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LncRNA BLACAT1 regulates the viability, migration and invasion of oral squamous cell carcinoma cells by targeting miR-142-5p

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Abstract. – OBJECTIVE: Oral squamous cell carcinoma (OSCC) is one of the most common head and neck tumors with high incidence and mortality. Long noncoding RNA bladder cancer-associated transcript 1 (lncRNA BLACAT1) was involved in several cancers development. However, the roles of BLACAT1 in OSCC have not been investigated.

MATERIALS AND METHODS: The expressions of BLACAT1 and miR-142-5p in OSCC cells were measured by quantitative real-time polymerase chain reaction (qRT-PCR). Cell viability was evaluated by MTT assay. Cell migration and invasion were evaluated by transwell migration assay and transwell invasion assay, respectively. The protein levels of CyclinD1, p21, p27, MMP-2, MMP-9 and MMP-14 were detected by Western blot analysis. The interaction of BLACAT1 and miR-142-5p was verified by luciferase reporter assay.

RESULTS: The expression of BLACAT1 was increased and the expression of miR-142-5p was decreased in OSCC cells. The knockdown of BLACAT1 suppressed the viability, migration and invasion of OSCC cells. miR-142-5p was identified as a target of BLACAT1 and BLACAT1 overexpression suppressed miR-142-5p expression. Furthermore, overexpression of miR-142-5p showed similar effects on OSCC cells viability, migration and invasion with BLACAT1 knockdown, and inhibition of miR-142-5p restored the effects of BLACAT1 knockdown OSCC cells viability, migration and invasion.

CONCLUSIONS: LncRNA BLACAT1 knockdown suppressed the viability, migration and invasion of OSCC cells by sponging miR-142-5p, indicating that BLACAT1 might be a novel target for the treatment of OSCC.

Key Words:
OSCC, BLACAT1, MiR-142-5p, Viability, Migration, Invasion.

Abbreviations

OSCC: Oral squamous cell carcinoma, lncRNA BLACAT1: Long noncoding RNA bladder cancer associated transcript 1, HOTAIR: HOX antisense intergenic RNA, UCA1: LncRNA urothelial carcinoma-associated 1, TSCC: tongue squamous cell carcinoma.

Introduction

Oral squamous cell carcinoma (OSCC) is the most common oral malignancy with high incidence and mortality, and its five-year survival rate is only 50%[1,2]. The occurrence of OSCC involves multiple complex processes, and it has been reported that smoking and drinking are potential risk factors of high occurrence OSCC[3,4]. Although diagnosis and treatment strategies have been improved in the past few decades, the five-year survival rate of OSCC patients is still very low[5]. Therefore, it is imperative to search for new effective treatment methods for OSCC patients.

Long noncoding RNAs (lncRNAs), a class of noncoding RNAs with more than 200 nucleotides, play important roles in a series of biological processes[6-8]. Emerging evidence has demonstrated that the abnormal expression of lncRNAs participated in OSCC progression. For example, HOX antisense intergenic RNA (HOTAIR) was upregulated in OSCC and HOTAIR knockdown repressed OSCC cell proliferation, invasion and metastasis[9]. LncRNA urothelial carcinoma-associated 1 (UCA1) was notably increased in tongue squamous cell carcinoma (TSCC) tissues and UCA1 knockdown could inhibit OSCC cell proliferation and promoted apoptosis[10]. TUG1 was highly expressed in OSCC cells, the highly expressed of

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TUG1 could promote OSCC cell proliferation and invasion, promote tumor growth and metastasis *in vivo*[11]. However, the functions of bladder cancer associated transcript 1 (BLACAT1) on OSCC development have not been studied yet.

MicroRNAs (miRNAs) are a group of highly conserved noncoding RNAs with about 22 nucleotides[12]. miRNAs are involved in many biological processes, such as proliferation, apoptosis, differentiation and migration[13,14]. Literature has showed that the dysregulation of miRNAs was associated with OSCC development. For example, Wu et al[15] showed that miR-101 was significantly decreased in OSCC cells and suppressed OSCC cell proliferation, migration and invasion. Zhang et al[16] suggested that miR-148 was downregulated in OSCC cells and miR-148 mimic transfection led to significant repression of viability, migration and invasion of OSCC cells. Yu et al[17] showed that the expression of miR-9 was downregulated in OSCC cells and the overexpression of miR-9 suppressed tumor cell proliferation *in vivo*. However, it has not been investigated whether miR-142-5p participates in OSCC development.

In this study, we detected the expressions of BLACAT1 and miR-142-5p in OSCC cells. Further functions and mechanisms analysis were conducted to verify the functional roles of BLACAT1 and miR-142-5p and their synergistic effects in OSCC. This study contributed to our understandings of OSCC pathogenic mechanism and might provide a new method for OSCC treatment.

Materials and Methods

Cell Culture

Human OSCC cell lines SCC9, CAL-27 and FaDu were obtained from the American Type Culture Collection (ATCC; Manassas, VA, USA), HSC3 was purchased from the Japanese Collection of Research Bioresources Cell Bank (JCRB, Osaka, Japan), OECM-1 was purchased from Merck KgaA (Darmstadt, Hesse, Germany) and normal oral keratinocyte (NOK) was purchased from ScienCell (Carlsbad, CA, USA). SCC9 cells and OECM-1 cells were cultured in 1:1 Dulbecco's modified Eagle's medium (DMEM; Gibco, Rockville, MD, USA) and Ham's F12 medium (Gibco, Rockville, MD, USA) supplemented with 10% fetal bovine serum (FBS; Invitrogen, Carlsbad, CA, USA), 1% penicillin-streptomycin (Invitrogen) and hydrocortisone (Invitrogen, Carlsbad, CA, USA). Other cells were cultured in DMEM (Gibco, Rockville, MD, USA) supplemented with 10% FBS (Invitrogen, Carlsbad, CA, USA) and 1% penicillin-streptomycin (Invitrogen, Carlsbad, CA, USA). All cells were incubated at 37°C in a humidified incubator containing 5% CO_2.

Cell Transfection

Small interfering RNA (siRNA) targeting BLACAT1 (si-BLACAT1) and negative control (si-NC), miR-142-5p mimic (miR-142-5p) and its negative control (miR-NC), miR-142-5p inhibitor (anti-miR-142-5p) and corresponding negative control (anti-miR-NC), pcDNA3.1-BLACAT1 overexpression vector (pcDNA-BLACAT1) and empty vector (pcDNA) were synthesized by GenePharma (GenePharma, Shanghai, China). After cells were transfected for 48 h by using Lipofectamine 2000 (Thermo Fisher Scientific, Waltham, MA, USA), they were harvested for the following experiments.

Total RNA Extraction and Quantitative Real-Time Polymerase Chain Reaction (qRT-PCR)

Total RNAs were isolated from OSCC cells by TRIzol (Thermo Fisher Scientific, Waltham, MA, USA) according to the manufacturer's instructions. NanoDrop 2000 (Thermo Fisher Scientific, Waltham, MA, USA) was used to detect RNAs concentrations. Then the RNAs were reverse transcribed to cDNAs by using High-Capacity cDNA Reverse Transcription Kit (Thermo Fisher Scientific, Waltham, MA, USA) or All-in-One miRNA First Strand cDNA Synthesis Kit (GeneCopoeia, FulenGen, China). qRT-PCR was conducted using iQ™ SYBR® Green Supermix (Bio-Rad Laboratories, Hercules, CA, USA) on an ABI 7500 fast system (Thermo Fisher Scientific, Waltham, MA, USA). The expressions of BLACAT1 and miR-142-5p were analyzed *via* $2^{-\Delta\Delta Ct}$ method with GAPDH or U6 as internal control. The primers used in this study were as follows: BLACAT1: 5'-CAAG AGGAG-CCGGCTTAGCATCTA-3' (forward) and 5'-AC-GGTTCCAGTCCTCAGTCAG-3' (reverse); GAPDH: 5'-TCCACCACCCTGTTGCTGTA-3' (forward) and 5'-ACCACAGTCCATGCCATCAC-3' (reverse); miR-142-5p: 5'-CCGGTCATAA AGTAGAAAGC-3' (forward) and 5'- GTGCAGGGTCCGAGGT -3' (reverse); U6 5'-CTCGCTTCGGCAGAC-3' (forward) and 5'-AACGCTTACGAATTT-3' (reverse).

MTT Assay

Firstly, cells were seeded into 96-well plates at a density of 5.0×10^3 cells per well and cultured at 37°C for 24 h. Then 3-(4, 5-dimethyl-2-thiazolyl)-2, 5-diphenyl-2-H-tetrazolium bro-

mide (MTT; Millipore, Billerica, MA, USA) solution was added into every well at indicated times and cultured for another 4 h. Subsequently, supernatant was removed and dimethyl sulfoxide (DMSO; Millipore, Billerica, MA, USA) was added to dissolve formazan crystals. The absorbance value was measured at 490 nm using a microplate reader (Bio-Rad, Hercules, CA, USA).

Transwell Assay

Cell migration and invasion were evaluated by transwell chambers with a pore size of 8 μm. For cell migration assay, cells were resuspended in serum-free DMEM (Gibco, Rockville, MD, USA) and added into the upper chamber; DMEM (Gibco, Rockville, MD, USA) with 10% fetal bovine serum (FBS; Invitrogen, Carlsbad, CA, USA) was added into the lower chamber. After cells were incubated for 24 h, cells that were on the upper surface of the upper chamber were removed and cells that were on the lower surface of the upper chamber were fixed in 90% ethyl alcohol and stained with 0.1% crystal violet (Millipore, Billerica, MA, USA) for 15 min. The number of migrated cells was analyzed under a microscope. For cell invasion assay, the procedures were the same as cell migration assay except that the upper chambers were pre-coated with matrigel matrix (BD Biosciences, Franklin Lakes, NJ, USA).

Luciferase Reporter Assay

The full sequences of BLACAT1 or its matched mutant containing the potential binding sites of miR-142-5p were amplified and inserted into psiCHECK-2 vectors (Promega, Madison, WI, USA) to construct WT-BLACAT1 and MUT-BLACAT1, respectively. Then the luciferase vector WT-BLACAT1 or MUT-BLACAT1 was transfected into cells together with miR-142-5p or miR-NC by using Lipofectamine 2000 (Thermo Fisher Scientific, Waltham, MA, USA) according to the user manual. Subsequently, luciferase activities were detected using a Dual-Luciferase Reporter Assay Kit (Promega, Madison, WI, USA).

Western Blot Assay

Cells were lysed in RIPA lysis buffer (Beyotime, Shanghai, China) to isolate proteins. Next, proteins concentrations were examined by using BCA protein assay kit (Beyotime). Then, an equal amount of proteins was separated by sodium dodecyl sulphate-polyacrylamide gel electrophoresis (SDS-PAGE) and transferred onto polyvinylidene difluoride (PVDF) membranes (Millipore, Billerica, MA, USA). After the membranes were blocked in non-fat milk for 1 h at room temperature, they were incubated with primary antibodies (including anti-CyclinD1, anti-p21, anti-p27, anti-MMP-2, anti-MMP-9, anti-MMP-14 and anti-GAPDH) overnight at 4°C. Subsequently, the membranes were washed with TBST three times, they were incubated with horseradish peroxidase (HRP)-conjugated secondary antibody at room temperature for 2 h. Finally, protein signals were detected using Enhanced Chemiluminescent (ECL) kit (Millipore, Billerica, MA, USA) and quantified using Image J software.

Statistical Analysis

The experiments in this study were all repeated at least three times and the data were presented as mean ± standard deviation (SD). The differences in two groups and multiple groups were analyzed by Student's *t*-test or one-way analysis of variance (ANOVA) followed by Tukey's test. $p<0.05$ was considered as statistically significant.

Results

BLACAT1 was Upregulated and miR-142-5p was Downregulated in OSCC Cells

In order to explore the potential roles of BLACAT1 and miR-142-5p in OSCC development, we detected the expressions of BLACAT1 and miR-142-5p in five OSCC cell lines (SCC9, HSC3, CAL-27, FaDu and OECM-1) and one normal oral keratinocyte (NOK) by qRT-PCR. The data showed that BLACAT1 expressions were significantly elevated and miR-142-5p expressions were significantly decreased in SCC9, HSC3, CAL-27, FaDu and OECM-1 cell lines compared to NOK cell line (Figure 1A and B), indicating that BLACAT1 and miR-142-5p might be involved in the progression of OSCC. In addition, we selected SCC9 and HSC3 cell lines for the following experiments due to the higher expression of BLACAT1 and the lower expression of miR-142-5p compare to other cell lines.

BLACAT1 Knockdown Suppressed Cell Viability in OSCC Cells

To explore the functional roles of BLACAT1 in OSCC development, si-BLACAT1 was transfected into SCC9 and HSC3 cells to knockdown the expression of BLACAT1. qRT-PCR was performed to detect the transfection efficiency and the data showed that BLACAT1 expressions were strikingly decreased after si-BLACAT1 transfection (Figure 2A). MTT assay results indicated that cell viability was markedly inhibited in si-BLA-

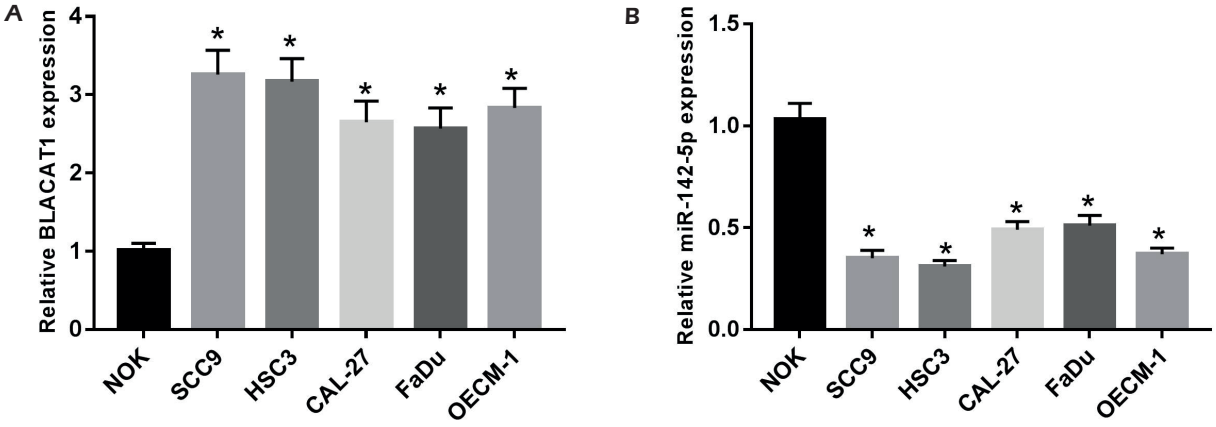

Figure 1. The expressions of BLACAT1 and miR-142-5p in OSCC cell lines. **A,** The expressions of BLACAT1 in five OSCC cell lines and NOK cell line were detected by qRT-PCR. **B,** The expressions of miR-142-5p in five OSCC cell lines and NOK cell line were detected by qRT-PCR. **p*<0.05.

CAT1 transfected SCC9 and HSC3 cells compared to si-NC transfected group (Figure 2B and C). Meanwhile, the protein expression levels of cell cycle-associated proteins (including CyclinD1, p21 and p27) were determined by Western blot assay. We found that BLACAT1 knockdown led to a significant decrease of CyclinD1 and an obvious increase of p21 and p27 in both SCC9 and HSC3 cells (Figure 2D-F). Collectively, these results suggested that knockdown of BLACAT1 could inhibit OSCC cells viability.

BLACAT1 Knockdown Suppressed Migration and Invasion of OSCC Cells

To further explore the functional roles of BLACAT1 in OSCC development, we also analyzed si-BLACAT1 transfected SCC9 and HSC3 cells migration and invasion by transwell migration and invasion assay, respectively. The results showed that the migratory and invasive numbers of SCC9 and HSC3 cells were significantly decreased in si-BLACAT1 transfected groups compared to si-NC transfected groups (Figure 3A-C). Then, we

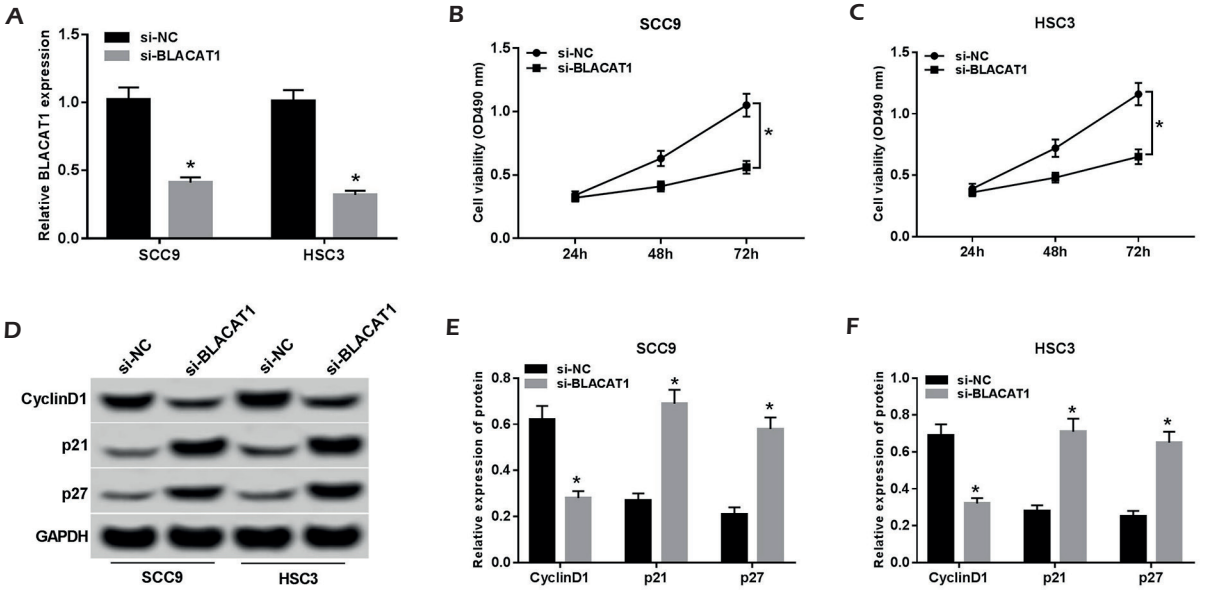

Figure 2. Cell viability was suppressed in si-BLACAT1 transfected OSCC cells. **A,** The expressions of BLACAT1 in SCC9 and HSC3 cells transfected with si-BLACAT1 were examined by qRT-PCR. **B** and **C,** Cell viability in si-BLACAT1 transfected SCC9 and HSC3 cells were evaluated by MTT assay. **D-E,** The relative protein levels of CyclinD1, p21 and p27 in si-BLACAT1 transfected SCC9 and HSC3 cells were measured by Western blot analysis. **p*<0.05.

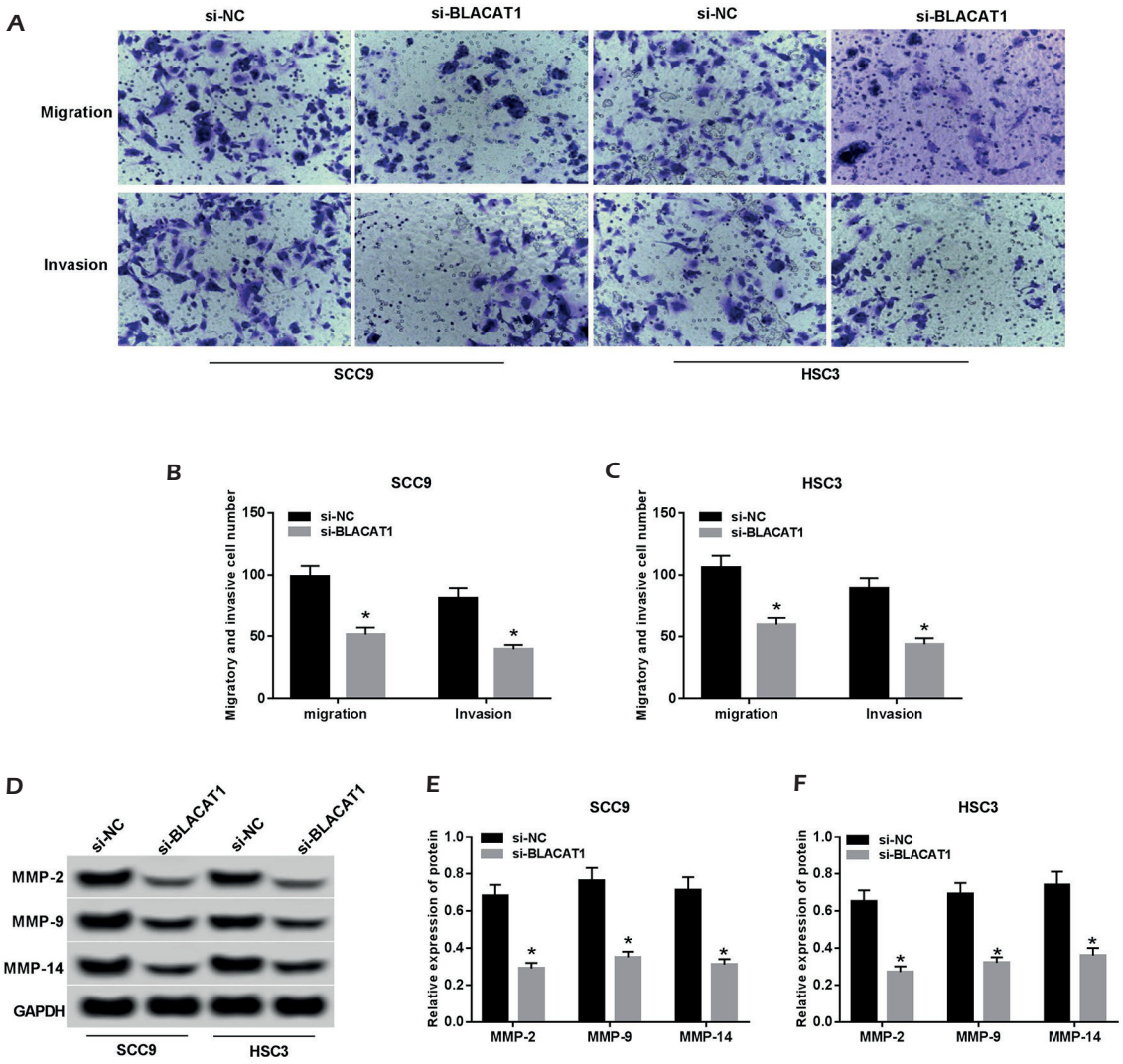

Figure 3. Cell migration and invasion were suppressed in si-BLACAT1 transfected OSCC cells. **A-C,** Cell migration and invasion abilities in si-BLACAT1 transfected SCC9 and HSC3 cells were evaluated by transwell migration and invasion assay, respectively (magnification × 300). **D-E,** The relative protein levels of MMP-2, MMP-9 and MMP-14 in si-BLACAT1 transfected SCC9 and HSC3 cells were measured by Western blot analysis. *p<0.05.

evaluated the protein levels of metastasis-related hallmarks (including MMP-2, MMP-9 and MMP-14) by Western blot analysis and found that the protein levels of MMP-2, MMP-9 and MMP-14 were apparently downregulated in si-BLACAT1 transfected SCC9 and HSC3 cells (Figure 3D-F). Taken together, knockdown of BLACAT1 suppressed OSCC cell migration and invasion.

BLACAT1 Directly Targeted to miR-142-5p and Negatively Regulated miR-142-5p Expression in OSCC Cells

Through bioinformatics software starBase analysis, we found that BLACAT1 contained the potential binding sites of miR-142-5p (Figure 4A). After luciferase reporter assay was performed to verify this prediction, the data showed that the luciferase activities in WT-BLACAT1 and miR-142-5p co-transfected SCC9 and HSC3 cells were greatly inhibited compared to WT-BLACAT1 and miR-NC co-transfected group, while the luciferase activities were not affected in MUT-BLACAT1 and miR-142-5p/miR-NC co-transfected SCC9 and HSC3 cells (Figure 4B and C). Subsequently, we transfected pcDNA-BLACAT1 or si-BLACAT1 or their matched controls into SCC9 and HSC3 cells to detect the expression of miR-142-5p. The results showed that BLACAT1 overexpression significantly restrained miR-142-5p expression, while BLACAT1 knockdown significantly increased miR-

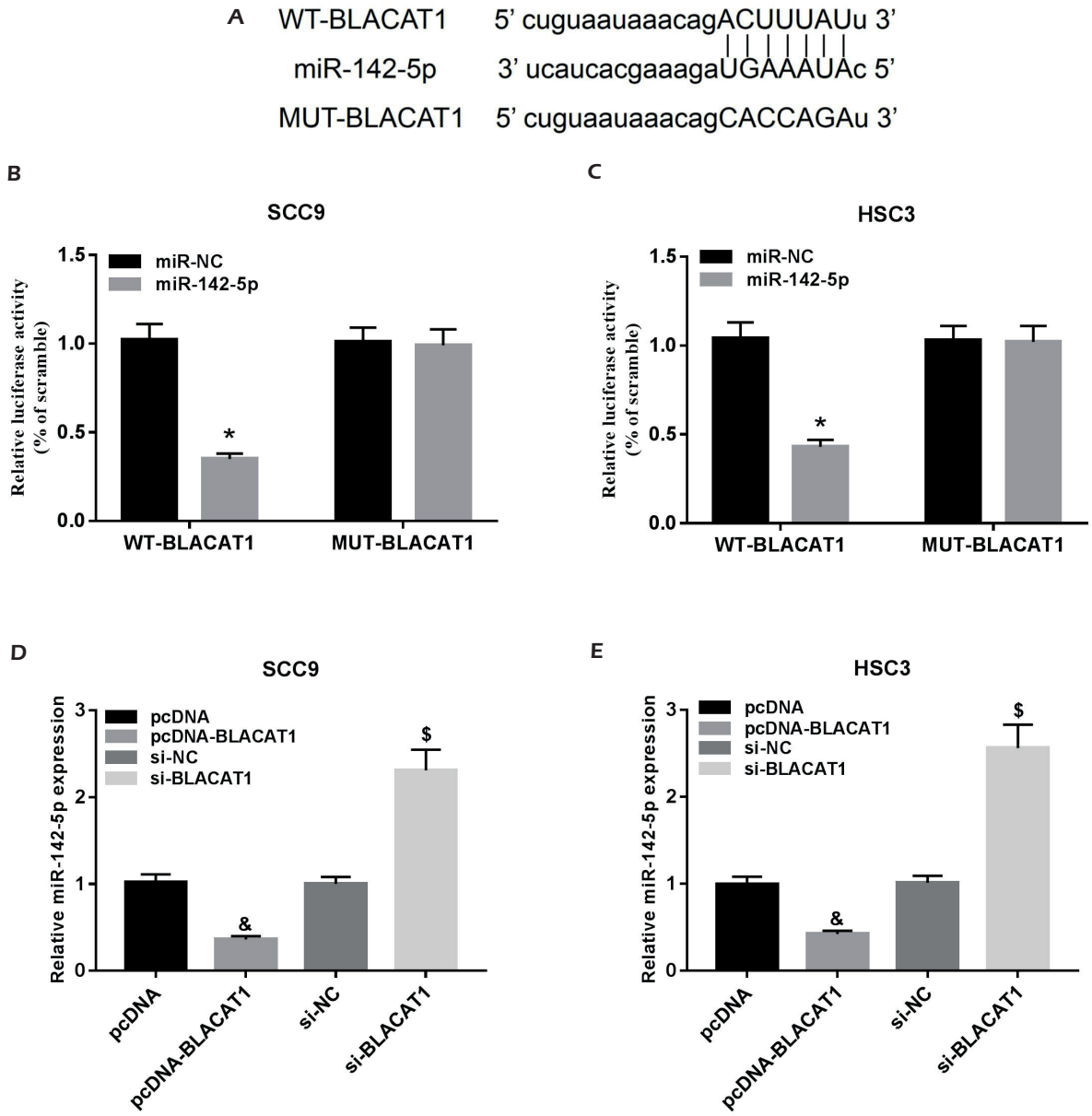

Figure 4. BLACAT1 negatively regulated miR-142-5p expression by direct targeting. **A,** The putative binding sites between BLACAT1 and miR-142-5p. **B** and **C,** The luciferase activities in WT-BLACAT1 or MUT-BLACAT1 and miR-142-5p or miR-NC co-transfected SCC9 and HSC3 cells were evaluated by luciferase reporter assay. **D** and **E,** The relative expression of miR-142-5p in BLACAT1 overexpression, BLACAT1 knockdown and their corresponding controls treated SCC9 and HSC3 cells. Note: When compared to miR-NC group, pcDNA group and si-NC group, $^{*}p<0.05$, $^{\&}p<0.05$ and $^{\$}p<0.05$ were considered as statistically significant, respectively.

142-5p expression (Figure 4D and F). These data demonstrated that BLACAT1 could regulate the expression of miR-142-5p via sponging.

Overexpression of miR-142-5p Suppressed Cell Viability, Migration and Invasion in OSCC Cells

In order to explore the biological roles of miR-142-5p in OSCC, miR-142-5p was transfected into SCC9 and HSC3 cells to achieve the overexpression of miR-142-5p. The transfection efficiency was examined by qRT-PCR. The results presented that miR-142-5p transfection led to a significant elevation of miR-142-5p expression in SCC9 and HSC3 cells (Figure 5A). MTT assay showed that cell viability of SCC9 and HSC3 cells was repressed by miR-142-5p transfection (Figure 5B and C). We also evaluated the levels of CyclinD1

and p21 by Western blot analysis and found that CyclinD1 expression was downregulated and p21 expression was upregulated in SCC9 and HSC3 cells after miR-142-5p transfection (Figure 5D-F). Additionally, transwell migration and invasion assays revealed that overexpression of miR-142-5p suppressed SCC9 and HSC3 cells migration and invasion abilities (Figure 5G and H). Moreover, Western blot analysis showed that the protein levels of MMP-2 and MMP-9 were markedly downregulated in miR-142-5p transfected SCC9 and HSC3 cells compared to miR-NC group (Figure 5H-K). The results were consistent with transwell migration and invasion assays results. All

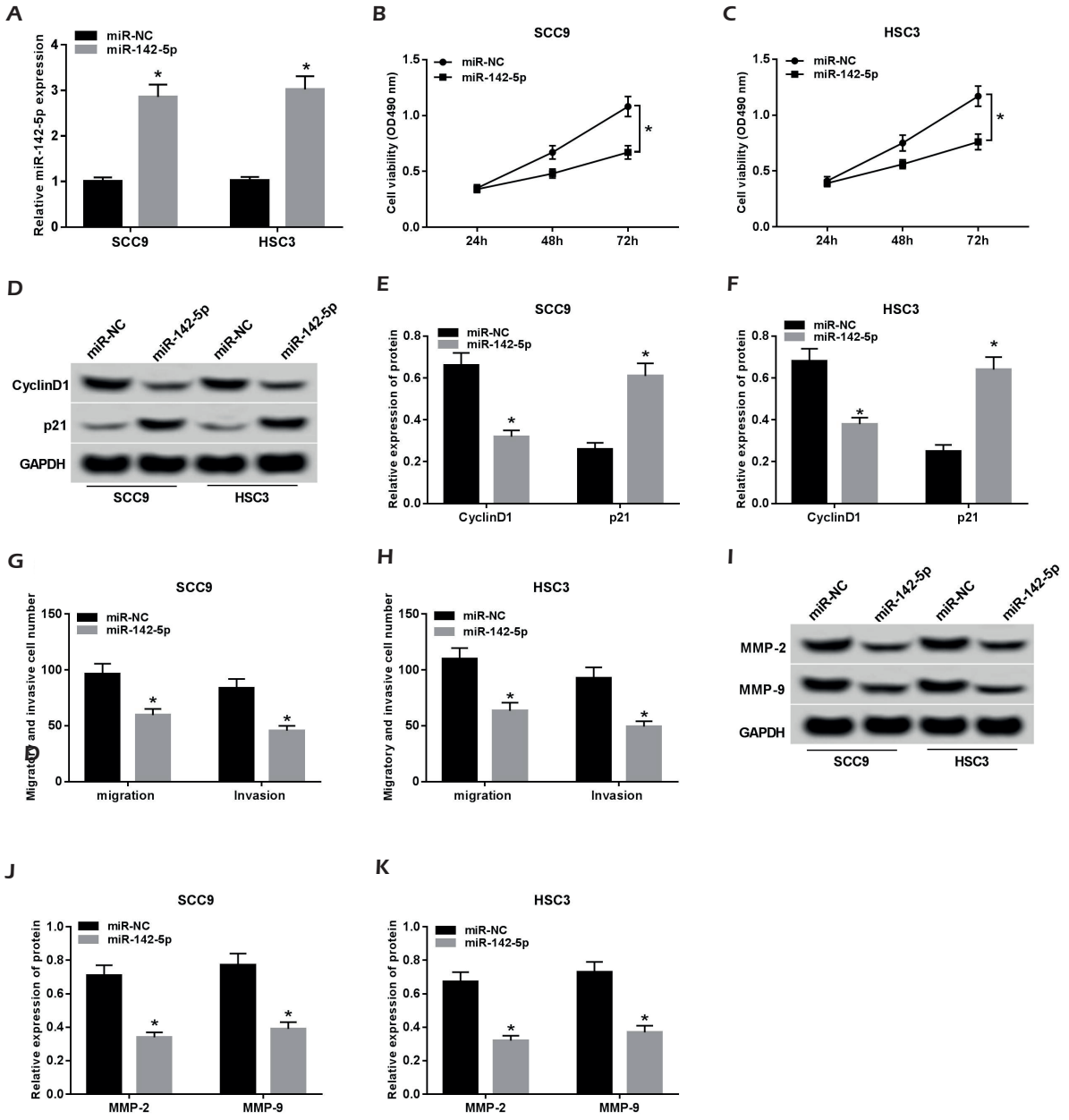

Figure 5. The roles of miR-142-5p in OSCC cells viability, migration and invasion. **A,** The expressions of miR-142-5p in miR-142-5p transfected SCC9 and HSC3 cells were determined by qRT-PCR. **B** and **C,** Cell viability in miR-142-5p transfected SCC9 and HSC3 cells were determined by MTT assay. **D-E,** The protein levels of CyclinD1 and p21 in miR-142-5p transfected SCC9 and HSC3 cells were measured by Western blot analysis. **G** and **H,** Cell migration and invasion abilities in SCC9 and HSC3 cells transfected with miR-142-5p were evaluated by transwell migration and invasion assay, respectively. **I-K,** The protein levels of MMP-2 and MMP-9 in miR-142-5p transfected SCC9 and HSC3 cells were measured by Western blot analysis. *$p<0.05$.

these data demonstrated that miR-142-5p overexpression could repress cell viability, migration and invasion in OSCC cells.

Inhibition of miR-142-5p Overturned the Effects of BLACAT1 Knockdown on Cell Viability, Migration and Invasion in OSCC Cells

To investigate whether BLACAT1 knockdown repressed cell viability, migration and invasion by regulating miR-142-5p, si-BLACAT1, si-NC, si-BLACAT1+anti-miR-142-5p or si-BLACAT1+anti-miR-NC was transfected into SCC9 and HSC3 cells. We found that miR-142-5p expression was significantly upregulated by BLACAT1 knockdown in SCC9 and HSC3 cells, whereas this effect was abolished by miR-142-5p inhibition (Figure 6A). MTT assay showed that miR-142-5p inhibition reversed the effect of BLACAT1 knockdown on cell viability in both SCC9 and HSC3 cells (Figure 6B and C). miR-142-5p inhibition also reversed the decrease of CyclinD1 and the increase of p21 caused by BLACAT1 knockdown in both SCC9 and HSC3 cells (Figure 6D-G). Furthermore, transwell migration and invasion assays showed that anti-miR-142-5p transfection overturned the suppression of migration and invasion of SCC9 and HSC3 cells caused by si-BLACAT1 transfection (Figure 6H and I). Western blot assay implicated that si-BLACAT1 transfection led to an obvious reduction of MMP-2 and MMP-9 protein expression levels, whereas the effects were abolished after anti-miR-142-5p transfection in SCC9 and HSC3 cells (Figure 6J-M). These data demonstrated that inhibition of miR-142-5p could attenuate the inhibitory effects of cell viability, migration and invasion caused by BLACAT1 knockdown in OSCC cells.

Discussion

OSCC is one of the most common malignancies in developing countries, seriously threatening people's physical and mental health[18]. Although the diagnosis and treatment methods for OSCC have been improved in recent years, the underlying molecular mechanisms of OSCC occurrence are still not clear and the survival rate of OSCC patients is very low[1]. Some reports[9-11] have shown that lncRNAs were involved in OSCC. In this study, we explored the functional roles of lncRNA BLACAT1 in OSCC progression and found that BLACAT1 knockdown suppressed the viability, migration and invasion of OSCC cells, while miR-142-5p inhibition abolished these effects.

BLACAT1 has been demonstrated to be associated with many tumors' development. For instance, Chen et al[19] revealed that BLACAT1 was significantly upregulated in small-cell lung cancer cells and regulated small-cell lung cancer cells proliferation, migration and invasion. Su et al[20] in colorectal cancer (CRC) showed that BLACAT1 was highly expressed in CRC tissues and BLACAT1 knockdown repressed CRC cells proliferation and promoted apoptosis *in vitro* and *vivo*. Wang et al[21] in cervical cancer (CC) also demonstrated that BLACAT1 was distinctly increased and promoted CC cells proliferation and metastasis by activating wnt/β-catenin pathway. In agreement with these findings, our study indicated that BLACAT1 was significantly increased in five OSCC cell lines and downregulation of BLACAT1 suppressed the viability, migration and invasion of SCC9 and HSC3 cells. Moreover, we examined the protein levels of cell cycle associated proteins (including CyclinD1, p21 and p27) and metastasis-related hallmarks (including MMP-2, MMP-9 and MMP-14). We found that CyclinD1 was decreased; p21 and p27 were increased in BLACAT1 knockdown SCC9 and HSC3 cells, consistently with MTT assay result. We also found that MMP-2, MMP-9 and MMP-14 were downregulated after BLACAT1 knockdown, and it is consistent with transwell assay results. Moreover, the results suggested that BLACAT1 played important roles in OSCC development.

More and more reports have shown that lncRNAs could sponge to miRNAs[22]. In this study, we demonstrated that miR-142-5p was a target of BLACAT1 and BLACAT1 overexpression suppressed the expression of miR-142-5p. miR-142-5p has been demonstrated to be abnormally expressed in human cancers and regulated cancers progression. For example, Lou et al[23] in hepatocellular carcinoma (HCC) suggested that miR-142-5p was aberrantly expressed in HCC and miR-142-5p led to significant suppression of HCC cells viability and significant promotion of apoptosis. Yan et al[24] demonstrated that miR-142-5p was downregulated in gastric cancer (GC) and miR-142-5p upregulation suppressed GC cells migration and invasion *in vitro* and *vivo*. In this study, miR-142-5p was apparently decreased in OSCC cells and miR-142-5p overexpression showed similar effects with BLACAT1 knock-

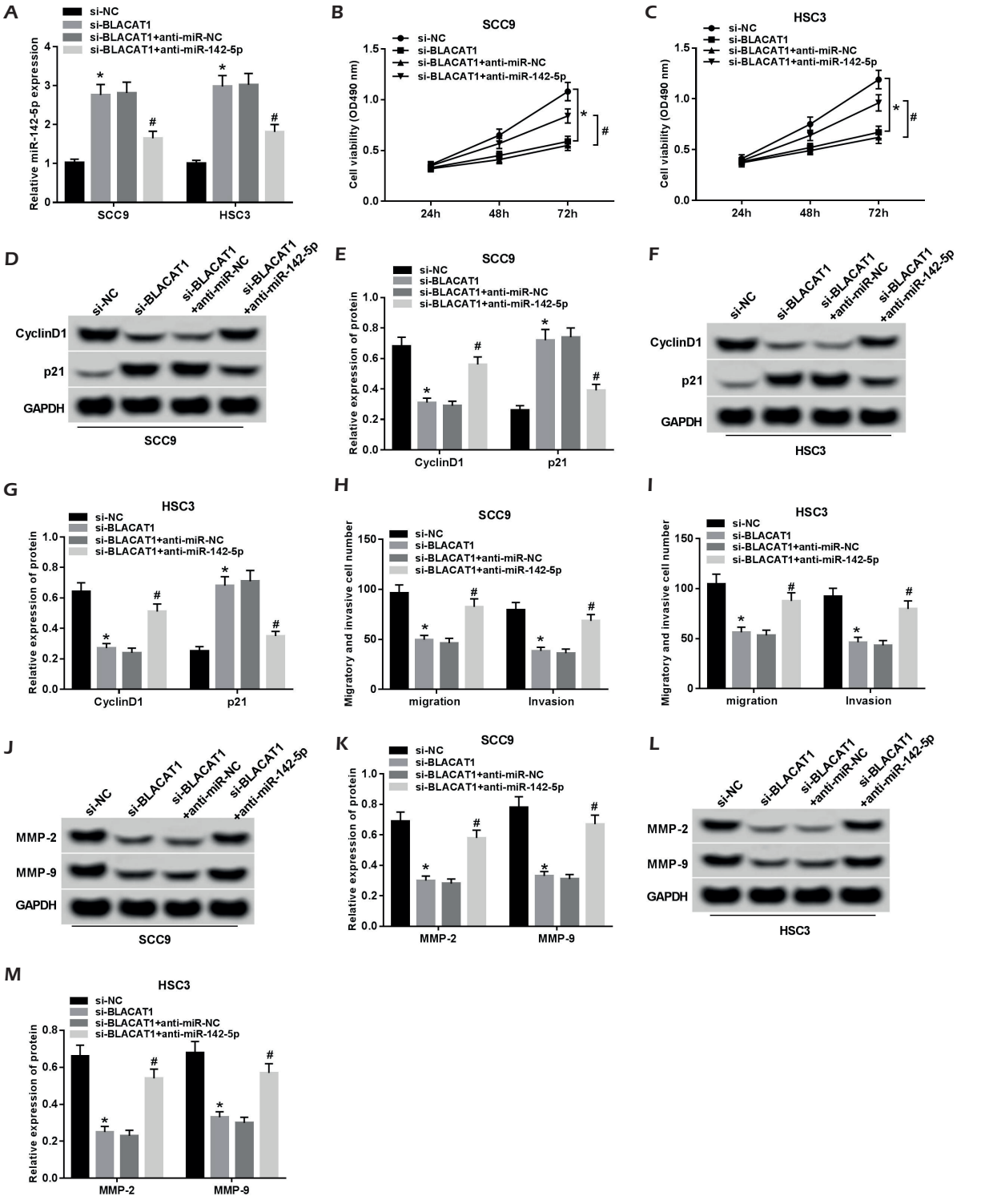

Figure 6. miR-142-5p inhibition overturned the effects of BLACAT1 knockdown on cell viability, migration and invasion in OSCC cells. si-BLACAT1, si-NC, si-BLACAT1+anti-miR-142-5p or si-BLACAT1+anti-miR-NC was transfected into SCC9 and HSC3 cells. **A,** miR-142-5p expression was determined by qRT-PCR. **B** and **C**, Cell viability was evaluated by MTT assay. **D-G,** CyclinD1 and p21 protein expressions were determined by Western blot analysis. **H** and **I,** Cell migration and invasion were detected by transwell migration and invasion assay. **J-M,** MMP-2 and MMP-9 protein expressions were detected by Western blot analysis.

down on OSCC cell viability, migration and invasion. Furthermore, inhibiting the expression of miR-142-5p could reverse the inhibitory effects of BLACAT1 knockdown on cell viability, migration and invasion in SCC9 and HSC3 cells. These findings indicated that BLACAT1 regulated OSCC cell viability, migration and invasion by targeting miR-142-5p.

However, we did not verify the functions of BLACAT1 and miR-142-5p in animal, and more clinical experiments should be performed in OSCC patients. We will conduct these experiments in future.

Conclusions

This study demonstrated that BLACAT1 could promote OSCC cell viability, migration and invasion by regulating the expression of miR-142-5p. These findings contributed to our understandings of OSCC pathogenesis and might provide a novel target for OSCC treatment.

Conflict of Interests

The Authors declare that they have no conflict of interests.

Declarations - Ethics approval and consent to participate

This study was approved by the Ethics Committee of Beijing Tongren Hospital, Capital Medical University. The methods used in this study were performed in accordance with relevant guidelines and regulations. Written consent was obtained from the participants or guardians of participants under 16 years old.

Availability of data and materials

All original data and materials are available from the corresponding author upon request. .

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