Supplementary Methods

Animal Models and Group Design

Six-week-old female C57BL/6J mice were obtained from the Laboratory Animal Research Center of the Fourth Military Medical University, Xi'an, China. All care administered to animals was approved by the Ethics Committee of Fourth Military Medical University and performed according to institutional guidelines and "Animal Research: Reporting In Vivo Experiments (ARRIVE)" guidelines (Supplementary Information). In the experimental groups, a unilateral anterior crossbite (UAC) prosthesis was bound to the lower incisors [Sun et al. 2020]. In the sham control group, the mice were subjected to a mock surgical procedure with no permanent bonding of the UAC prosthesis. AgomiR-29b, antagomiR-29b, or their corresponding negative control (NC), were injected separately into local areas of the mouse's bilateral TMJs, together with a BMSC-targeting aptamer that specifically binds to BMSCs [Li et al. 2015]. Sham control and UAC mice received either 25 μl of the aptamer-agomiR-29b complex (100 nM), the aptamer-antagomiR-29b complex (100 nM), or the equivalent volume of NC twice per week for 3 weeks. We determined the doses of the complexes based on the results of a previous study [Li et al. 2015], and according to our own pilot study. The same standardized diet was provided to all the mice. During the experiment, no mouse displayed any indications of disability.

Experimental Operation and Sample Assignment

After 3 weeks, all mice from sham control and UAC groups were sacrificed. In the first run of the experiment, 38 UAC mice and 38 sham control mice were used. Our previous studies identified no differences in the change in degradation between the right and left side of the TMJs in the UAC mice [Jiao et al. 2014]; therefore, the left condylar subchondral bones of 18 mice were used for BMSCs isolation, culture, the miRNA microarray assay (N = 3), and quantitative real-time PCR (qRT-PCR) analysis (N = 5), and their right condylar subchondral bones were used for BMSCs isolation, culture, and western blotting (N = 5). For the other 20 mice in each group, the condylar subchondral bones were used for BMSC isolation and culture,

stimulation by the *Mir29b* mimic or inhibitor, quantitative real-time polymerase chain reaction (qRT-PCR) (N = 5), and western blotting (N = 5). In the second run of the experiment, 76 UAC mice and 76 sham control mice were used. The UAC and sham control mice were treated with aptamer-agomiR29b, aptamer-antagomiR29b, or their negative controls (NC), respectively (N = 19). The left condylar subchondral bones of 10 mice in each group were used for micro-CT, histomorphometry, and tartrate-resistant acid phosphatase (TRAP) staining, respectively (N = 10), and their right condylar subchondral bones were used for qRT-PCR analysis (N = 5). For the other nine mice in each group, tissue blocks of the left condylar cartilage were fixed, decalcified, and embedded in paraffin to prepare 5 μm-thick sagittal sections. Immunohistochemical and histochemical staining were performed on these sections (N = 9). For their right condylar tissue blocks, the condylar cartilages were carefully dissected under a dissecting microscope (SZX9, Olympus, Tokyo, Japan) and used for qRT-PCR (N = 3). In the third run of the experiment, 40 six-week old normal female mice were used. Their condylar subchondral bones were used for BMSCs isolation and culture, stimulation by the *Mir29b* mimic or its inhibitor, for luciferase reporter assays, qRT-PCR of osteogenic genes (N = 5), and alkaline phosphatase (ALP) and alizarin red staining and quantification (N = 5).

Cell Isolation and Culture

A previously described method was used to isolate BMSCs from the condylar subchondral bone of UAC and sham control mice [Yang et al. 2015]. Digestion with 4 mg/ml dispase II and 3 mg/ml collagenase type I and (Roche Diagnostic, Indianapolis, IN, USA) was used to obtain all nucleated cells (ANCs) from the condylar subchondral bones. We then produced ANC single-cell suspensions, which were seeded in 100-mm dishes at 1.5×10^6 cell per dish. Single colony forming cells were passaged to P1 and then used in subsequent experiments. BMSCs were cultured in α-MEM with 100 mg/ml streptomycin, 2% 100 U/ml penicillin, 1% glutamine, and 10% heat-inactivated fetal bovine serum. Subsequent experiments used BMSCs from passage 3 (P3). Markers (SCA1, CD29, CD45, and CD11b) on the cell surface of the harvested MSCs (P3) were stained and then the cells were subjected to flow

cytometry analysis. Chondrogenic, osteogenic, and adipogenic culture conditions were used to assess their multi-differentiation abilities.

Flushing of the small pieces of condylar subchondral bone of UAC and sham control mice was used to harvest mononuclear cells. The harvested bone marrow cells were then grown on α-MEM containing 30 ng/ml macrophage colony-stimulating factor (MCSF; R&D Systems Inc., Minneapolis, MN, USA 100 μg/ml streptomycin sulfate, 100 U/ml penicillin, and 10% FBS, in Petri dishes overnight. Adherent cells were discarded, and mononuclear cells were obtained from the floating cells by incubation with MCSF (30 ng/ml). To obtain preosteoclasts, mononuclear cells were seeded at 1×10^5 cells per well in 24-well plates containing 30 ng/ml MCSF and 60 ng/ml RANKL per well (Pepro Tech, London, UK) and cultured for 3 days.

MiRNA Microarray Experiment

Total RNA was extracted from BMSCs of UAC and sham mice from which small RNAs were isolated and labeled with Cy3. The miRNA microarray and the gene microarray were performed using the LC Sciences microarray platform (LC Sciences, Houston, TX, USA), which contained probe sequences based on Sanger miRBase database 17.0 (http://www.mirbase.org/).

Delivery System for BMSC-specific Aptamers

Genscript Co. (Piscataway, NJ, USA) synthesized the BMSC-specific aptamers. The sequence of the aptamer was 5′-GAATTCAGTCGGACAGCGACGAC GGTGATATGTCAAGGTCGTATGCACGAGTCAGAGGGATGGACGAATATCG TCTCCC-3′ [Li et al. 2015]. RiboBio Co. (Guangzhou, China) synthesized agomiR-29b, antagomiR-29b, and their respective negative controls (NCs). The sequence of agomiR-29b was 5′-GCUGGUUUCAUAUGGUGGUUUA-3′, and the sequence of antagomiR-29b was 5′-GCUGGUUUCAUAUGGUGGUUUA-3′ [Liu et al. 2015]. One volume of polyethyleneimine (PEI) solution (100 μg/ml, pH 6.0) was mixed with six volumes of 4.2 μM sodium citrate to form the PEI-citrate nanocore structure. The nanocomplexes were then assembled by adding three volumes of synthetic BMSC aptamers (50 nM) and agomiR-29b (1 μM) or antagomiR-29b (1 μM) to the nanocore for 5 minutes [Li et al. 2015]. Either 25 μl of the

aptamer-agomiR-29b complex (100 nM), the aptamer-antagomiR-29b complex (100 nM), or a comparable volume of the appropriate NCs were delivered to mice via injection into the joint capsule of the TMJ twice a week for 3 weeks.

Flow Cytometry Analysis

Cultured BMSCs (P3) were washed with PBS and detached with nonenzymatic dissociation buffer for 15 minutes. After washing, the cells were incubated with the FITC-labeled ssDNA pool dissolved in 200μl binding buffer at a final concentration of 250 nM for 45 minutes. Cells were washed twice with 1 ml washing buffer and resuspended in 200 μl binding buffer. Fluorescence was determined with a FACScan cytometer (BD Immunocytometry Systems). Percentage of FITC-positive cells were calculated.

Temporomandibular Joint Local Area Injection.

To inject the temporomandibular joint local area, 25 μl each of the above-mentioned drugs were injected into the local TMJ regions of the experimental mice twice each week, from day 1 when the anterior crossbite prosthesis was inserted. The injection technique was the same as that detailed in our previous studies [Jiao et al. 2016].

Analysis Using Micro-computed Tomography (micro-CT)

Micro-CT was used to assess the bone mineral density (BMD) and bone microstructure of the subchondral bone, as previously described [Jiao et al. 2014]. Briefly, samples were subjected to scanning at 80 keV and 500 mA. Then, we generated 8-μm isotropic resolution, two-dimensional slices for three-dimensional reconstruction. As regions of interest (ROIs) for subsequent analyses, two cubes (0.3 × 0.3 × 0.3 mm each) were selected from the midcenter and midposterior. Within these ROIs, MicroView software (GE Healthcare, Pittsburgh, PA, USA) was used to quantify structural parameters such as the trabecular number (Tb.N), trabecular thickness (Tb.Th), trabecular separation (Tb.Sp), bone volume fraction (BV/TV), and the BMD, after setting a determinate threshold.

Immunohistochemical and Histochemical Staining

Hematoxylin-eosin (HE) and Safranin O/fast green staining of sagittal sections from each condyle were used for histological evaluation [Sun et al. 2020]. In addition, collagen II levels were detected using immunohistochemical staining [Sun et al. 2020]. In the Safranin O/fast green staining protocol, sections were deparaffinized to distilled water and then stained using 0.02% Fast Green solution for 5 min. The sections were the washed quickly (< 15 s) using 1% acetic acid solution. The sections were then stained using 0.1% safranin O solution for 5 min; dehydrated with 95% ethyl alcohol, absolute ethyl alcohol, and xylene; before being mounted with resinous medium. In the immunohistochemistry protocol, 1.5% goat serum was used to block the sections (Millipore-Sigma, Burlington, MA, USA), which were then incubated with the primary antibodies. Negative control sections were incubated with non-immune goat serum instead of the primary antibodies. Thereafter, peroxidase-conjugated secondary antibodies (Jackson Immuno Research Laboratories, Inc., West Grove, P, USA) were incubated with the sections, which were then visualized using 3,3-diaminobenzidine solution (DAB Substrate Kit1, Vector Laboratories, Inc., Burlingame, CA, USA). Counter staining was achieved using hematoxylin. The primary antibody comprised: goat anti-collagen II (1:50, sc-7763; Santa Cruz Biotechnology, Santa Cruz, CA, USA). We measured the number of immunopositive cells, the percentage of the Safranin O-positive area chondrocyte density, and the thickness of the condylar cartilage according to a previously described method [Sun et al. 2020].

MiR-29b **Mimic or Inhibitor Stimulation**

The sequence of *miR-29b* mimic used in the study was 5′-UAGCACCAUUUGAAAUCAGUGUU-3′, and the sequence of *miR-29b* inhibitor was 5′-AACACUGAUUUCAAAUGGUGCUA-3′ [Liu et al. 2015]. Cultured BMSCs (P3) were transfected by *miR-29b* mimics (50 nM), inhibitors (100 nM) or their corresponding control vehicles according to manufacturer's introduction. For qRT-PCR and western blot analysis of Wnt family member 5A (WNT5A), receptor activator of nuclear factor kappa B ligand (RANKL), stromal cell derived factor-1 (SDF-1), the cells were harvest after 30min of mimics or inhibitors treatment. For ALP assay and qRT-PCR analysis of osteogenic genes, BMSCs were harvest after 7

days of mimics or inhibitors treatment. For Alizarin Red staining, BMSCs were harvest after 21 days of mimics or inhibitors treatment. Osteogenesis induction medium with mimics, inhibitors or their corresponding controls were replenished every 3 days.

Osteogenic Mineralization and Differentiation Assay

Osteoblastic differentiation induction of BMSCs was achieved using culture in 24-well plates (5×10^5 cells per well) in osteogenesis induction medium (5 mM β-glycerol phosphate, 50 μg/ml ascorbic acid, and 300 ng/ml BMP-2). A commercial kit (Nanjing Jiancheng Bioengineering Ltd., Nanjing, China) was used to measure the ALP activity according to the manufacturer's protocol. BMSCs were stained with alizarin red after culture in 6-well plates (2.5×10^6 cells per well) in osteogenesis induction medium for 21 days. Then, Alizarin Red S (Sigma-Aldrich, St. Louis, MO, USA) at 2% and pH 4.2 was used to stain cells to evaluate cell matrix mineralization.

Luciferase Reporter Assay

The luminescence emitted by the dual luciferase reporter assay system (Promega Corp., Madison, WI, USA) was quantified using a luminometer (Glomax; Promega Corp.). The renilla luciferase value from the cotransfected phRL-null vector (Promega Corp.) was used to normalize each value from the firefly luciferase assay. BMSCS were cultured in 96-well plate and then, using Lipofectamine 2000 (Invitrogen, Waltham, MA, USA), they were co-transfected with 2 ng of pRL-TK (Promega), 50 ng of a firefly luciferase reporter including the 3′ UTR of *Hmga2*, and 150 ng of either empty vector or *miR-29b*. The non-target 3′ UTR of *Hmga2* served as the negative control. A Luciferase assay kit (Promega) was used on cells harvested 48 h later. The transfection protocol was performed in triplicate.

Quantitative Real-time Polymerase Chain Reaction (qRT-PCR)

In each subgroup, every 2 condyle subchondral bone or every 3 condylar cartilage were respectively pooled together as a single sample for qRT-PCR. Therefore, the qRT-PCR sample number of condyle subchondral bone was 5 (from condyles of 10 mice), and that of condylar cartilage was 3 (from condyles of 9 mice). The primer sequences of the genes used in this study were listed in Supplementary

Table 1, and the mRNA level of those genes were determined by qRT-PCR following a previously described protocol [Sun et al. 2020]. Briefly, TRIzol reagent (Thermo Fisher Scientific, Waltham, MA, USA) was used to extract total RNA, which was then reverse transcribed using a PrimeScript RT reagent kit (Takara Bio Inc., Shiga, Japan). A 7500 Real-Time PCR system (Thermo Fisher Scientific) was used to perform the PCR reactions. The internal control comprised *Gapdh* (encoding glyceraldehyde 3-phosphate dehydrogenase). The $2^{-\Delta\Delta CT}$ method was used to calculate the expression of a target gene relative to that of *Gapdh*. For each sample, qRT-PCR was performed in 3 replicates and the mean value of cycle thresholds (Ct) was calculated and used for the $2^{-\Delta\Delta CT}$ calculating method. For the $2^{-\Delta\Delta CT}$ calculating method, Ct values of experimental groups with their corresponding control in the same batch were tested, and the results were calculated as the relative quantification compared to the control group, which was set at 1. Hence, all values of control samples were 1 and their SD values were all 0.

Western Blotting

The protein levels of WNT5A, RANKL and SDF-1 were assessed using western blotting, as previously indicated [Sun et al. 2017]. Briefly, total extracted proteins were treated using loading buffer and then subjected to polyacrylamide gel electrophoresis (5% to 15% gels). The separated proteins were transferred onto a polyvinylidene difluoride (PVDF) membrane (Millipore-Sigma). Then, 5% bovine serum albumin was used to block the membrane for 1 h at room temperature. After washing, the membrane was incubated with anti-WNT5A (ab235966, Abcam, Cambridge, United Kingdom), anti-SDF1 (ab9797, Abcam), or anti-RANKL (ab45039, Abcam) primary antibodies at 4 °C overnight. The membrane was then incubated with horseradish peroxidase-conjugated secondary antibodies (Santa Cruz Biotechnology) at room temperature for 4 h. To reveal the signals from the immunoreactive protein bands, enhanced chemiluminescence detection (GE Healthcare, Piscataway, NJ, USA) was used. A densitometer (Syngene Bioimaging System; Frederick, MD, USA) was used to scan the membranes. Image Lab software

(version 4.1, Bio-Rad Laboratories, Inc., Hercules, CA, USA) was used to quantify the obtained scans.

Statistical Analysis

Two independent, blinded observers quantified the parameters of the micro-CT data and images. To verify the agreement level between the two observers, the intraclass correlation coefficient (ICC) was employed (ICC value = 0.907). Subsequent analyses used the average values recorded by them.

All derived parameter data are shown as the mean ± standard deviation (SD) derived from the independent values. The Shapiro-Wilk test was used to test for a normal distribution (95% confidence interval). Levene' test was used to assess homogeneity of variance. The Kruksal-Wallis test or one-way analysis of variance (ANOVA) were used to evaluate the assumptions of the parametric test were fulfilled and the differences among groups. Significance was accepted at $P < 0.05$ for all tests.

References

Jiao K, Zhang M, Niu L, Yu S, Zhen G, Xian L, Yu B, Yang K, Liu P, Cao X, et al. 2014. Overexpressed TGF-β in subchondral bone leads to mandibular condyle degradation. J Dent Res. 93(2): 140-147.

Jiao K, Zen G, Niu LN, Yang HX, Ren G, Xu XY, Li FF, Tay F, Wang MQ. 2016. Activation of α2A-adrenergic signal transduction in chondrocytes promotes degenerative remodelling of temporomandibular joint. Sci Rep. 6: 30085.

Li CJ, Cheng P, Liang MK, Chen YS, Lu Q, Wang JY, Xia ZY, Zhou HD, Cao X, Xie H, et al. 2015. MicroRNA-188 regulates age-related switch between osteoblast and adipocyte differentiation. J Clin Invest. 125(4): 1509-1522.

Liu S, Liu D, Chen C, Hamamura K, Moshaverinia A, Yang R, Liu Y, Jin Y, Shi S. 2015. MSC Transplantation Improves Osteopenia via Epigenetic Regulation of Notch Signaling in Lupus. Cell Metab. 22(4):606-618.

Sun JL, Jiao K, Niu LN, Jiao Y, Song Q, Shen LJ, Tay FR, Chen JH. 2017. Intrafibrillar silicified collagen scaffold modulates monocyte to promote cell homing, angiogenesis and bone regeneration. Biomaterials. 113: 203-216.

Sun JL, Yan JF, Li J, Wang WR, Yu SB, Zhang HY, Huang F, Niu LN, Jiao K. 2020. Conditional deletion of Adrb2 in mesenchymal stem cells attenuates osteoarthritis-like defects in temporomandibular joint. Bone. 133: 115229.

Yang T, Zhang J, Cao Y, Zhang M, Jing L, Jiao K, Yu S, Chang W, Chen D, Wang M. 2015. Wnt5a/Ror2 mediates temporomandibular joint subchondral bone remodeling. J Dent Res. 94(6): 803-812.

Supplementary Infromation

Supplementary Table 1. Primer sequences used for RT-PCR.

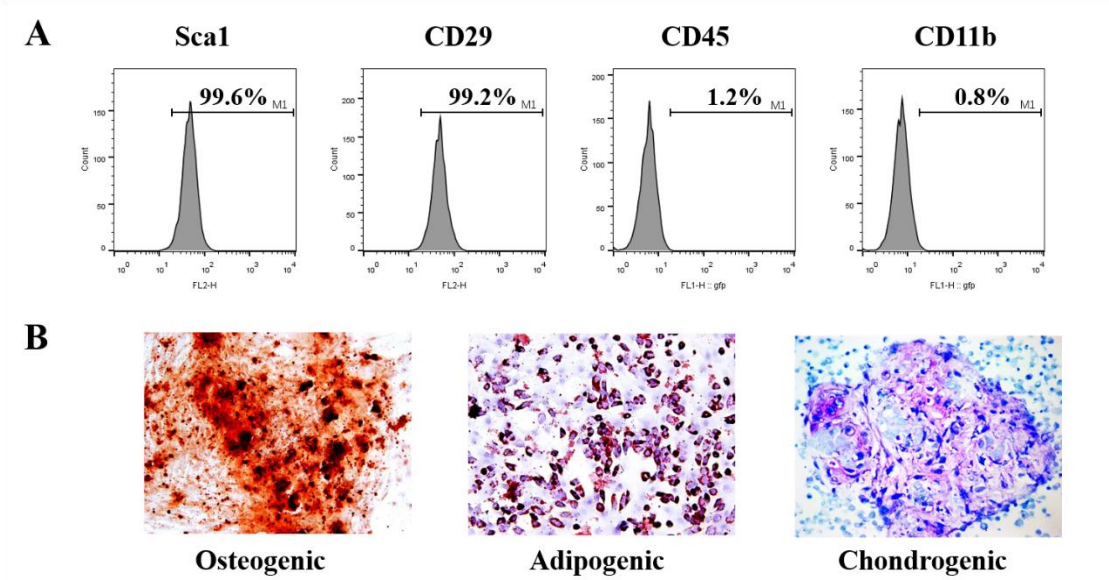
Primer	Aliase	Sequences
m-pri-miR29b-F	/	5′-GAGGTAGCACCATTTGAAATCAGTGT-3′
m-pri-miR29b-R	/	5′-GGAGCTGGTTTCATATGGTGGTTT-3′
m-mir29b-F	/	5’-CTAGCTAGCCAGTGCTGCAATGATACCGC -3’
m-mir29b-R	/	5’-CCGGAATTCGGGAACCGGAGCTGAATGAA -3’
m-miR29a-F	/	5′-CGCGGATCCTGGATTTAGTAAGATTTGGGC-3′
m-miR29a-R	/	5′-CCGGAATTCACATGCAATTCAGGTCAGTG-3′
m-miR29c-F	/	5'-ACACTCCAGCTGGGTAGCACCATTTGA-3'
m-miR29c-R	/	5'-TGGTGTCGTGGAGTCG-3'
m-Acp5-F	TRAP	5'-GTCTCTGGGGGACAATTTCTACTR-3'
m-Acp5-R	TRAP	5'-GTTTGTACGTGGAATTTTGAAGC-3'
m-ctsk-F	/	5'-AAGTTTCTGCTGCTACCCA-3'
m-ctsk-R	/	5'-CAGAGATTTGCTTCAGGTT-3'
m-Bglap-F	OCN	5'-CTGCTCACTCTGCTGACC-3'
m-Bglap-R	OCN	5'-GGACTGAGGCTCCAAGGT-3'
m-runx2-F	/	5'-TCCGAAATGCCTCTGCTGTT-3'
m-runx2-R	/	5'-GCCACTTGGGGAGGATTTGT-3'
m-Acan-F	aggrecan	5'-TTCCACCAGTGCGATGCAG-3'
m-Acan-R	aggrecan	5'-TGGTGTCCCGGATTCCGTA-3'
m-col2a1-F	Col2	5'-CATCCAGGGCTCCAATGATGTA-3'
m-col2a1-R	Col2	5'-ATGTCCATGGGTGCGATGTC-3'
m-Mmp3-F	/	5'-CTCATGCCTATGCACCTGGAC-3'
m-Mmp3-R	/	5'-TCATGAGCAGCAACCAGGAA-3'
m-Mmp13-F	/	5'-TCCCTGGAATTGGCAACAAAG-3'
m-Mmp13-R	/	5'-GCATGACTCTCACAATGCGATTAC-3'

m-Wnt5a-F	/	5'-TAAGCCCGGGAGTGGCTTTGG-3'
m-Wnt5a-R	/	5'-GGGCGAAGGAGAAAAACGTGG-3'
m-Tnfrsf11b-F	Opg	5'-CCCCTCATTAACCAGCACAA-3'
m-Tnfrsf11b-R	Opg	5'-TTCCCAAACCGGTCCTCT-3'
m-Tnfsf11-F	RANKL	5`-ACGCAGATTTGCAGGACTCGAC-3`
m-Tnfsf11-R	RANKL	5`-TTCGTGCTCCCTCCTTTCATC-3`
m-Cxcl12-F	SDF-1	5`-GAGAGCCACATCGCCAGAG-3`
m-Cxcl12-R	SDF-1	5`-TTTCGGGTCAATGCACACTTG-3`
m-Csf1-F	M-CSF	5′-GCCATC AAAGAAGCCCTGAA-3′
m-Csf1-R	M-CSF	5′-GCGGGTCTGCACACATGTTA-3′
m-il6-F	/	5′-ACAACCACGGCCTTCCCTACTT-3′
m-il6-R	/	5′-AGCCTCCGACTTGTGAAGTGGT-3′
m-Col1a1-F	Col1	5′-GTTGTGCGATGACGTGATCTGT-3′
m-Col1a1-R	Col1	5′-TTGGTCGGTGGGTGACTCTG-3′
m-tnfa-F	TNF-α	5′-GAAAGTCAGCCTCCTCTCCG-3′
m-tnfa-R	TNF-α	5′-CTCCAAAGTAGACCTGCCCG-3′
m-Sp7-F	OSX	5′-CAAGCACCAATGGACTCCTCTC-3′
m-Sp7-R	OSX	5′-TAGACACTAGGCAGGCAGTCAG-3′
m-GAPDH-F	/	5`-TCA ACG GCA CAG TCA AGG-3`
m-GAPDH-R	/	5`-GAT GTT AGT GGG GTC TCG C-3`

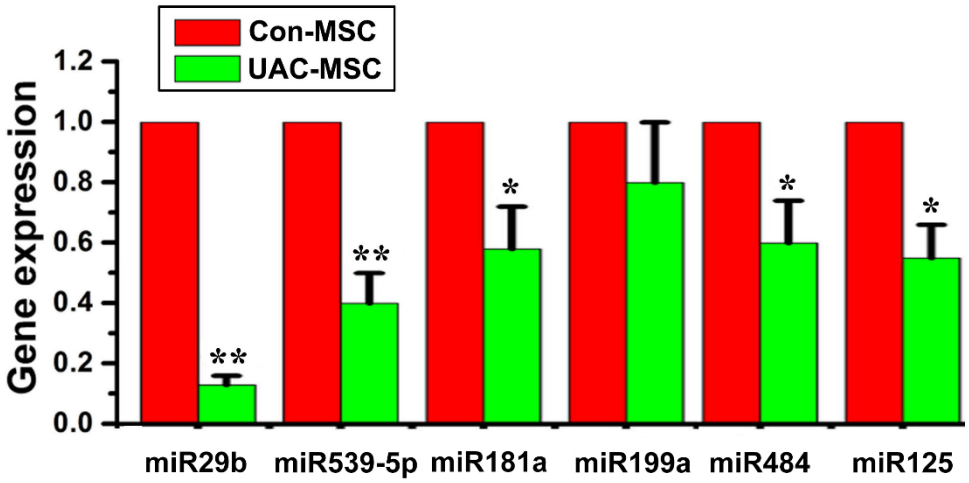
Acp5: acid phosphatase 5; Bglap: bone gamma carboxyglutamate protein; Tnfrsf11b: tumor necrosis factor receptor superfamily, member 11b; Tnfsf11: tumor necrosis factor (ligand) superfamily, member 11; Cxcl12: chemokine (C-X-C motif) ligand 12; Csf1: colony stimulating factor 1; Sp7: transcription factor 7.

Supplementary Table 2. Differentially expressed miRNAs with greater than 3-fold expression level changes.

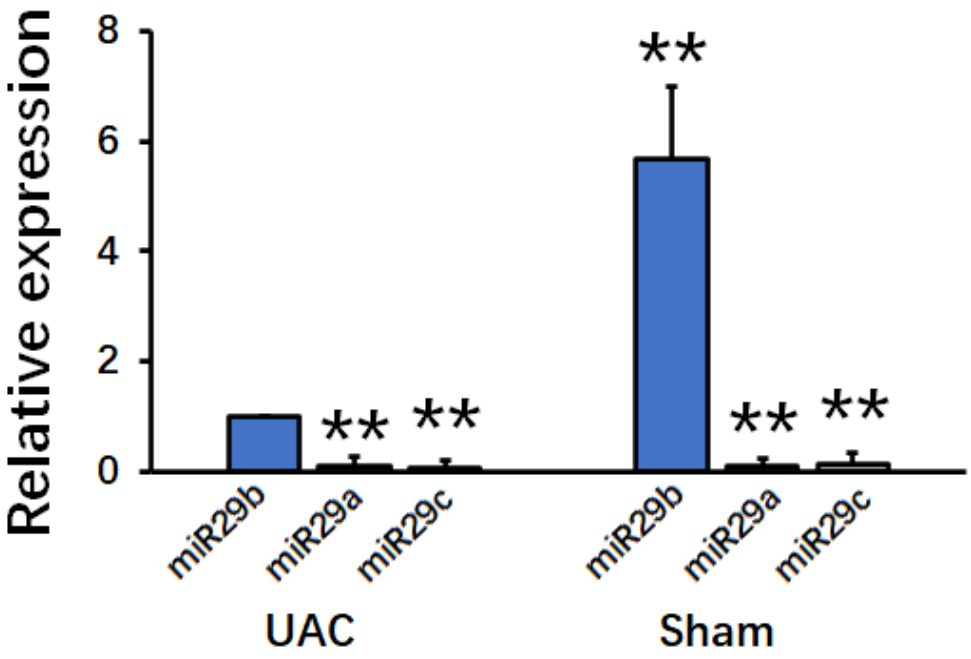
ProbeName	Fold changes	Changes	Symbol
ASMM10P049298	8.144692	up	*mmu-miR103a-2*
ASMM10P018826	5.654705	up	*mmu-miR130b*
ASMM10P050085	5.341612	up	*mmu-miR21*
ASMM10P001999	4.584977	up	*mmu-miR27b*
ASMM10P055285	3.777533	up	*mmu-miR-628-3p*
ASMM10P007608	3.682369	up	*mmu-miR-410*
ASMM10P017925	3.615134	up	*mmu-miR-134*
ASMM10P016228	3.653598	up	*mmu-miR-110b*
ASMM10P019394	3.313618	up	*mmu-miR-220a*
ASMM10P049749	3.326106	up	*mmu-miR-181a-sp*
ASMM10P015859	14.70075	down	*mmu-miR29b*
ASMM10P024182	8.170392	down	*mmu-miR484*
ASMM10P027238	5.99889	down	*mmu-miR125b*
ASMM10P052843	5.535187	down	*mmu-miR181a*
ASMM10P029598	5.405979	down	*mmu-miR199a*
ASMM10P024800	4.941271	down	*mmu-miR193b*
ASMM10P053951	4.530032	down	*mmu-miR197*
ASMM10P051721	4.343417	down	*mmu-miR24*
ASMM10P023210	4.167521	down	*mmu-miR335*
ASMM10P036062	3.519515	down	*mmu-miR370a*
ASMM10P054340	3.350355	down	*mmu-miR202b*
ASMM10P047295	3.332322	down	*mmu-miR134b*
ASMM10P052641	3.481084	down	*mmu-miR433a*
ASMM10P033417	3.77708	down	*mmu-miR539-5p*



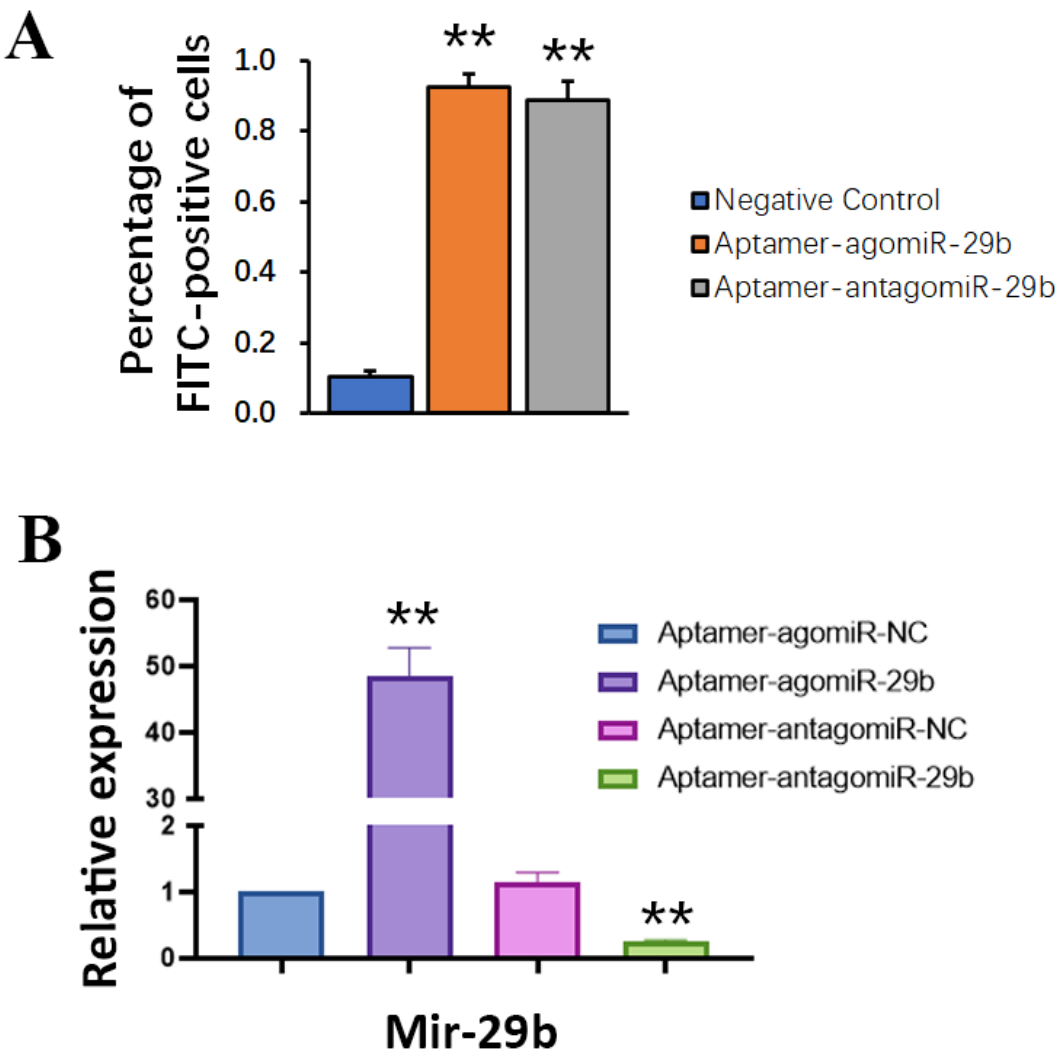
Supplementary Figure 1. (A) Flow cytometry of BMSCs identification. Expression of Sca1, CD29, CD45 and CD11b were examined. (B) Multi-lineage differentiation capacities of BMSCs.



Supplementary Figure 2. Results of RT-PCR analysis confirming the significant down-regulated miRNAs between BMSCs from the sham and UAC mice. (*P < 0.05, ** P < 0.01).



Supplementary Figure 3. Results of RT-PCR analysis evaluating expression of *miR-29b*, *miR-29a* and *miR-29c* in BMSCs from UAC and sham control mice. ** P < 0.01.



Supplementary Figure 4. (A) Flow-cytometry analysis evaluating the percentages of agomiR and antagomiR introduced via the aptamer constructs. The aptamers were labeled by FITC, and the BMSCs were treated by negative control, aptamer-agomiR-29b and aptamer-antagomiR-29b for 60 min. (B) RT-PCR result of *miR-29* expression by BMSC treated by aptamer-agomiR-29b, aptamer-antagomiR-29b or their corresponding controls for 60 min. ** $P < 0.01$.