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Lnc-RNA BLACAT1 regulates differentiation of bone marrow stromal stem cells by targeting miR-142-5p in osteoarthritis

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Abstract. – OBJECTIVE: Osteogenic differentiation of bone marrow stromal stem cells (BMSCs) is beneficial to the treatment of osteoarthritis (OA). Lnc-RNA BLACAT1 involves in occurrence and development of various diseases. However, the role of Lnc-RNA BLACAT1 in BMSCs differentiation under inflammation remains unclear.

MATERIALS AND METHODS: Rat BMSCs were isolated and randomly divided into control group and inflammation group (addition of IL-6). The inflammation group was further divided into BLACAT1 siRNA group and BLACAT1 siRNA+miR-142-5p inhibitor group, followed by analysis of Lnc-RNA BLACAT1 expression by real time PCR, BMSCs proliferation, Caspase 3 activity, ALP activity, expression of Runx2, OC and PPARγ2 by real time PCR, and secretion of TNF-α and IL-1β by enzyme-linked immunosorbent assay (ELISA). The bioinformatics software and the Luciferase reporter system analyze the targeted relationship between BLACAT1 and miR-142-5p.

RESULTS: In inflammation group, Lnc-BLACAT1 expression was increased, along with inhibited BMSCs proliferation, increased Caspase 3 activity, decreased ALP activity, and expression of Runx2 and OC, increased PPARγ2 expression and secretion of TNF-α and IL-1β. The difference was statistically significant compared with control group ($p<0.05$). MiR-142-5p is the target miRNA of Lnc-RNA BLACAT1. BLACAT1 siRNA down-regulated BLACAT1 expression, promoted cell proliferation, inhibited Caspase 3 activity, increased ALP activity and Runx2 and OC expression, decreased PPARγ2 expression and TNF-α and IL-1β secretion. Compared with inflammation group, the difference was statistically significant ($p<0.05$). Of note, BLACAT1 siRNA+miR-142-5p inhibitor group reversed the effect of siRNA-mediated knockdown of BLACAT1.

CONCLUSIONS: Lnc-RNA BLACAT1 expression was increased in inflammatory BMSCs, and knockdown of BLACAT1 promoted proliferation and osteogenic differentiation of BMSCs targeting miR-142-5p.

Key Words:
Inflammation, BMSCs, BLACAT1, MiR-142-5p, Proliferation, Osteogenic differentiation.

Introduction

Osteoarthritis (OA) is a multifactorial disease that causes articular cartilage to degenerate and affect the components of related joints[1]. Osteoarthritis is a musculoskeletal disorder that is usually occult, progressive, and slow. It usually affects the hand, spine, hip, and knee joints, impairing the working ability and daily activities of these patients[2,3]. OA is the most common joint disease affecting 6% to 12% of adults, and more than one-third of older people can develop osteoarthritis[4-6]. Cartilage damage is a major feature of common joint diseases of osteoarthritis. Evidence to date suggests that cartilage damage remains a significant challenge in clinical care[7,8]. Cartilage is maintained by chondrocytes that secrete extracellular matrix (ECM) components, such as collagen and aggrecan[9]. Among them, knee OA progression is the most common cause of total joint replacement, which imposes a heavy burden on patients and social medical expenses[10]. Age, hormones, obesity, inflammatory factors, signaling molecules, etc. can lead to the development of OA[11].

Bone marrow mesenchymal stem cells (BMSCs), which are stem cells derived from mesoderm in early development, have high renewing ability and multi-lineage differentiation potential, and can differentiate into cells of other lineages, including myoblasts, chondrocytes, as well as fat cells. It is considered to be an ideal source of cells for tissue damage[12,13]. BMSCs are the main source of cells for the body to increase and maintain bone mass and maintain bone health[14]. Koch et al[15] showed that BMSCs can provide intrinsic

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osteogenesis and thus supplement a large amount of bone loss. Long-chain non-coding RNAs (lncRNAs) are non-coding RNAs that are more than 200 nucleotides in length. Xu et al[16] have shown that lncRNAs have very limited protein-coding potential and can regulate disease by regulating the function of competitive endogenous RNA. LncRNA BLACAT1 is a recently discovered lncRNA that plays a role in a variety of diseases including tumors, metabolic diseases, cardiovascular diseases, etc. It has also been shown to be involved in the development and progression of various body physiology, pathological activities including inflammation and immune diseases[17,18]. However, the expression and related role of LncRNA BLACAT1 in BMSCs in OA has not been elucidated.

Materials and Methods

Experimental Animals

Five healthy female Sprague-Dawley rats, 2 months old, SPF grade, body weight (250±20) g, were purchased from the Experimental Animal Center of the Unit and fed by the SPF animal experiment center. Feeding conditions include maintaining the temperature at (21±1)°C and maintaining relative humidity (50-70%) under constant temperature and constant humidity conditions, ensuring a 12/day cycle every 12 h. Animal experiments were carried out in strict accordance with the experimental design and performed by experienced technicians to minimize animal suffering and meet animal welfare experiment requirements. This investigation was approved by the Ethics Committee of Yantaishan Hospital (Yantai, Shandong, China).

Reagents and Instruments

Sodium pentobarbital was purchased from Shanghai Zhaohui Pharmaceutical Co., Ltd. LncRNA BLACAT1 siRNA and miR-142-5p mimics and inhibitor were purchased from Shanghai Gene Pharmaceutical Co., Ltd. for design and synthesis. Fetal bovine serum (FBS) and cyan chain double antibody were purchased from Hyclone Corporation (San Angelo, TX, USA). Dimethyl sulfoxide (DMSO), MTT powder was purchased from Gibco (Grand Island, NY, USA); trypsin- ethylenediaminetetraacetic (EDTA) acid digest was purchased from Sigma-Aldrich (St. Louis, MO, USA). The lipo2000 transfection reagent was purchased from Invitrogen (Carlsbad, CA, USA). TNF-α and IL-1β1 enzyme-linked immunosorbent assay (ELISA) kits were purchased from R&D Systems (Minneapolis, MN, USA). Western blot related chemical reagents were purchased from Shanghai Biyuntian Biotechnology Co., Ltd. (Shanghai, China), enhanced chemiluminescence (ECL) reagents were purchased from Amersham Biosciences (Little Chalfont, Buckinghamshire, UK), rabbit anti-mouse NF-κB monoclonal antibody, and goat anti-rabbit horseradish peroxidase (HRP)-labeled IgG secondary antibody was purchased from the Cell Signaling company (Danvers, MA, USA). The Caspase 3 activity assay kit was purchased from Wuhan Dr. Bio-Limited (Bio-Rad, Hercules, CA, USA). The RNA extraction kit and the reverse transcription kit were purchased from Axygen Scientific (Union City, CA, USA). Other commonly used reagents were purchased from Shanghai Shenggong Biological Co., Ltd (Shanghai, China). The pmirGLO vector was purchased from Promega (Madison, WI, USA). The ALP activity detection kit was purchased from Wuhan Boster Bio Co., Ltd. The ABI 7700 Fast Quantitative PCR Reactor was purchased from ABI (Port Washington, NY, USA). The Labsystem Version 1.3.1 microplate reader was purchased from Bio-Rad Corporation (Hercules, CA, USA). The clean workbench was purchased from Suzhou Purification Equipment Factory (Suzhou, Jiangsu, China). Surgical microscopy equipment was purchased from Suzhou Medical Instrument Factory. The Hera cell CO_2 incubator was purchased from Thermo Fisher Scientific (Port Washington, NY, USA).

Isolation and Culture of Rat BMSCs

The rats were sacrificed by cervical dislocation and immersed in 70% alcohol for 20-30 min. The skin was cut layer by layer to expose the femur and tibia which was separated and placed into a sterile plate, in which 3 ml of phosphate-buffered saline (PBS) was added to make a cell suspension followed by centrifugation at 1000 rpm for 10 min and removal of the supernatant and fat. Then, equal volume of lymphocyte separation solution was added and centrifuged at 25 rpm for 25 min at room temperature followed by addition of 5 ml of a suspension containing bone marrow mesenchymal cells into the mononuclear cell separation solution and centrifugation at room temperature for 20 min at 2000 rpm to isolate the BMSCs. BMSCs were then cultured in a medium containing 5 ml of α-MEM containing 10% FBS, 1% double anti-

body in a 37°C incubator with 5% CO_2. After 48 h, the medium was changed, the non-adherent cells were removed, the culture was continued, and passage was performed until the cell colonies reached 80% confluence. The 3-5 generation BMSCs cells were taken for testing.

BMSCs Grouping and Processing

The BMSCs of the 3-5th generation logarithmic growth phase were randomly divided into 4 groups, control group (normal cultured cells); and inflammation group (2 μM IL-6 was added to culture BMSCs). The inflammation group was further divided into BLACAT1 siRNA group and BLACAT1 siRNA+miR-142-5p inbibitor group, which was transfected with BLACAT1 siRNA and BLACAT1 siRNA+miR-142-5p inhibitor in IL-6-treated cells, respectively. The BLACAT1 siRNA sequence was 5'-GCCAGUUACUCGUATAUAT-3'. MiR-142-5p inbibitor was 5'-AUGGCCGAGACGUGAUAUG-3'. The cell density was fused to 70-80% in a 6-well plate; BLACAT1 siRNA and miR-142-5p inhibitor liposomes were separately added to 200 μl of serum-free medium, mixed well, and incubated at room temperature for 15 min. The mixed lipo2000 was separately mixed and incubated for 30 min at room temperature. The serum of the cells was removed, PBS was gently rinsed, 1.6 ml of serum-free medium was added, and each system was added to each system, and cultured in a 5% CO_2 incubator at 37°C for 6 h. The serum culture solution was replaced and cultured for 48 h for experimental research.

MTT Assay for Analysis of Proliferation of Inflammatory BMSCs

BMSCs from each group of bone inflammation in the logarithmic growth phase were collected and inoculated into the 96-well culture plate with 10% fetal bovine serum DMEM/F12 medium in the number of 5×10^3 cells. After 24 h of culture, the supernatant was discarded and randomly divided into groups mentioned above. After 48 of cell treatment in each group, 20 μl of sterile MTT was added to the wells to be tested, and 3 replicate wells were set in each treatment group. After 4 hours of continuous culture, the supernatant was completely removed, 150 μl/well of dimethyl sulfoxide (DMSO) was added, and the shaker was shaken 10 min, after the purple crystals were fully dissolved, the absorbance (A) value was measured at a wavelength of 570 nm by a microplate reader, and the proliferation rate of each group was calculated. The experiment was repeated three more times.

ELISA Detection of TNF-α and IL-1β Secretion

All the samples were tested for the expression of TNF-α and IL-1β in the supernatant of each group by enzyme-linked immunosorbent assay (ELISA) kit according to the ELISA kit instructions. The main operational steps included: taking out a 96-well plate and adding 50 μl of the diluted standard in the corresponding reaction well to prepare a standard curve. Add 50 μL of the sample to be tested to the reaction well. Make three duplicate holes for each sample. According to the kit instructions, the blank value was zeroed, and the absorbance value (A value) of each well was measured by a microplate reader at a wavelength of 450 nm. The linear regression equation of the standard curve was calculated according to the concentration of the standard product and the corresponding A value, and the corresponding sample concentration was calculated on the regression equation according to the OD value of the sample.

Caspase 3 Activity Detection

The changes in Caspase 3 activity in each group of cells were examined according to the kit instructions. Trypsin digested cells were centrifuged at 600 g at 4°C for 5 min and cell lysate was lysed on ice for 15 min, followed by centrifugation at 20000 g at 4°C for 5 min. Then, 2 mM Ac-DEVD-pNA was added and OD value at 405nm was measured to calculate Caspase 3 activity.

Real time PCR detection of the expression of BLACAT1, miR-142-5p, Runx2, OC and PPARγ2 in BMSCs

RNA from each group of rat BMSCs was extracted from ice by TRIzol reagent, and DNA reverse transcription synthesis was performed according to the kit instructions. The primers were designed according to each gene sequence by Primer 6.0 and synthesized by Shanghai Yingjun Biotechnology Co., Ltd. (Table I). Real-time PCR reaction conditions: 55°C 1 min, 92°C 30 s, 58-60°C 45 s, 72°C 35 s, a total of 35 cycles. U6 was used as a reference. According to the fluorescence quantification, the starting cycle number (CT) of all samples and standards was calculated. Based on the standard CT value, a standard curve was drawn and then the semi-quantitative analysis was carried out by the 2-ΔCt method.

Table I. Primer sequences.

Gene	Forward 5′-3′	Reverse 5′-3′
U6	TGCTTCGGCAGCACATATAC	AGGGGCCATGCTAATCTTCT
OC	TCCAGGCTCCTGGCTC GA	ACACTGAAGTTCGATTCGAG
miR-142-5p	CAGGGGGATCAAA	GATTCAAAAGTCTCAAA
BLACAT1	CATGCGAATGGGAGGA	GCCTTCAAA CGTGGTA
PPARγ2	GGTGGGCTCAAATTC	CTTG CTCTCGTTTCGCTTA
Runx2	CTGGCCAGTTCGCTCC GA	ACTGAAATTCGTTACCGGAG

Determination of ALP Content

The ALP content was determined according to the instructions of the ALP test kit, centrifuged at 1000 rpm for 10 min, the supernatant was discarded, and the precipitate was collected by adding Triton-X100. After mixing, various OD values were measured at 520 nm, and the ALP content was calculated.

Dual-Luciferase Reporter Gene Assay

The predicted BLACAT1 sequence was inserted into the pmirGLO vector, and the reporter and mir-142-5p mimics were co-transfected into BMSCs for 48 h. BLACAT1MT and MUT reporters were constructed, Luciferase activity was determined using Dual-Luciferase assay kit.

Statistical Analysis

All data were expressed as mean ± standard deviation (SD). The mean values of the two groups were compared by *t*-test test, analyzed by SPSS 11.5 (Chicago, IL, USA) statistical software, and the differences between groups were analyzed by ANOVA test by Tukey HSD. $p<0.05$ indicated a statistical difference.

Results

Expression of BLACAT1 In Inflammatory BMSCs

Real time PCR analysis analyzed the expression of BLACAT1 in inflammatory BMSCs and showed that the expression of BLACAT1 was increased in BMSCs cells in inflammatory environment. The difference was statistically significant compared with control group ($p<0.05$). BLACAT1 siRNA transfection into inflammatory BMSCs down-regulated the expression of BLACAT1, compared with the inflammation group, the difference was statistically significant ($p<0.05$; Figure 1).

Targeting MiRNA Analysis of BLACAT1 In BMSCs

Dual-Luciferase reporter assay was performed to analyze BLACAT1 targeting miRNA in BMSCs, establishing BLACAT1 wild type (WT) and variant (Mut), respectively, and the results showed that targeted report analysis predicted that miR-142-5p was a BLACAT1 target gene and transfection of mir-142-5p mimics inhibited BLACAT1 Luciferase activity (Figure 2).

Effect of BLACAT1 MiR-142-5p Expression In Inflammatory BMSCs

Real time PCR analyzed the effect of BLACAT1 on the expression of miR-142-5p in inflammatory BMSCs and showed that the expression of miR-142-5p was decreased in BMSCs cells in inflammatory environment. Compared with the control group, the difference was statistically significant ($p<0.05$). BLACAT1 siRNA transfection

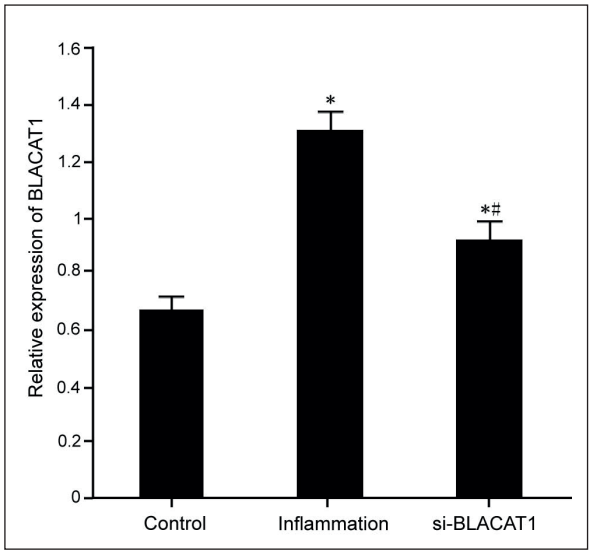

Figure 1. Expression of BLACAT1 in inflammatory BMSCs. Compared with the control group, $*p<0.05$; compared with the inflammation group, $^{\#}p<0.05$.

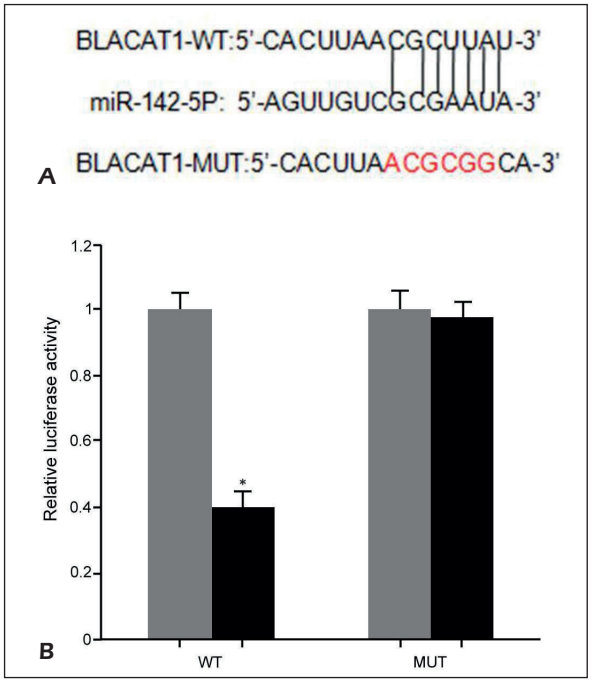

Figure 2. Targeting miRNA analysis of BLACAT1 in BMSCs. **A**, BLACAT1 targeted predictive analysis in BMSCs; **A**, luciferase report statistical analysis, compared with NC group, *p<0.05.

into inflammatory BMSCs can down-regulate the expression of BLACAT1 and promote the expression of miR-142-5p. Compared with the inflammation group, the difference was statistically significant (p<0.05; Figure 3).

Effect of BLACAT1 on Cell Proliferation of Inflammatory BMSCs

BMSCs proliferation was inhibited in the inflammatory environment, and the difference was statistically significant compared with control group (p<0.05). Transfect BLACAT1 siRNA into inflammatory BMSCs can down-regulate the expression of BLACAT1 and promote cell proliferation. Compared with the inflammation group, the difference was statistically significant. However, after transfection of miR-142-5p inhibitor into BLACAT1 siRNA group, cell proliferation was inhibited, compared with the BLACAT1 siRNA group, the difference was statistically significant (p<0.05; Figure 4).

Effect of BLACAT1 on Caspase 3 Activity In Inflammatory BMSCs

The activity of Caspase 3 in BMSCs cells was increased in the inflammatory environment,

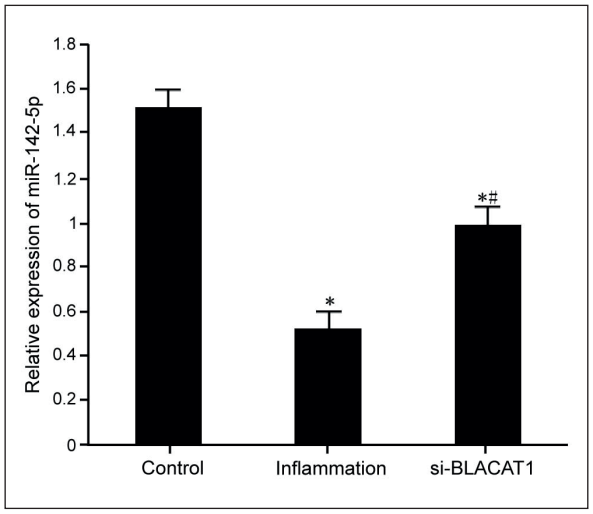

Figure 3. Analysis of expression of miR-142-5p in inflammatory BMSCs regulated by BLACAT1. Compared with the control group, *p<0.05; compared with the inflammation group, $^{\#}p$<0.05.

and the difference was statistically significant compared with control group (p<0.05). BLACAT1 siRNA transfection into inflammatory BMSCs could down-regulate the expression of BLACAT1 and decrease the activity of Caspase 3, and the difference was statistically significant compared with the inflammation group. After transfection of miR-142-5p inhibitor into BLACAT1 siRNA group, Caspase 3 activity was inhibited and the difference was statistically significant compared with the BLACAT1 siRNA group (p<0.05; Figure 5).

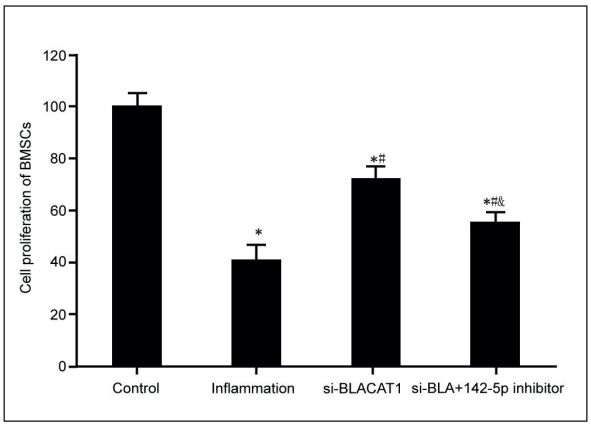

Figure 4. Effect of BLACAT1 on cell proliferation of inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, $^{\#}p$<0.05; compared with the BLACAT1 siRNA group, $^{\&}p$<0.05.

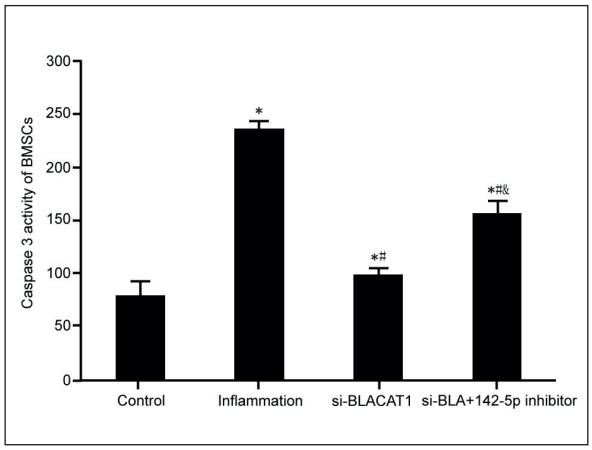

Figure 5. Effect of BLACAT1 on Caspase 3 Activity in Inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, #p<0.05; compared with the BLACAT1 siRNA group, &p<0.05.

Effect of BLACAT1 on ALP Activity In Inflammatory BMSCs

The ALP activity of BMSCs cells in the inflammatory environment was decreased, compared with the control group, the difference was statistically significant (p<0.05). BLACAT1 siRNA transfection into inflammatory BMSCs down-regulated BLACAT1 expression, and increased ALP activity, compared with the inflammation group, the difference was statistically significant; while transfection of miR-142-5p inhibitor in BLACAT1 siRNA group decreased ALP activity. The difference was statistically significant compared with the BLACAT1 siRNA group (p<0.05; Figure 6).

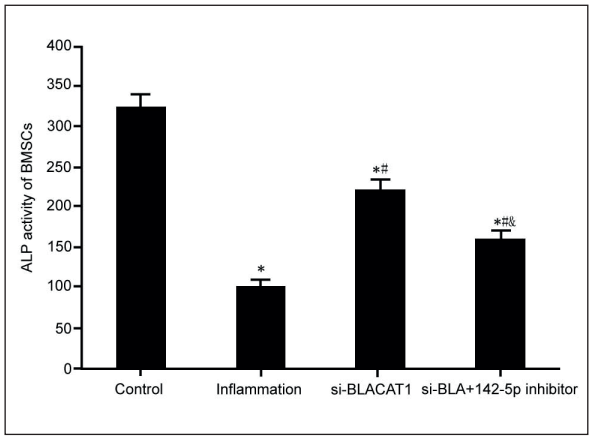

Figure 6. Effect of BLACAT1 on ALP activity in inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, #p<0.05; compared with the BLACAT1 siRNA group, &p<0.05.

Effect of BLACAT1 on Osteogenesis-Related Genes Expression In Inflammatory BMSCs

The osteogenic genes Runx2 and OC of BMSCs expressions were decreased in the inflammatory environment, and the difference was statistically significant compared with control group (p<0.05). BLACAT1 siRNA transfection into inflammatory BMSCs down-regulated BLACAT1 expression, increased Runx2 and OC expression, and the difference was statistically significant compared with the inflammation group; transfection of miR-142-5p inhibitor into BLACAT1 siRNA group decreased Runx2 and OC expression. The difference was statistically significant compared with the BLACAT1 siRNA group (p<0.05; Figure 7).

Effect of BLACAT1 on PPARγ2 Expression In Inflammatory BMSCs

The PPARγ2 expression was increased in the inflammatory environment, and the difference was statistically significant compared with control group (p<0.05). BLACAT1 siRNA transfection into inflammatory BMSCs down-regulated the expression of BLACAT1 and decreased PPARγ2 expression, compared with the inflammation group, the difference was statistically significant; while the transfection of miR-142-5p inhibitor in the BLACAT1 siRNA group increased PPARγ2 and BLACAT1 expression. The difference was statistically significant compared with the BLACAT1 siRNA group (p<0.05; Figure 8).

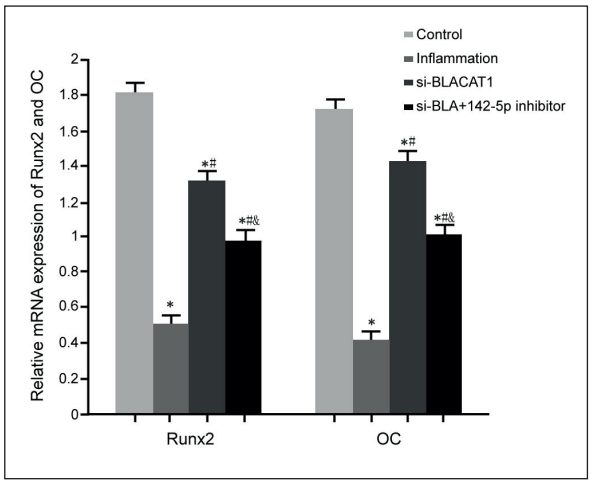

Figure 7. Effect of BLACAT1 on osteogenesis-related genes in inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, #p<0.05; compared with the BLACAT1 siRNA group, &p<0.05.

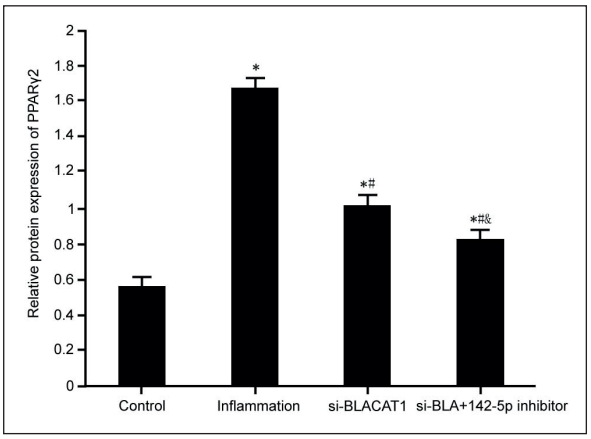

Figure 8. Effect of BLACAT1 on adipogenic differentiation genes of inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, $^{\#}p$<0.05; compared with the BLACAT1 siRNA group, $^{\&}$p<0.05.

Effect of BLACAT1 on the Secretion of Inflammatory Factors

The secretion of inflammatory factors TNF-α and IL-1β in BMSCs supernatant was increased in the inflammatory environment, and the difference was statistically significant compared with control group (p<0.05). BLACAT1 siRNA transfection into inflammatory BMSCs down-regulated TNF-α and IL-1β secretion, compared with the inflammation group, the difference was statistically significant; and transfection of miR-142-5p inhibitor in BLACAT1 siRNA group increased secretion of TNF-α and IL-1β secretion. The difference was statistically significant compared with the BLACAT1 siRNA group (p<0.05; Figure 9).

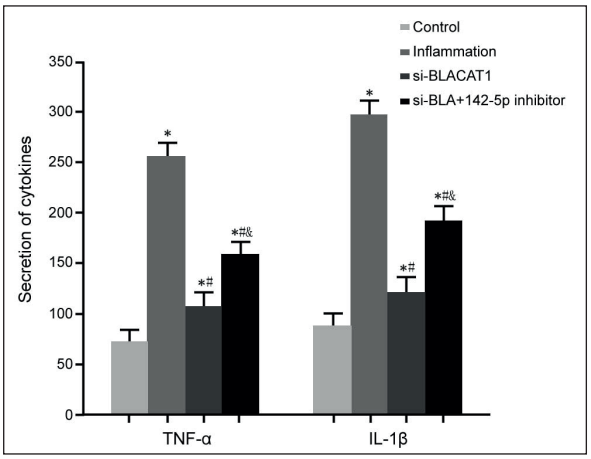

Figure 9. Effect of BLACAT1 on the secretion of inflammatory factors in the supernatant of inflammatory BMSCs. Compared with the control group, *p<0.05; compared with the inflammation group, $^{\#}p$<0.05; compared with the BLACAT1 siRNA group, $^{\&}p$<0.05.

Discussion

With the development of osteoarthritis, osteoarthritis pain is caused by bone deformation and joint effusion, rest can reduce pain, and moving the joint or aggravating the weight of the joint will increase the pain[19]. Pathological changes of osteoarthritis include chondrocyte number and metabolic disorder, abnormal extracellular matrix (ECM) structure, increased secretion of matrix metalloproteinase family, inflammatory reaction and remodeling of synovial and subchondral bone, resulting in deep chondrocyte apoptosis, irreversible injury, joint deformities and dysfunction, and severe OA can lead to disability and even death[20-22]. However, due to the limited proliferation of chondrocytes, finding effective targets to regulate the proliferation and apoptosis of articular chondrocytes is beneficial to the repair of osteoarthritis[23].

With the advancement of tissue engineering, BMSCs have been increasingly used as useful tools for bone tissue engineering because of their potential to differentiate into multiple lineages[24]. Due to various advantages, BMSC has been widely used as a seed cell in gene therapy, cell replacement therapy and tissue engineering research[25]. Therefore, to find a mechanism to promote BMSCs regulation of osteogenic differentiation is conducive to the repair of orthopedic diseases[26]. Therefore, the role of BMSCs in osteoarthritis has become one of the research hotspots. Lnc-RNA BLACAT1 is a newly discovered Lnc-RNAs, which has been shown to play an important role in tumor, immunity, inflammation and other diseases, and can participate in tumorigenesis, metastasis, etc., while Lnc-RNA BLACAT1 can participate in tissue and organogenesis as well as non-neoplastic diseases[17,18]. Therefore, our aim is to analyze the role of Lnc-RNA BLACAT1 in the survival and osteogenic differentiation of BMSCs in an inflammatory environment. Increased expression of BLACAT1 was found in inflammatory BMSCs. Further analysis of the regulation of BLACAT1 on the proliferation and apoptosis of inflammatory BMSCs showed that down-regulation of BLACAT1 expression promoted the proliferation of inflammatory BMSCs, decreased Caspase 3 activity and inflammatory factors TNF-α and IL-1β secretion. These results suggest that by regulating the expression of BLACAT1 in inflammatory BMSCs, it can effectively regulate the proliferation and apoptosis of inflammatory BMSCs. Further analysis confirmed that the expression of osteogenesis-related genes Runx2 and OC in BMSCs was decreased, and the expression of adipogenic-related genes

was increased. Transfection of BLACAT1 siRNA in BMSCs cells could down-regulates BLACAT1 expression and promotes the expression of Runx2, OC and inhibits the expression of adipogenic related genes. This result suggests that BLACAT1 can be involved in the osteogenic differentiation of inflammatory BMSCs. MiR-142-5p is one of the miRNAs and plays an important role in various diseases such as inflammation and tumor. Lnc-RNA can be regulated by miRNA targeting, which regulates the progression of diseases such as inflammation and immunity[27,28]. This research indicated that miR-142-5p is a targeted miRNA of BLACAT1, which down-regulates miR-142-5p expression in BMSCs in an inflammatory environment, and can down-regulate BLACAT1 expression by transfecting BLACAT1 siRNA into inflammatory BMSCs. The expression of miR-142-5p regulates cell proliferation and osteogenic differentiation of BMSCs and inhibits its differentiation into adipogenic direction. Transfection of miR-142-5p inhibitor reverses the effect of BLACAT1 siRNA, resulting in inhibited cell proliferation in inflammatory environment, increased apoptosis, and inhibited osteogenic differentiation, suggesting that Lnc-RNA BLACAT1 in the inflammatory BMSCs regulates the proliferation and osteogenic differentiation of BMSCs by targeting miR-142-5p.

Conclusions

The expression of Lnc-RNA BLACAT1 is increased in BMSCs of inflammatory group. BLACAT1 can promote the proliferation and osteogenic differentiation of BMSCs. Lnc-RNA BLACAT1 regulates the survival and differentiation of BMSCs in an inflammatory environment by targeting miR-142-5p.

Conflict of Interests

The Authors declare that they have no conflict of interests.

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