 4% paraformaldehyde and permeabilized with permeabilization buffer. The cells were blocked with 10% normal goat serum. And then incubated with rabbit anti-GAPDH Antibody (A00227-1, 1 ug/1x106 cells) for 30 min at 20°C. DyLight®488 conjugated goat anti-rabbit IgG (BA1127, 5-10 ug/1x106 cells) was used as secondary antibody for 30 minutes at 20°C. Isotype control antibody (Green line) was rabbit IgG (1 ug/1x106) used under the same conditions. Unlabelled sample without incubation with primary antibody and secondary antibody (Red line) was used as a blank control.

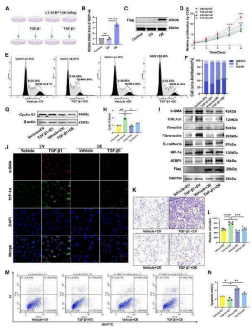


Continuous effects of EMF on BMSC chondrogenic differentiation capacity. a SOX9, CoL2, and Aggrecan mRNA levels of two groups determined by RT-PCR. GAPDH used as internal control for quantification (n = 3). b Expression of Col2 and Sox9 proteins of both groups detected by western blot analysis. Relative densitometer values quantified by ImageJ software, GAPDH served as loading control (n = 3). c Presentation of Alcian Blue staining of both groups. Scale bar = 100 um. d Semi-quantitative analysis of Alcian Blue staining among both groups (n = 6). Data shown as mean ± SD. * P

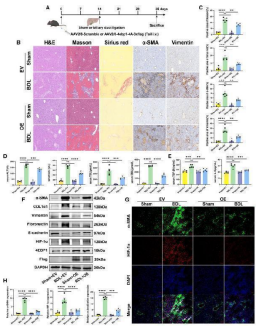
(L and M) (L) The apoptosis of LX-2 cells determined using flow cytometry and (M) percentages of apoptotic cells (early and late apoptotic cells). NC, LV-control-shRNA; KD, LV-4EBP1-shRNA. Values are the mean ± SD (unpaired two-



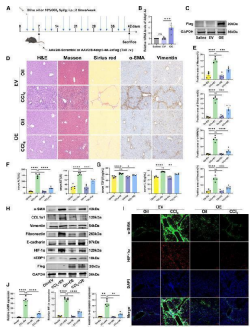
sample Student’s t test). ∗∗p < 0.01, ∗∗∗p < 0.001, and ∗∗∗∗p < 0.0001; ns, not significant. alpha-SMA, alpha-smooth muscle actin; DAPI, 4′,6-diamidino-2-phenylindole; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; HIF-1alpha, hypoxia inducible factor 1 subunit alpha; LV, lentivirus; PI, propidium iodide; TGF-beta1, transforming growth factor beta 1.Index in iScience under a CC BY license. DOI: 10.1016/j.isci.2025.113412



(M and N) (M) The apoptosis of vehicle or TGF-beta1-stimulated LX-2 cells transducted with LV-empty or LV-4EBP1-4A-3xflag determined using flow cytometry and (N) percentages of apoptotic cells (early and late apoptotic cells). EV, LV-empty; OE, LV-4EBP1-4A-3xflag. Values are the mean ± SD (unpaired two-sample Student’s t test). ∗p < 0.05, ∗∗p < 0.01, ∗∗∗p < 0.001, and ∗∗∗∗p < 0.0001; ns, not significant. alpha-SMA, alpha-smooth muscle actin; DAPI, 4′,6-diamidino-2-phenylindole; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; HIF-1alpha, hypoxia inducible factor 1 subunit alpha; LV, lentivirus; PI, propidium iodide; RT-qPCR, quantitative reverse transcription polymerase chain reaction; TGF-beta1, transforming growth factor beta 1.Index in iScience under a CC BY license. DOI: 10.1016/j.isci.2025.113412



(H) Relative quantification of alpha-SMA, HIF-1alpha, and colocalization of alpha-SMA and HIF-1alpha. EV, AAV2/8-scramble; OE, AAV2/8-4ebp1-4A-3xflag. Values are the mean ± SD (unpaired two-sample Student’s t test). ∗∗p < 0.01, ∗∗∗p < 0.001, and ∗∗∗∗p < 0.0001; ns, not significant. alpha-SMA, alpha-smooth muscle actin; AAV, adeno-associated virus; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BDL, bile duct ligation; DAPI, 4′,6-diamidino-2-phenylindole; DBIL, direct bilirubin; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; H&E, hematoxylin-eosin; HIF-1alpha, hypoxia inducible factor 1 subunit alpha; IL-6, Interleukin 6; TBIL, total bilirubin; TGF-beta1, transforming growth factor beta 1.Index in iScience under a CC BY license. DOI: 10.1016/j.isci.2025.113412



(J) Relative quantification of alpha-SMA, HIF-1alpha, and colocalization of alpha-SMA and HIF-1alpha. EV, AAV2/8-scramble; OE, AAV2/8-4ebp1-4A-3xflag. Values are the mean ± SD (unpaired two-sample Student’s t test). ∗p < 0.05, ∗∗p < 0.01, ∗∗∗p < 0.001, and ∗∗∗∗p < 0.0001; ns, not significant. alpha-SMA, alpha-smooth muscle actin; AAV, adeno-associated virus; ALT, alanine aminotransferase; AST, aspartate aminotransferase; DAPI, 4′,6-diamidino-2-phenylindole; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; H&E, hematoxylin and eosin; HIF-1alpha, hypoxia inducible factor 1 subunit alpha; IL-6, interleukin 6; RT-qPCR, quantitative reverse transcription polymerase chain reaction; TGF-beta1, transforming growth factor beta 1.Index in iScience under a

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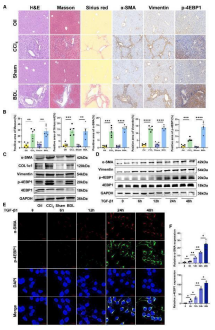
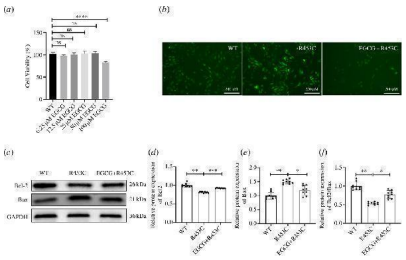
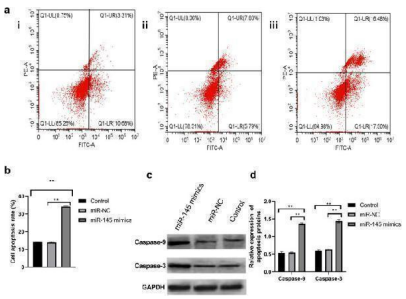


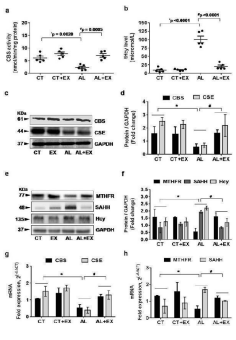
(E and F) Immunofluorescence double staining of alpha-SMA and p-4EBP1 in TGF-beta1-stimulated LX-2 cells for 0, 6, 12, 24, and 48 h (×400 magnification). Values are the mean ± SD (unpaired two-sample Student's t test). ∗p < 0.05, ∗∗p < 0.01, ∗∗∗p < 0.001, and ∗∗∗∗p < 0.0001. alpha-SMA, alpha-smooth muscle actin; BDL, bile duct ligation; DAPI, 4′,6-diamidino-2-phenylindole; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; H&E, hematoxylin and eosin; TGF-beta1, transforming growth factor beta1.Index in iScience under a CC BY license. DOI: 10.1016/j.isci.2025.113412



Effects of EGCG on the MYH7 R453C mutation H9C2 cells. (a) Effects of EGCG on the MYH7 R453C mutation H9C2 cell survival rate. (b) Protective effects of EGCG in MYH7 R453C mutation on ROS production. (c) Effect of EGCG on Bax and Bcl-2 activities of the MYH7 R453C mutation H9C2 cells. (d) Bcl-2 expression were quantified by densitometry and normalized to GAPDH levels. (e) Bax expression was quantified by densitometry and normalized to GAPDH levels. (f) Quantitative analysis of the ratio of Bcl-2 to Bax in protein expression was evaluated. Data are expressed as the mean ± s.d. *p < 0.05, **p < 0.01, ***p < 0.001, ****p < 0.0001. n = 3 biologically independent samples. Index in PubMed under a CC BY license. PMID: 38862020



(a) The representative cell apoptosis photos using flow cytometry, (i) The blank control group; (ii) The miR-NC transfection group; (iii) The miR-145 mimics transfection group. (b) The apoptosis rate of the miR-145 mimics transfection group increased significantly, ** p



Alcohol interferes with Hcy metabolism leading to hyperhomocysteinemia (HHcy). (a,b) Scatter dot plots represent data for the CBS enzyme activity and total homocysteine (tHcy) levels in the different mice groups. (c–f) Representative western blot analysis for the vital enzymes (CBS, CSE, MTHFR, SAHH and Hcy) involved in homocysteine metabolism in different mice groups. Bar graphs showing quantitative estimation of key proteins after normalization with GAPDH. (g,h) q-PCR analysis showing the data for real-time transcript levels of CBS, CSE, MTHFR and SAHH mRNAs in the different groups of mice. All the data are represented as mean values ± standard error (SE) in 5 independent experiments. * ,# p<0.05 considered significant. *p<0.05 vs. CT and # p<0.05 vs. AL group.Uncropped blots for c and e are presented in Supplementary Figs and . Index in PubMed under a CC BY license. PMID: 29581524

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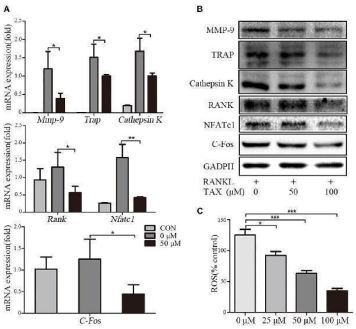
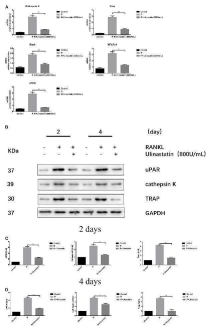
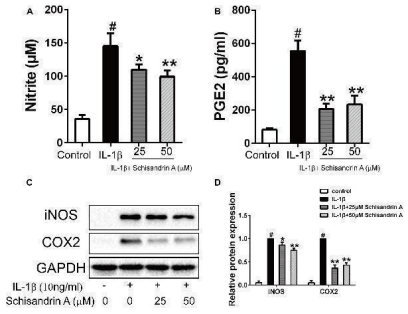


Taxifolin suppresses expression of osteoclast specific genes and proteins. RAW264.7 cells were treated with RANKL and with or without different concentrations of taxifolin for 3 days, (A) expression of Trap, Mmp-9, Cathepsin K, C-Fos, Nfatc1 , and Rank was determined by qRT-PCR and calculated in relation to the internal control GAPDH mRNA by the comparative Ct method; (B) immunoblots with MMP-9, TRAP, Cathepsin K, RANK, NFATc1 and C-Fos antibodies demonstrating that taxifolin repressed osteoclast-specific protein expression. GAPDH antibody was used as loading controls. (C) RAW264.7 cells were cultured with taxifolin for 36 h, then stimulated with RANKL (50 ng/ml) for 30 min, and RAW264.7 cells without RANKL or taxifolin was considered as “100% control,” our results showed taxifolin decreased the release of intracellular ROS. Data are presented as mean ± SD. n = 3, ∗ P < 0.05, ∗∗ P < 0.01, ∗∗∗ P < 0.001.Index in PubMed under a CC BY license. PMID: 30483128



Ulinastatin reduces the expression of uPAR, NFATc1 and osteoclast marker genes induced by RANKL. C represents control group, R represents RANKL group, R + ulinastatin represents RANKL + 800 units/mL ulinastatin group. (A) Ulinastatin reduces RANKL-induced mRNA expression of cathepsin K, Trap, Rank, NFATc1, and uPAR. BMMs were cultured with M-CSF (30 ng/mL) and RANKL (100 ng/mL), and treated with or without ulinastatin (800 units/mL) for 4 days. mRNA expression was detected by qRT-PCR. The qRT-PCR experiments have been repeated with different RNA preparations for 3 times independently. Data are represented as mean ± SD . ∗ P < 0.05 and ∗∗ P < 0.01. (B,C,D) Ulinastatin reduces RANKL-induced protein expression of uPAR, cathepsin K and Trap. BMMs were cultured with M-CSF (30 ng/mL) and RANKL (100 ng/mL), and treated with or without ulinastatin (800 units/mL) for 2 or 4 days. Protein expression levels of uPAR, cathepsin K and Trap were examined by western blotting at the indicated times. The amount of loaded protein was 25 ug. The experiment was performed three times independently and GAPDH was used as a loading control. ∗ P < 0.05, ∗∗ P < 0.01 versus RANKL group.Index in PubMed under a CC BY license. PMID: 30245631



Effects of Schisandrin A on IL-1beta-induced production of NO, PGE2, iNOS, and COX2 in rat chondrocytes. Cells were treated with Schisandrin A (25, 50 uM) in the presence or absence of IL-1beta (10 ng/ml) for 24 h. Cell culture supernatants were collected. (A) Griess reaction was used to measure the NO concentration (n = 3). (B) PGE2 level was accessed by ELISA (n = 3). (C) Expression of iNOS and COX2 were detected by Western blot. (D) Relative protein expression was qualified by ImageJ software, GAPDH was served as the loading control (n = 3). # P < 0.05 vs. control group; ∗ P < 0.05 vs. IL-1beta group; ∗∗ P < 0.01 vs. IL-1beta group.Index in PubMed under a CC BY license. PMID: 30761007

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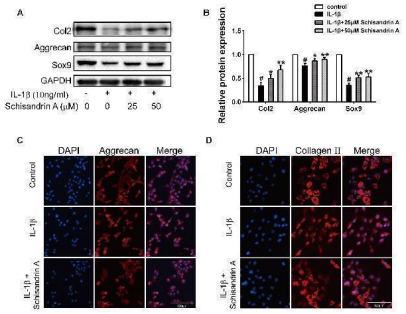
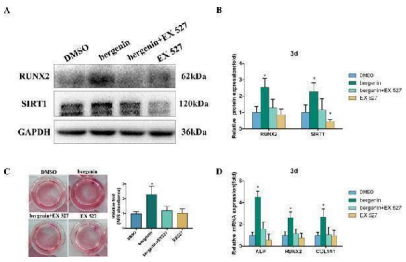
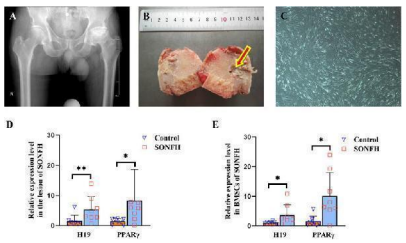


Effects of Schisandrin A on IL-1beta-induced cartilage degradation. (A) Cells were treated with Schisandrin A (25, 50 uM) in the presence or absence of IL-1beta (10 ng/ml) for 24 h. Protein levels of Collagen II, Aggrecan and Sox9 were determined by Western Blot. (B) Relative protein expression was qualified by Image-J software, GAPDH was used as the loading control (n = 3). (C) Aggrecan and (D) Collagen II were observed by Immunofluorescence after cells were treated with IL-1beta (10 ng/ml) with or without Schisandrin A (50 uM) for 24 h. # P < 0.05 vs. control group; ∗ P < 0.05 vs. IL-1beta group; ∗∗ P < 0.01 vs. IL-1beta group.Index in PubMed under a CC BY license. PMID: 30761007



Effects of a SIRT1-specific inhibitor (EX 527) on the enhanced expression of an osteoblast-specific gene of bone marrow mesenchymal stem cells by Bergenin. (A and B) The expression of RUNX2 and SIRT1 in blank, control + EX 527, Bergenin (10 uM), and Bergenin (10 uM) + EX 527 groups was determined by Western blot analysis. EX 527 (10 uM) was applied for 1 h, followed by culture in osteogenic induction medium with Bergenin for 3 days. Protein expression levels were normalized to glyceraldehyde-3-phosphate dehydrogenase. Data are expressed as the mean ± standard deviation (SD) of three independent experiments, and one of three independent experiments is shown. Data are expressed as the mean ± SD. * P < 0.05 vs. group with osteogenic induction medium alone. (C) Alizarin red staining and quantification of mineralization at day 12 of osteogenic differentiation. (D) The mRNA expression of RUNX2, ALP, and COL1A1 in blank, control + EX 527, Bergenin (10 uM), and Bergenin (10 uM) + EX 527 groups was determined by quantitative reverse transcription polymerase chain reaction. EX 527 (10 uM) was applied for 1 h, followed by culture in osteogenic induction medium with Bergenin for 3 days. mRNA expression levels were normalized to GAPDH. * P < 0.05 vs. BMSCs treated with osteogenic induction medium alone.Index in PubMed under a CC BY license. PMID: 31258473



H19 and PPARgamma are upregulated in the femoral head and BMSCs of patients with SONFH. (A, B) X-ray photo and pathological structure of the femoral head from an ARCO stage V SONFH patient. The images show alterations in the morphology of the femoral head, characterized by collapse and flattening, as well as radiographic signs indicative of hip osteoarthritis. (C) Morphology of BMSCs from a patient with SONFH. (D, E) Expression levels of H19 and PPARgamma in the femoral head and BMSCs from a patient with SONFH. All experimental procedures were performed in triplicate with internal normalization to GAPDH expression levels. The relative expression levels of each gene were analyzed using the 2 −△△Ct method (n = 8, all data are shown as the mean ± SD of three independent experiments, *p < 0.05, **p < 0.01).Index in PubMed under a CC BY license. PMID: 40259926

Effect of exercise on alcohol induced DNA methyltransferase

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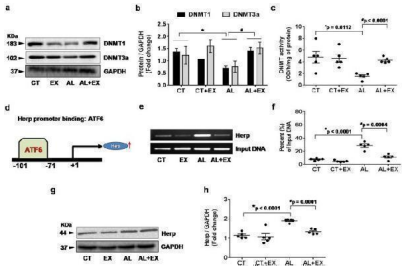
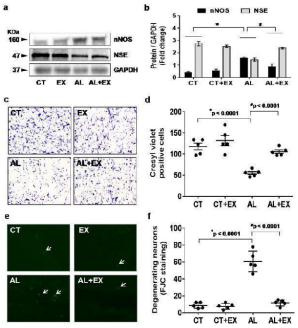


(DNMT) activity and transcriptional regulation through ATF6-Herp signaling. (a–c) Representative western blot analysis showing the levels of DNMT1 and DNMT3a in brain tissue extract from the different mice groups. Bar graphs showing the quantitative estimation of DNMT1 and DNMT3a proteins after normalization with GAPDH. Scatter dot plot representing the DNMT activity in brain tissue of different mice groups. (d–f) ChIP assay to examine the ER activated ATF6 binding to the endogenous Herp promoter in vivo . After cross linking and immunoprecipitation with ATF6 antibody from isolated brain tissue extract, PCR was performed to identify the presence of Herp promoter DNA using primers flanking the CpG islands in the Herp promoter sites. Scatter dot plot representing the percent of input DNA in different experimental mice groups. (g,h) Representative western blot analysis showing the levels of Herp protein expression in brain tissue extract from the different mice groups. Scatter dot plot showing the quantitative estimation of Herp proteins after normalization with GAPDH. All the data are represented as mean values ± standard error (SE) in 5 independent experiments. * ,# p<0.05 considered significant. *p<0.05 vs. CT and # p<0.05 vs. AL group. Uncropped blots for a,e,g are presented in Supplementary Figs and . Index in PubMed under a CC BY license. PMID: 29581524



Effect of exercise on alcohol induced neuronal damage. (a,b) Representative western blot analysis showing the levels of neuronal proteins (NeuN and NSC) in different mice groups (a). Histogram showing the quantitative estimation of nNOS and NSE proteins after normalization with GAPDH (b). (c,d) Representative images showing coronal slices of mice brains stained with cresyl violet (40× magnification) (c). Scatter dot plot showing the number of cresyl violet positive cells in different groups of mice (d). (e,f) Representative images showing Fluoro-Jade C (FJC) staining in brain sections of the different groups of mice (10× magnification). A marked decrease of FJC-stained degenerating neurons (arrows) were observed in CT, EX and AL+EX groups, indicating a lesser degree of neuronal cell death. Brain sections of AL treated mice showing a greater number of FJC-positive neurons (arrows), reflecting increased neuronal cell death (e). Scatter dot plot showing the numbers of degenerating neurons in different experimental mice groups (f). All the data are represented as mean values ± standard error (SE) in 5 independent experiments. * ,# p<0.05 considered significant. *p<0.05 vs. CT and # p<0.05 vs. AL group. Uncropped blots for a are presented in Supplementary Fig. . Index in PubMed under a CC BY license. PMID: 29581524

D206008 activates beta-catenin signaling pathway via the phosphorylation of GSK-3beta and Akt. Notes: (A) Cells were treated with different concentrations (0–100 μM) of D206008 for 48 hours, and the levels of p-Akt, p-GSK-3beta, p-beta-catenin, and beta-catenin proteins were analyzed relative to beta-actin expression. (B) B16 cells treated with either vehicle (0.1% DMSO) or D206008 (100 μM) for 48 hours were analyzed for the levels of nuclear and

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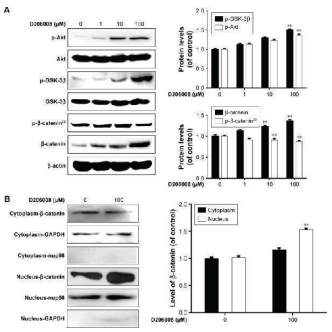
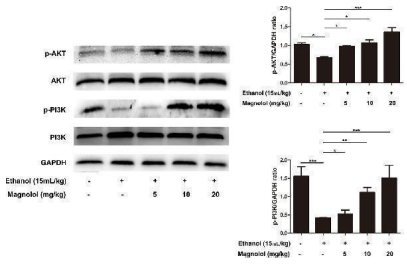
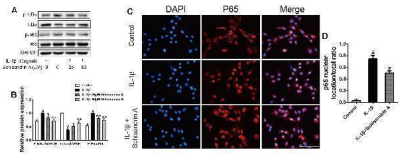


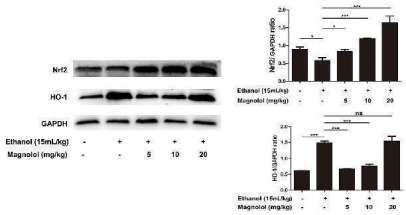
cytoplasmic beta-catenin. Cytoplasmic protein levels were normalized against GAPDH and nuclear protein against nup98. The band densities of proteins were measured by the Quantity One program. The data are shown as mean ± SD and analyzed by one-way ANOVA followed by Tukey's test. ** P



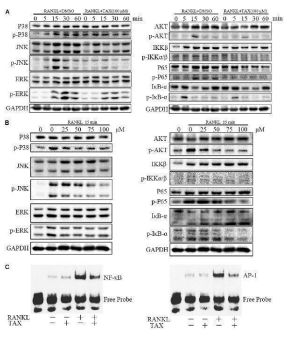
Effects of magnolol on mice alcohol-induced liver damage in the AKT/PI3K signaling pathway. Liver tissues were extracted for protein analysis by western blotting. AKT and PI3K, proteins expression were detected. The levels of AKT and PI3K were compared with GAPDH. The data were demonstrated as means ± SD. (*P < 0.05, **P < 0.01, ***P < 0.001).Index in PubMed under a CC BY license. PMID: 31920652



Effects of Schisandrin A on NF-kappaB signaling pathway. Cells were exposed to Schisandrin A (25, 50 uM) with or without IL-1beta (10 ng/ml) for 30 min. (A) Protein levels of p-IkappaBalpha, IkappaBalpha, p-p65, p65 were detected by Western blot. (B) Relative protein expression was qualified by ImageJ software, GAPDH and p65 were used as the loading control, respectively (n = 3). (C) p65 translocation was observed by Immunofluorescence. (D) Quantitative analysis of p65 nuclear location/total ratio of three groups. # P < 0.05 vs. control group; ∗ P < 0.05 vs. IL-1beta group; ∗∗ P < 0.01 vs. IL-1beta group.Index in PubMed under a CC BY license. PMID: 30761007

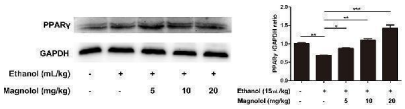


Effects of magnolol on mice alcohol-induced liver damage in the Nrf2/HO-1 signaling pathway. After the completion of modeling and samples were collected, the liver of mice was lysed to detect the proteins by western blotting analysis. The levels of Nrf2 and HO-1 were compared with GAPDH. The data were demonstrated as means ± SD. (*P < 0.05, ***P < 0.001 and "ns" means not significant).Index in PubMed under a CC BY license. PMID: 31920652

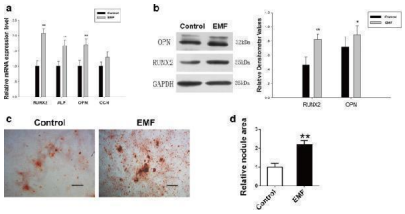


Taxifolin represses multiple pathways of osteoclastogenesis in RAW264.7 cells. (A) RAW264.7 cells were pre-treated with 100 uM taxifolin for 2 h, then stimulated with RANKL (50 ng/ml) for the indicated time, protein was extracted for immunoblotting, the same GAPDH was used as loading control. (B) RAW264.7 cells were pre-treated with taxifolin in indicating concentrations for 2 h, then stimulated with RANKL (50 ng/ml) for 15 min, protein was extracted for immunoblotting, the same GAPDH was used as loading control. (C) Electrophoretic mobility shift assay for DNA binding activity of NF-kappaB and AP-1. After treatment with 100 uM taxifolin for 2 h, RAW264.7 cells were stimulated with RANKL (50 ng/ml) for 30 min, then nuclear extracts

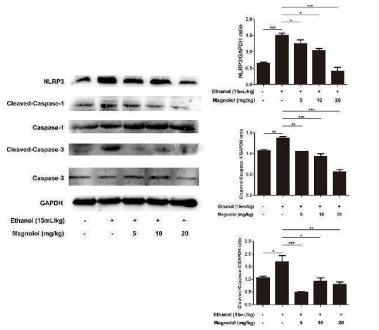
were prepared and analyzed for DNA binding activity. Data are of three independent experiments.Index in PubMed under a CC BY license. PMID: 30483128



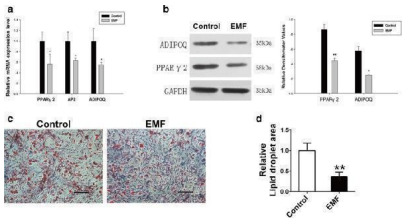
Effects of magnolol on mice alcohol-induced liver damage in PPARgamma expression. The collected samples were analyzed for the expression of PPARgamma using western blotting analysis. The expression of PPARgamma was compared with GAPDH. The data were demonstrated as means ± SD. (*P < 0.05, **P < 0.01, ***P < 0.001 and "ns" means not significant).Index in PubMed under a CC BY license. PMID: 31920652



BMSCs pretreated with EMF exhibited stronger osteogenic differentiation potential. a RUNX2, ALP, OPN, and OCN mRNA levels of two groups analyzed by RT-PCR. GAPDH used as loading control for quantification (n = 3). b Expression of OPN and RUNX2 proteins of both groups determined by western blot analysis. Relative densitometer values quantified by ImageJ software, GAPDH used as internal control (n = 3). c Images of Alizarin Red S staining exhibited plaques of calcified extracellular matrix of both groups. Scale bar = 100 um. d Semi-quantitative analysis of Alizarin Red S staining among both groups (n = 6). Data shown as mean ± SD. * P



Effects of magnolol on NLRP3 inflammasome, caspase-1 and caspase-3 signaling pathway in ALD mice. Magnolol was given to mice for three times, and then alcohol was gavaged. The level of NLRP3 inflammasome, caspase-1 and caspase-3 was detected by western blotting analysis with the compared with the internal control (GAPDH). The data is presented as mean ± SD. (*P < 0.05, **P < 0.01, ***P < 0.001).Index in PubMed under a CC BY license. PMID: 31920652



EMF treatment had lasting impact on BMSC adipogenic differentiation potential. a PPARgamma2, AP2, and ADIPOQ mRNA levels of two groups detected by RT-PCR. GAPDH served as loading control for quantification (n = 3). b Expression of ADIPOQ and PPARgamma2 proteins of both groups determined by western blot analysis. Relative densitometer values quantified by ImageJ software, GAPDH used as internal control (n = 3). c Images of Oil Red O staining showed lipid droplets of both groups. Scale bar = 25 um. d Semiquantitative analysis of Oil Red O staining among both groups (n = 6). Data shown as mean ± SD. * P

Different PPARalpha ligands exhibited different abilities to inhibit tumour sizes and metastasis. a Serum triglyceride levels in TC-1 tumour-bearing mice (n = 6–11). * P

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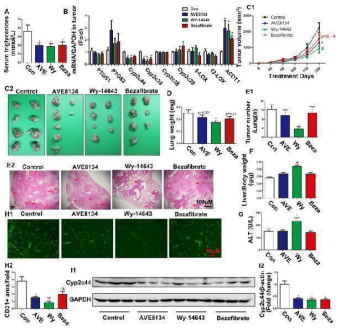
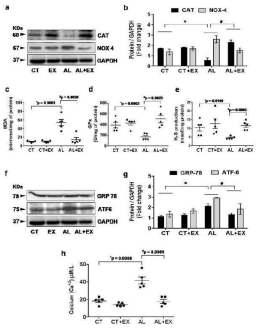
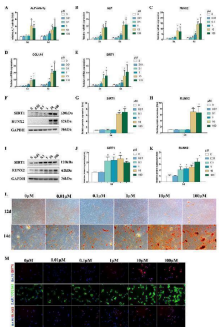




Effect of exercise on alcohol induced oxidative and endoplasmic reticular (ER) stress. (a,b) Representative western blot analysis showing the levels of antioxidant marker CAT and oxidative stress marker NOX4 in the different mice groups. Bar graphs showing the quantitative estimation of CAT and NOX4 proteins after normalization with GAPDH. (c–e) Scatter dot plots representing the levels of malondialdehyde (MDA), glutathione peroxidase (GPx) and production of H 2 S in brain tissue in different mice groups. (f,g) Representative western blot analysis showing the levels of GRP78 and ATF6 (hallmarks of ER stress) in the different groups of mice. Bar graphs showing the quantitative estimation of GRP78 and ATF6 proteins after normalization with GAPDH. (h) Scatter dot plot represents data for the cellular calcium ion (Ca +2) level in brain tissue extract of different mice groups. All the data are represented as mean values ± standard error (SE) in 5 independent experiments. * ,# p<0.05 considered significant. *p<0.05 vs. CT and # p<0.05 vs. AL group. Uncropped blots for a and f are presented in Supplementary Fig. . Index in PubMed under a CC BY license. PMID: 29581524



Effects of Bergenin on osteogenesis of bone marrow mesenchymal stem cells (BMSCs). (A) The effects of Bergenin on alkaline phosphatase activity at days 3 and 5 during the osteogenic differentiation of BMSCs. (B – E) mRNA expression of RUNX2, ALP, and COL1A1 was determined by quantitative reverse transcription polymerase chain reaction at day 3 and day 5 during osteogenic differentiation. mRNA expression levels were normalized to GAPDH. (F – K) The expression of RUNX2 and SIRT1 protein was determined by Western blot analysis after osteogenic differentiation at days 3 and 5. Protein expression levels were normalized to glyceraldehyde-3-phosphate dehydrogenase. Data are expressed as the mean ± standard deviation (SD) of three independent experiments, and one of three independent experiments is shown. Data are expressed as the mean ± SD, n = 3. * P < 0.05 vs. BMSCs treated with osteogenic induction medium alone. (L) Alizarin red staining at days 12 and 14 of osteogenic differentiation. Magnification ×40. (M) Immunofluorescence staining showing that the protein levels of RUNX2, COL1A1, and SIRT1 are upregulated by the addition of Bergenin (10 or 100 uM) at day 3 of osteogenic differentiation. COL1A1 is stained green. RUNX2 and SIRT1 are stained red. Nuclei are stained with 4′,6-diamidino-2-phenylindole (blue). Magnification

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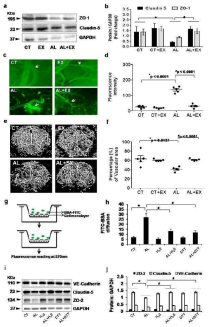
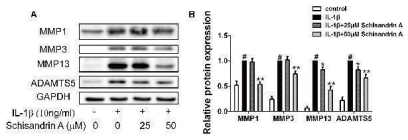


(a , b) Effect of exercise on alcohol induced vascular permeability and BBB dysfunction. Representative western blot analysis showing the levels of tight junction (TJ) proteins (ZO-1 and Claudin-5) in the different mice groups (a). Histogram showing the quantitative estimation of ZO-1 and Claudin-5 proteins after normalization with GAPDH (b). (c,d) Representative images showing fluorescent protein (FITC-BSA) leakage from pial vessels into brain parenchyma – indicating alteration in microvascular permeability in the different groups of mice (c). Scatter dot plot showing quantitative estimation of fluorescent intensity units (FIU) in the different mice groups after FITC-BSA injection (d). (e , f) Representative images of cerebral angiogram with barium sulfate contrast in experimental mice groups (e). Scatter dot plot showing the pattern of vascular density in the form of percentage of vascular area in the different mice groups (f). (g , h) Representative images for the in vitro model showing microvascular permeability in brain endothelial cells (bEnd.3 cells) by FITC-BSA diffusion assay. Fluorescence intensity of bovine serum albumin conjugated with FITC (BSA-488) in lower chambers of Transwells was measured by fluorimetry and presented as FIU (g). Histogram showing quantitative estimation of FIU in different experimental conditions after FITC-BSA treatment in Transwell chambers (h). (i , j) Representative western blot analysis showing the levels of junctional proteins (VE-Cadherin, Claudin-5 and ZO-2) in different experimental conditions of mouse brain endothelial cells (i). Histograms showing the quantitative estimation of ZO-2, Claudin-5 and VE-Cadherin proteins after normalization with GAPDH (j). All the data are represented as mean values ± standard error (SE) in 5 independent experiments. * ,# p<0.05 considered significant.*p<0.05 vs. CT and # p<0.05 vs. AL group. Uncropped blots for Fig. are presented in Supplementary Fig. and . Index in PubMed under a CC BY license. PMID: 29581524



Effects of Schisandrin A on IL-1beta-induced MMPs and ADAMTS5 protein expression. Chondrocytes were exposed to Schisandrin A (25, 50 uM) with or without IL-1beta (10 ng/ml) for 24 h. (A) Western blot was employed to determine the expression of MMP1, MMP3, MMP13, and ADAMTS5. (B) Relative protein expression was qualified by Image-J software, GAPDH was used as the internal control (n = 3). # P < 0.05 vs. control group; ∗ P < 0.05 vs. IL-1beta group; ∗∗ P < 0.01 vs. IL-1beta group.Index in PubMed under a CC BY license. PMID: 30761007

246 Publications Citing This Product

1. PubMed ID: 10.3389/fphar.2018.01016, Ulinastatin Inhibits Osteoclastogenesis and Suppresses Ovariectomy-Induced Bone Loss by Downregulating uPAR

2. PubMed ID: 10.3389/fphar.2019.00618, Bergenin Activates SIRT1 as a Novel Therapeutic Agent for Osteogenesis of Bone Mesenchymal Stem Cells

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3. PubMed ID: 10.2217/epi-2020-0155, miRNA 146b-5p protects against atherosclerosis by inhibiting vascular smooth muscle cell proliferation and migration

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