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# Effects of LINC00261 Targeting miR-93 on Proliferation and Migration of Cutaneous Squamous Cell Carcinoma

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## Abstract

**Objective:** To investigate the effect of long intergenic non-protein coding RNA 261 (LINC00261) on the proliferation and migration of cutaneous squamous cell carcinoma (CSCC) cells and its mechanism.

**Methods:** The cancer tissues of 41 patients with CSCC who underwent surgical treatment in our hospital from December 2017 to December 2018 were selected, and normal skin tissues of 41 healthy people in the same period were collected. The expression of LINC00261 in these tissues was determined by real-time quantitative PCR. The CSCC SCL-1 cells were taken as research objects, and separately transfected with LINC00261 overexpression vector and miR-93 inhibitor. The effects of LINC00261 overexpression and inhibition of miR-93 on the proliferation, cell cycle and migratory ability of SCL-1 cells and the expressions of p21 and E-cadherin protein were detected by MTT assay, flow cytometry, Transwell assay and Western blotting, respectively. The regulatory relationship between LINC00261 and miR-93 was verified by dual luciferase reporter assay.

**Results:** The expression level of LINC00261 in CSCC tissues was significantly lower than that in normal skin tissues ( $P < 0.05$ ). After overexpression of LINC00261 or inhibition of miR-93 expression, the survival rate of SCL-1 cells, the number of migrating cells and the percentage of cells in S phase decreased ( $P < 0.05$ ), while the percentage of cells in G0 phase and the expression levels of p21 and E-cadherin protein rose ( $P < 0.05$ ). The luciferase activity of cells co-transfected with wild-type LINC00261 in miR-93 group was lower than that in miR-NC group ( $P < 0.05$ ), while the luciferase activity of cells co-transfected with mutant LINC00261 in miR-93 group was not significantly different from that in miR-NC group ( $P > 0.05$ ). After overexpression of LINC00261, the expression level of miR-93 in SCL-1 cells was down-regulated ( $P < 0.05$ ). After overexpression of LINC00261 and miR-93 at the same time, the survival rate of SCL-1 cells, the number of migrating cells and the percentage of cells in S phase increased ( $P < 0.05$ ), while the percentage of cells in G0 phase and the expression levels of p21 and E-cadherin protein declined ( $P < 0.05$ ).

**Conclusion:** The expression of LINC00261 is down-regulated in CSCC tissues, and overexpression of LINC00261 may weaken the proliferating and migratory abilities of CSCC SCL-1 cells through targeted inhibition of miR-93 expression.

**Keywords:** cutaneous squamous cell carcinoma, LINC00261, miR-93, cell proliferation, metastasis.

## 1. Introduction

Cutaneous squamous cell carcinoma (CSCC) is a

type of tumor derived from keratinocytes of epidermal disease whose incidence has increased worldwide annually, severely affecting human life and health (ZHU Lude et al, 2018). The Tumor frequency and progression is closely linked to

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abnormal expression of the epigenetic modifications in the genes (FAN Yueying et al., 2018). Therefore, it is of great importance for diagnosing and treatment of CSCC that the aberrant genes in CSCC patients are being investigated and their impacts on malignant biological activities such as tumor cell proliferation, migration, and invasion. Long RNA non-coding (LNCRNAs) and MicroRNAs (miRNAs) are two types of little RNAs, which play an important role as diseases, especially tumours, occur and evolve (HUANG Jiabin et al. 2018; ZHAO Susu et al. 2013). Studies have also shown that lncRNAs can serve to control the expression of objective genes and, thus, the development of diseases as competitive endogenous RNAs (WANG Jing et al. 2018). RNA 261 (LINC00261) is a long intergenic non-protein coding newly found lncRNA that participates in numerous tumors and is involved in their incidence and growth over the last few years. Zhou et coll. (2019) has shown that LINC00261 is down-regulated in colon cancer and its low expression is strongly linked to the clinical level, metastases of the lymph node and the recurrence free survival of colon cancer, allowing for an evaluation of metastasis-free and recurrence-free survival of colon cancer as a new molecular biological marker patients. According to the study of Gao *et al.* (2020), the expression of LINC00261 was up-regulated in cholangiocarcinoma tissues and cell lines, and the down-regulation of LINC00261 expression inhibited the proliferation, migration and invasion of cholangiocarcinoma cells. These studies suggest that LINC00261 plays different roles in different tumors. At present, the expression of LINC00261 in CSCC and its effect on the malignant biological behaviors remain unknown. It was predicted by bioinformatics software that miR-93 is the target gene of LINC00261. Studies have shown that the expression of miR-93 is up-regulated in tumor tissues and cells such as non-small cell lung cancer and gastric cancer, inhibition of its expression can reduce malignant biological behaviors such as proliferation, migration and invasion of tumor cells, and miR-93 is a potential target for the treatment of tumors (Li C. et al., 2017)(Ma D.H. et al., 2017). Currently, the role of miR-93 in CSCC has not been reported. Hence, in this study, the expression level of LINC00261 in CSCC tissues was first detected, and the CSCC SCL-1 cells were taken as the research objects. The effect of LINC00261 overexpression on the proliferation and migration

of SCL-1 cells and whether regulating the expression of miR-93 can influence tumor cell proliferation and migration were explored, aiming to provide a new method for targeted molecular treatment of CSCC.

## 2. Materials and methods

### 2.1. Baseline clinical data

In total, from December 2017 to December 2018, 41 patients with CSCC diagnosis were chosen as subjects and received surgical treatment in the Dermatology Department at our hospital. The average age was (57, 25±8,76) years, including 29 males and 12 females. The TNM stage revealed 22 phase I and II cases, as well as 19 phase III and IV cases. 11 well-defined carcinomas, 16 moderately differentiated and 14 poorly differentiated cases were identified. There were 3 deaths. Of the patients, 15 had metastasis of the lymph node, while the remaining 26 had no metastasis. Both patients were not treated before surgery, for example radiation therapy and chemotherapy. In addition, normal tissues were chosen as a control group of 41 healthy individuals who were surgically resected during the same time. The tissues consisted of 25 men and 16 women, with an average age of (55.24±9.13) years. In general data such as age and gender there were no significant discrepancies between the two classes. The research was accepted by the hospital's Ethics Committee and informing consent was openly signed by the patient's family members.

### 2.2. Experimental cells and reagents

Shanghai Cell Bank of the Chinese Academy of Sciences was responsible for CSCC SCL-1 cells. Cytiva (HyClone®) has been acquired by fetal bovine serum (FBS) and RPMI 1640 scale. Sigma (US) is derived from methyl thiazolyl tetrazolium (MTT) and trypsin. Transfection Reagent kit from Thermo Fisher Scientific (Invitrogen™), 2000, were purchase from the RNA and Lipofectamine™ Extraction Package. Shenzhen Jingmei Biological Engineering Co., Ltd, and PCR primers have been developed and synthesized by Sangon Biotech, Shanghai, Ltd. in the reverse transcription kit and polymerase chain reaction kit (PCR). Beijing bought the cell cycle package and the dual luciferase active test set, plus a protein assay package for bicinchoninic acid (BCA). Solarbio Science & Technology Co, Ltd. Technology Co. Santa Cruz Biotechnology (US) was acquired with rabbit anti-human p21 and polyclonal antibodies against e-cadherin. Shanghai GenePharma Co., Ltd. all purchased the following

vector INCO0261 over-expression (pcDNA3.1), vector empty (pcDNA3.1), miR-93 inhibitor (anti-miR-93), anti-meR-NC inhibitor, miR-93 mimics and simulated negative regulation (miR-NC).

### 2.3. Experimental methods

#### Cell culture and transfection

In RPMI 1640, medium 10% phosphatic buffer saline (PBS) stored in the incubator with 5 % CO<sub>2</sub> and 97% of humidity, the SCL-1 cells were resuscitated and cultivated. Every 2–3 d the medium was changed to new. The medium was aspirated and discarded at 80–90% confluence, and the cells were washed in a sufficient amount of FBS. Then the solution was digested and subcultured with 0.25% of trypsin. The cells were inoculated into a six-well plate with 1 in 10<sup>5</sup> cells per well during the Logarithmic growth stage. As cells converged to 60%, the medium was substituted with FBS-free water. SCL-1 cells were transfected to PcDNA3.1-LINC00261 (group of pcDNA3.1-LIMA-261) (group of pcDNA3.1-LINC00261), pcDNA3.1 (group of pcDNA3.1), Anti-miR-93 (group of antimirus), Anti-miR-NC (group of antimirus), pcDNA3.1-LINC00261 and mics miR-93 (group of pcDNA3.1-LINC00261 + miR-NC) and pcDNA3.1. The medium was replaced with fresh one at 12 h after transfection. After 48 h culture, LINC00261 or miR-93 expression level in cells was calculated with the objective to evaluate the transfectional effect in real time quantitative PCR (RT-QPCR) and the cells had been assembled for follow-up experiments.

#### Measurement of LINC00261 and miR-93 expression levels by RT-qPCR

Total RNA has been extracted using an RNA extraction kit from tissues or cells. RNA purity and concentration were measured with the aid of an analyser for micro-nucleic acid. The RNA has been transcribed reverse into complementary deoxyribonucleic acid (cDNA) following the reverse transcript kit instructions. The cDNA was then performed as a prototype with PCR amplification. Verstärkungsverfahren (pre-denaturation with 95 ° C for 5 minutes, dematurations with 95 ° C for 10 s, extension with 60 ° C for 30 s, and rinsing with 72 ° C for 30 s) have been defined in the following manner for a total of 35 cycles. LINC00261 upstream first:

5'-ATCAGGAGGCAGGCCA-3', downstream primary: 5'-TTCAGCTCT TAGGCAGGAC-3'. upstream first for GAPD: 5'-GACTCATGACCACAGTCATGC-3', downstream first: 5'-AGAG GCAGGGATGTCTG-3; upstream first

for miR-93: 5'-GTCCGATTGTGCGCCGC-3', downstream first: 5'- CCG gAAGATACGGTCGTC-3'. The internal references for LINC00261 and miR-93 respectively are Glyceraldehyde 3-phosphate dehydrogenase (GAPDH) and U6. LINC00261 and miR-93 relative expression levels were determined by means of the 2-to-Cet process.

#### Detection of cell viability by MTT assay

Every group of cells was inoculated in a 96-plate with 5 by 10<sup>3</sup> cells per well and three replicates every. After culture for 24 h, a 4-hour inoculation of 20 μL of MTT solution (5 mg) has been applied to each well. Later, the medium was aspirated and discarded and the reaction was 5 minutes with 150 μL of dimethyl sulfoxide. The absorption (A) was measured by using a microplate reader at 490 nm following uniform mixing. Cell-survival rate (percent) = An experimental group / A control group = 100 percent. Cell-survival rate (percent).

#### Detection of cell cycle by flow cytometry

Inoculated cells in each group on a plate of 24 wells with 2.5 cells in 10<sup>4</sup> cells per well, and three replicates for each group were determined. The medium was aspirated and discarded after a 24-hour fermentation, and the cells were trypsinised and selected. The cells were fixed for 24 hours with 70.0 percent ethanol at 4 ° C in compliance with the instructions of the cycle package, followed by propidium iodide stain and cytometry flux detection.

#### Detection of cell migration by Transwell assay

The cell concentration has been modified to 5 to 10<sup>4</sup> cells / mL in each community with FBS-free medium. Subsequently, in the upper Transwell chamber 100 μL was inserted and the lower chamber with 3 replicates for each category was supplemented by 500 μL of medium FBS. The medium was aspirated and discarded after 24 hours of cultivation and cells were removed and fixed with 4 % paraformaldehyde for 30 minutes. After washing with PBS, 0.4 percent crystal violet was stained for 15 minutes, then rinsed with PBS until there is no colouration. The cells were tracked by a microscope after air-dry and 5 visual fields. were randomly selected to count the transmembrane cells.

#### Measurement of P21 and E-cadherin protein expressions by Western blot

Cells were inoculated in each group in a 24-wave plate of 2.5 to 10<sup>4</sup> cells per sample, each with a 3-cell replicate collection. The medium was aspirated to and discarded after 24 hours of

fermentation, and the cells trypsinised. After washing the protein lysate with the PBS, the total protein from the cells was extracted and a quantitative assessment of the BCA protein test was made for the protein concentration. Then a sufficient amount of protein solution was added to the buffer filling, mixed well and boiled for 5 minutes at 100 ° C. Sodium sulfate-polyacrylamide gel electricity electrophoresis with 30 µg of protein in each well was performed after denaturation. Later, the protein was wately transferred to the difluoride membrane of polyvinylidene and sealed with skimmed milk powder for 5 percent for 2 hours. Following this, the protein had to be incubated separately at 4 ° C overnight in the form of p21, E- cadherin and GAPDH antibodies. The membrane washed and incubated in a secondary incubation solution of horseradish peroxidase marked with an incubation at 37 ° C for 1 hour. The membrane was washed again and the exposure photograph was then placed in the gel imaging method, with the ECL developer to improve the dark. Finally, the gray value of the target protein was analyzed using GAPDH-based ImageJ software.

#### Validation of targeting relationship between LINC00261 and miR-93 by dual luciferase reporter assay

Bioinformatics software predicts that the nuclear sequences LINC00261 and miR-93 have continuously been related to. The LINC00261 binding site sequence for miR-93 was amplified by PCR and the wild-type LINC00261 plasmid (WT-LINC00261) was created. After the binding site was mutated by means of site-directed mutagenesis technologies, the mutant plasmid LINC00261 (MUT-LINC00261) was created. WT-LINC00261, MUT-LINC00261, miR-93 and miR-NC have been transfected separately for the SCL-1 cells. The medium was replaced by fresh one at 12 o'clock after transfection. After 48 hours of culture, luciferase activity was identified in accordance with instructions from the dual luciferase test kit and expressed as the firefly luciferase activity ratio with the strength of the renilla luciferase in each community.

#### 2.4. Statistical analysis

SPSS 22.0 program evaluated all data statistically. Quarantine data were seen as a mean deflection of  $\pm$  norm ( $\bar{x} \pm s$ ). A separate sample test was used to allow comparisons between two groups, and comparisons between several groups were carried out on one-way variance analysis.

Comparisons have been made with the SNK-q test in pairs. Statistically important was found  $P < 0.05$ .

### 3. Results

#### 3.1 LINC00261 expression in CSCC tissues

LINC00261 expression was considerably less in CSCC tissue than in the normal tissue of the skin ( $P < 0.05$ ) (Table 1).

#### 3.2 Effects of LINC00261 overexpression on SCL-1 cell proliferation and migration

The LINC00261 expression level was higher for pcDNA3.1-LINC00261 cell than in pcDNA3.1, which suggested that the LINC00261 over-expression vector had been efficient in pcDNA3.1 ( $P < 0.05$ ). The cells were over-expressed with LINC00261 and transfected. Compared to those of the pcDNA3.1, the SCL-1 cells survival rates, the number of migrating cells, and S-phase percentages were reduced in the pcDNA3.1-LINC00261 group ( $P < 0.05$ ) and the expression of the P21 and E-cadherin ( $P < 0.05$ ) cells in the G0 step (Figure 1 and Table 2).

Table 1: LINC00261 expression in CSCC tissues

Group	Sample number	LINC00261
Normal skin tissue	41	1.00±0.04
CSCC tissue	41	0.24±0.03*
t	--	97.328
P	--	<0.001

Compared with normal skin tissue, \* $P < 0.05$ .

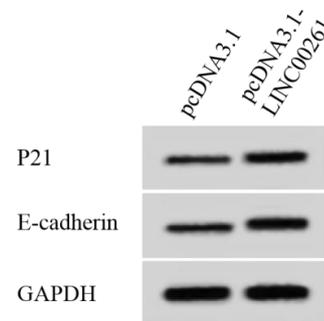


Figure 1: P21 and E-cadherin protein expressions.

#### 2.5 LINC00261 targetedly regulated miR-93 expression

Bioinformatics software has predicted continuous link sites between LINC00261 and miR-93 nucleotide sequences (Figure 2). Luciferase activity of the cells co-polluted in the WT-LINC00261 group miR-93 The luciferase activity of the cells co-polluted with MUT-LINC00261 was significantly different from that of miR-NC group ( $P < 0.05$ ). The cells of miR-NC were not significantly different from that of MUT-LINC0024. Furthermore, in pcDNA3.1-LINC00261, the

expression levels of miR-93 were lower than those of the pcDNA3.1 group ( $P < 0.05$ ) (Table 3 and 4).

Table 2: Effects of LINC00261 overexpression on SCL-1 cell proliferation and migration ( $\bar{x} \pm s, n=9$ )

Group	LINC00261	P21	E-cadherin	G0 (%)	S (%)	G1 (%)	Viability (%)	Number of migrating cells
pcDNA3.1	0.98±0.04	0.18±0.02	0.25±0.03	30.68±2.15	36.16±2.67	33.16±2.43	100.04±6.63	113±7.36
pcDNA3.1-LINC00261	3.15±0.06*	0.61±0.04*	0.75±0.05*	45.62±3.36*	19.17±1.53*	35.21±2.91	53.49±4.18*	58±5.67*
t	90.278	28.845	25.725	11.236	16.563	1.622	17.818	17.760
P	<0.001	<0.001	<0.001	<0.001	<0.001	0.1243	<0.001	<0.001

Compared with pcDNA3.1 group, \* $P < 0.05$ .



Figure 2. Binding sites between nucleotide sequences of LINC00261 and miR-93.

Table 3: Dual luciferase reporter assay results ( $\bar{x} \pm s, n=9$ )

Group	WT-LINC00261	MUT-LINC00261
miR-NC	1.04±0.06	1.02±0.06
miR-93	0.35±0.04*	1.00±0.05
t	28.706	0.7682
P	<0.001	0.4535

Compared with miR-NC group, \* $P < 0.05$ .

Table 4: LINC00261 regulated miR-93 expression ( $\bar{x} \pm s, n=9$ )

Group	miR-93
pcDNA3.1	1.01±0.05
pcDNA3.1-LINC00261	0.20±0.03*
t	41.674
P	<0.001

Compared with pcDNA3.1 group, \* $P < 0.05$ .

## 2.5. Effects of miR-93 inhibition on SCL-1 cell proliferation and migration

The level of expression miR-93 for SCL-1 cells was lower in the anti-miR-93 group than that in the anti-miR-NC group ( $P < 0.05$ ) indicating the effective transfection of the miR-93 inhibitor and a

Table 5: Effects of miR-93 inhibition on SCL-1 cell proliferation and migration ( $\bar{x} \pm s, n=9$ )

Group	miR-93	P21	E-cadherin	G0 (%)	S (%)	G1 (%)	Viability (%)	Number of migrating cells
anti-miR-NC	0.99±0.04	0.19±0.02	0.23±0.03	30.49±2.12	36.22±2.68	33.29±2.41	100.02±6.56	114±7.38
anti-miR-93	0.24±0.02*	0.52±0.03*	0.63±0.04*	41.16±3.34*	23.58±2.07*	35.26±3.22	64.51±4.98*	66±6.03*

suppression of the cell expression of miR-93. The survival ratio of the SCL-1 cells and the immigrant cell count and S cell count is smaller compared with the  $P < 0.05$  cell count. anti-miR-NC Group, while the proportion of cells in G0 and levels of expression were higher in anti-miR-93 (Figure 3 and table 5). The surviving rate of SCL-1 cells and the number of immigrant cells and the percentage of cells in S ( $P < 0.05$ ) were lower compared to those in the anti-miR-NC Group, while the proportion of cells in G0 and levels of expression were higher in anti-miR-93 (Figure 3 and table 5).

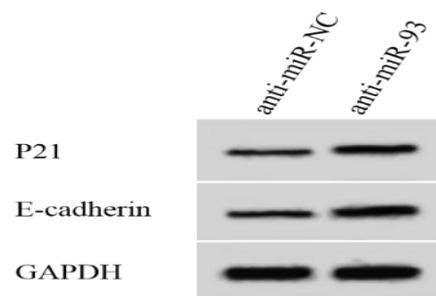


Figure 3: P21 and E-cadherin protein expressions.

t	50.312	27.458	24.000	8.092	11.198	1.4694	12.935	15.110
P	<0.001	<0.001	<0.001	<0.001	<0.001	0.1611	<0.001	<0.001

Compared with anti-miR-NC group, \*P<0.05.

## 2.6. Overexpression of miR-93 attenuated the effects of LINC00261 overexpression on SCL-1 cell proliferation and migration

Compared with those in pcDNA3.1-LINC00261 + miR-NC group, the survival rate of SCL-1 cells, the number of migrating cells and the percentage of In the S phase cell (P<0.05) the percentage was elevated while the pcDNA3.1-LINC00261+miR-93 cells were lowered in the G0 and p21 and e-cadherin (Figure 4 and Table 6) expression stages.

Figure 4. P21 and E-cadherin protein expressions.

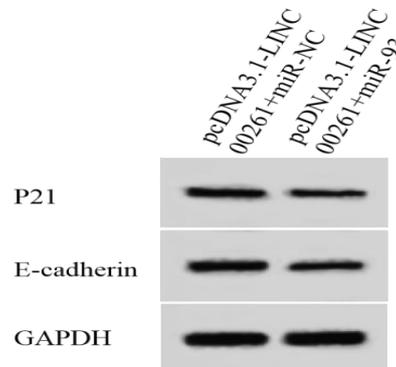


Table 6: Overexpression of miR-93 attenuated the effects of LINC00261 overexpression on SCL-1 cell proliferation and migration (  $\bar{x} \pm s$ , n=9)

Group	miR-93	P21	E-cadherin	G0 (%)	S (%)	G1 (%)	Viability (%)	Number of migrating cells
pcDNA3.1-LINC00261 + miR-NC	1.00±0.05	0.60±0.04	0.74±0.05	45.59±3.36	19.26±1.16	35.15±2.95	53.53±4.16	57±5.62
pcDNA3.1-LINC00261 + miR-93	4.16±0.07*	0.28±0.02*	0.35±0.03*	34.18±2.61*	30.95±2.34*	34.87±3.11	89.16±5.74*	103±6.38*
t	110.203	21.466	20.065	8.045	13.428	0.1960	15.078	16.231
P	<0.001	<0.001	<0.001	<0.001	<0.001	0.8471	<0.001	<0.001

Compared with pcDNA3.1-LINC00261 + miR-NC group, \*P<0.05.

## 4. Discussion

LncRNAs is a class of small non-coding RNAs, over 200 bp long, that exist widely in eukaryotes and participate in tumor development processes (Zhu D. et al., 2020). LINC00261 is a kind of LncRNA that has recently been discovered. In the case of lung noncellular cancer, the expression of LINC00261 has been downregulated, and patients with low LINC00261 exposure have a bad prognosis, according a report from Shi et al. (Shi J.L. et al., 2019). LINC00261 overexpression can also suppress the proliferation and invasion of tumor cells and induce apoptosis. LINC00261 also increased the SFRP2 expression by removing the Wnt signaling route through competitive adsorption of miR-522-3P. It was also a possible molecular target for non-small cell lung cancer treatment. The research by Yan et al. (Yan D.S. et al. 2019) has shown that LINC00261 expression has decreased in colon cancer cells and over-expressed LINC00261 has inhibited the growth, colonization, migration and invasion of colon cancer cell and encouraged the expression of miR-

324-3P by decreasing cell apoptose. LINC00261 is currently not known about its effect and mechanism on the malignant biological conduct of cells in CSCC.

The results of this study revealed that the expression level of LINC00261 in CSCC tissues is evidently lower than that in normal skin tissues, suggesting that LINC00261 may participate in the occurrence and development of CSCC as a tumor suppressor gene. After transfection of LINC00261 overexpression vector into CSCC SCL-1 cells, the cell proliferating and migratory abilities were decreased, and the cell cycle progress was blocked, implying that overexpression of LINC00261 can inhibit the proliferation and migration of CSCC cells, and that LINC00261 may be a potential molecular target for the treatment of CSCC. P21 is a tumor suppressor, and its increased expression inhibits the proliferation of tumor cells (Li X.J. et al., 2018). E-cadherin is a cell adhesion factor, and the loss or down-regulation of its expression can decrease cell adhesion, making the cells easy to fall off and migrate, and thus leading to tumor invasion and

metastasis (Peng S.Y. et al., 2018). According to the results of this study, overexpression of LINC00261 could promote the expressions of p21 and E-cadherin protein in SCL-1 cells, indicating that LINC00261 may repress the development of CSCC by influencing the expressions of p21 and E-cadherin protein.

LncRNAs can adsorb miRNAs competitively, controlling the expression of miRNA aim genes and thus affecting disease growth. The targeted binding of LINC00261 to miR-93 has been checked by a two-fold luciferase reporter test to further explore LINC00261 's mechanism inhibiting CSCC growth, and cells with LINC00261 overexpression have been detected at the level of miR-93 expression. The results showed that LINC00261 over-expression inhibited miR-93 expression in cells, which supported the targeting of LINC00261 and negative regulation of MIR-93 expression in the SCL-1 cells of the CSCC. A research has shown that miR-93 expression is up-regulated in squamous cell carcinoma of oenophagus and miR-93 can be used to treat oenophagous squamous cell carcinoms as a possible bio-brand (Ansari M.H. et al., 2016). According to other studies, miR-93 expression is regulated in three-fold negative breast cancer tissues, and miR-93 overexpression can enable breast cancer MCF-7 cells to spread, invade and migrate (Hu J.H. et al. 2014). MiR-93 actually does not affect the biological malignancies of CSCC. The results of this study show that inhibiting miR-93 expression can reduce the proliferation and migration of CSCC SCL-1 cells and block the processes of the cell cycle and indicate that miR-93 is involved in the oncogene production and development of CSCC, and that targeted down-regulation of miR-93 expression can delay the CSCC. Results of this study also demonstrated that miR-93 over-expression decreased the inhibitory effect on the proliferation, migration of SCL-1 cells and their ability to obstruct the cell cycle, which suggests that CSCC over-expression could be inhibited by targeted miR-93 down-regulation.

LINC00261 has been reduced to the level of expression in CSCC tissues. The over-expression of LINC00261 will inhibit proliferation and CSCC cell migration by decreasing the miR-93 expression and block cell cycle progression. A new target for the targeted molecular treatment of CSCC could be the LINC00261 / miR-93 axis.

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