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Long non-coding RNA linc01433 promotes tumorigenesis and progression in esophageal squamous cell carcinoma by sponging miR-1301

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Abstract. – OBJECTIVE: Long non-coding RNAs (lncRNAs) have emerged as pivotal participants of various tumors. This manuscript focuses on the function of lncRNA linc01433 (linc01433) in esophageal squamous cell carcinoma (ESCC) development.

PATIENTS AND METHODS: Quantitative Real Time-Polymerase Chain Reaction (qRT-PCR) was performed to detect expressions of linc01433 and microRNA-1301 (miR-1301) in ESCC tissues and cells. Cell counting kit-8 (CCK-8) and colony formation assays were used to verify proliferative ability changes in ESCC influenced by linc01433 and miR-1303. Wound healing and transwell assays were chosen to determine migratory ability in ESCC cells.

RESULTS: Linc01433 was abnormally up-regulated in ESCC tissues and cells. High level of linc01433 was positively correlated with tumor size and lymph node metastasis in ESCC patients. Up-regulation of linc01433 promoted cell proliferation and migration. MiR-1301 was a potential target of linc01433, and its level was negatively regulated by linc01433. MiR-1301 was responsible for linc01433-regulated proliferation and migration of ESCC.

CONCLUSIONS: Linc01433 participated in ESCC progression by regulating miR-1301 and it could function as a novel biomarker in ESCC diagnosis and treatment.

Key Words:
Linc01433, Proliferation, Migration, ESCC.

Introduction

As one of the digestive tract tumors, the incidence and mortality of esophageal cancer (EC) remain high[1,2]. EC is divided into two types according to pathological features: esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC)[3]. The 5-year survival rate of EC patients is low, and patients are often in the middle and late stages of EC when they are diagnosed[4]. At present, surgical resection is still the main method for the treatment of esophageal cancer, supplemented by radiotherapy, chemotherapy, and targeted therapy[5]. Therefore, developing diagnostic biomarkers for detecting ESCC as early as possible is imminent.

Long non-coding RNAs (lncRNAs), a subgroup of RNA molecules, are longer than 200 nucleotides with limited protein-coding ability[6]. LncRNAs work as a major part in the development and progression of various cancers[7-9]. Dysregulation of lncRNA in cancer contributes to the regulation of epigenetics, DNA damage, cell cycle regulation, microRNAs mediation, signal transduction pathways, and hormone-induced cancer[10,11]. Shen et al[12] have presented that lncRNA could serve as a cancer-promoting gene or tumor-suppressor gene in ESCC. In ESCC, lncRNA FAM201A mediated radiosensitivity by modulating ATM and mTOR expression *via* miR-101[13]. Linc00675 inhibits tumorigenesis and EMT *via* repressing the Wnt/β-catenin signaling[14].

It has been reported that the long intergenic non-protein-encoding RNA 1433 (linc01433) is involved in the development and progression of lung cancer, hepatocellular carcinoma, and breast cancer[15-17]. However, little is known whether it is a regulator of ESCC progression. In this manuscript, we demonstrated that linc01433 was up-regulated in ESCC tissues and cell lines, and linc01433 overexpression was related to tumor size and lymph node metastasis. In addition, linc01433 had the ability to promote cell proliferation and migration. MiR-1301 may be a potential target of linc01433 to regulate ESCC progression.

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Patients and Methods

Tissues and Clinical Data

With the approval of the Third Affiliated Hospital of Soochow University Ethics Committee and the written informed consent of all ESCC patients, we collected 40 pairs of ESCC tissues and adjacent esophageal tissues from patients who underwent radical surgery at The Third Affiliated Hospital of Soochow University between 2017 and 2019. No patients received radiotherapy or chemotherapy before surgery. All samples were quickly frozen in liquid nitrogen immediately after removal and stored at -80°C for follow-up study.

Cell Lines

Human esophageal epithelial cells (HEEC) and ESCC cell lines (Eca109, KYSE70, and TE-1) were purchased from the American Type Culture Collection (ATCC; Manassas, VA, USA). All cells were cultured at 37°C with 5% CO_2 in Roswell Park Memorial Institute-1640 (RPMI-1640; Invitrogen, Carlsbad, CA, USA) supplemented with 10% fetal bovine serum (FBS; Invitrogen, Carlsbad, CA, USA), 1000 U/mL penicillin and 100 mg/mL streptomycin.

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

Total RNA was extracted from ESCC tissues and cells using TRIzol reagent (Invitrogen, Carlsbad, CA, USA). Cytoplasmic RNA and nuclear RNA were separated and purified using RNAiso Blood (TaKaRa, Otsu, Shiga, Japan). Complementary deoxyribose nucleic acid (cDNA) was synthesized using a reverse transcriptase kit (Applied Biosystems, Foster City, CA, USA), following the manufacturer's instructions. 1 μL of cDNA was taken for RT-PCR amplification. The reaction conditions were 95°C for 10 s, 95°C for 5 s, and 65°C for 35 s for a total of 45 cycles. Glyceraldehyde 3-phosphate dehydrogenase (GAPDH) and U6 were used as internal controls. Primer sequences used in this study were as follows: linc01433, F: 5'-CCAGGAACGTCCGGCAACAC-3', R: 5'-GGCTATGGCTAGTCCGAATGA-3'; miR-1301, F: 5'-GCTGTCCAATCATGGCTCACTAG-3', R: 5'-GATCCGTGATGGAAGAG-3'; U6: F: 5'-CTCGCTTCGGCAGCACA-3', R: 5'-AACGCTTCACGAATTTGCGT-3'; GAPDH: F: 5'-CGCTCTCTGCTCCTCCTGTTC-3', R: 5'-ATCCGTTGACTCCGACCTTCAC-3'.

Transfection

Lentivirus-linc01433, linc01433 small hairpin RNA (sh-linc01433), miR-1301 mimics, miR-1301 inhibitor, and negative control were obtained from GenePharma (Shanghai, China). Lv-linc01433, sh-linc01433, and NC were transfected into ESCC cells by Lipofectamine 3000 (Invitrogen, Carlsbad, CA, USA), as the manufacturer's required. Stably transfected cells were screened with puromycin (2 μg/mL). MiR-1301 mimics, miR-1301 inhibitor, and NC were also transduced into ESCC cells by Lipofectamine 3000 (Invitrogen, Carlsbad, CA, USA).

Cell Counting Kit-8 (CCK-8) Assay

Transfected cells at a density of 1500 cells/well were seeded in 96-well plates (Corning, Corning, NY, USA). Before incubation for 2 h, CCK-8 solution (Promega, Madison, WI, USA) was added to each well in accordance with the manufacturer's instructions. The OD (optical density) value at 450 nm was measured after 2 h each day.

Colony Formation Assay

The stably transfected ESCC cells were inoculated in 6-well plates (Corning, Corning, NY, USA) at 1000/well. After the cells were attached, the culture medium was regularly replaced. After 14 days, cells were washed twice with phosphate-buffered saline (PBS; Beyotime, Shanghai, China) and stained with 0.1% crystal violet solution (Beyotime, Shanghai, China). The number of colonies which were confirmed as >50 cells/colony, was counted.

Wound Healing Assay

Transfected cells were incubated in a 6 cm culture dish (Corning, Corning, NY, USA) to achieve 95% confluence and the cell monolayer was scratched with a 200 μL pipette tip in a straight line. Cells were washed twice with PBS. After 0 and 48 h, the distance between wounds was measured on the side.

Transwell Assay

The migratory ability was evaluated using transwell chambers (BD Biosciences, Franklin Lakes, NJ, USA). Transfected cells (5×10^4) suspended in 300 μL serum-free medium were seeded into the upper chamber, and the bottom chamber was filled with 10% FBS. Thirty-six hours later, cells that migrated across the membrane were stained with crystal violet (Beyotime, Shanghai, China) and the cell number was counted under microscope.

Dual-Luciferase Reporter Assay

After being synthesized, the linc01433 sequences containing the miR-1301 binding sites were inserted into the pGL3 luciferase reporter vector (Promega, Madison, WI, USA). A construct comprising wild-type linc01433 (wt) or mutant linc01433 (mut) and miR-1301 mimics were co-transfected into ESCC cells by Lipofectamine 3000 (Invitrogen, Carlsbad, CA, USA). After being transfected for 36 h, cells were harvested and detected using a Dual-Luciferase assay kit (Promega, Madison, WI, USA) as the manufacturer's instructions required.

Statistical Analysis

All values were expressed as means ± SD (standard deviation). Differences between two groups were compared by an unpaired Student's *t*-test. A difference was considered as statistically significance if $p<0.05$. All analyses were carried out with GraphPad Prism 7.0 (GraphPad Software, San Diego, CA, USA).

Results

Expression Level and Localization of Linc01433

To investigate whether linc01433 was dysregulated, qRT-PCR was performed to determine linc01433 expression in ESCC tissues and adjacent esophageal tissues. The expression levels of linc01433 in 40 esophageal cancer tissues were markedly higher than that of matched adjacent tissues (Figure 1A). To further examine the role of linc01433 in ESCC carcinogenesis, ESCC patients were divided into high-level group (n = 20) and low-level group (n = 20), in accordance to the median expression. The associations between the clinicopathological characteristics of ESCC patients and linc01433 expression were evaluated and shown in Table I. Similarly, the expression level of linc01433 in the esophageal cancer cell lines (Eca109, KYSE70, and TE-1) was significantly up-regulated compared to human esophageal epithelial cells (HEEC) (Figure 1B). In addition, subcellular location of linc01433

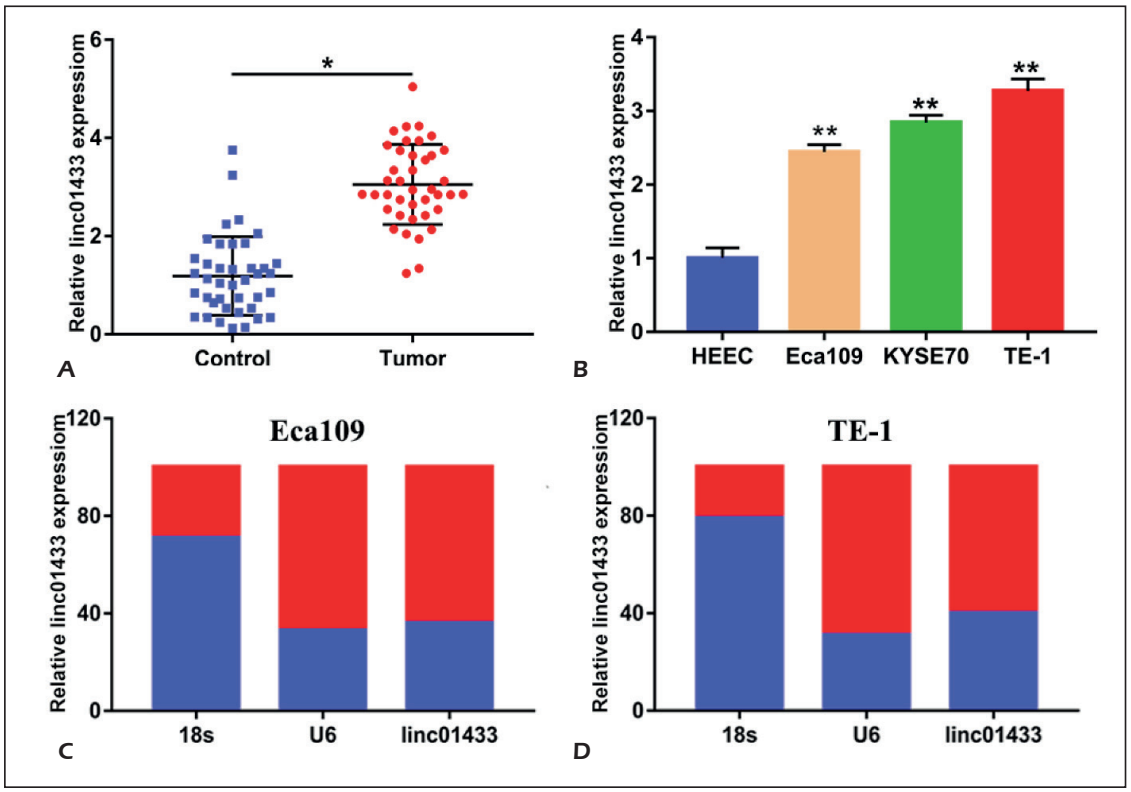

Figure 1. Linc01433 expression in ESCC tissues and cell lines. **A**, QRT-PCR analysis of linc01433 expression in 40 ESCC tissues and corresponding esophageal tissues. **B**, QRT-PCR analysis of linc01433 expression in ESCC cell lines and HEEC. **C**, QRT-PCR analysis of linc01433 expression levels in different subcellular fractions in Eca109. **D**, QRT-PCR analysis of linc01433 expression levels in different subcellular fractions in TE-1. *$p<0.05$, **$p<0.01$.

Table I. Expression of linc01433 according to patients' clinical features.

Factors	Linc01433 expression		*p*-value
	High	Low	
Gender			
Male	13	9	0.116
Female	7	11	
Age (years)			
≥50	8	14	0.112
<50	12	6	
Tumor size (cm)			
>5	14	5	0.011*
≤5	6	15	
Lymph node metastasis			
Yes	15	4	0.002*
No	5	16	

*indicates $p<0.05$ (Chi-square test)

was determined in the cells. U6 and 18s rRNA functioned as internal controls for the nucleus and cytoplasm, respectively. The expression levels of linc01433 in the nuclear and cytoplasm fractions of Eca109 and TE-1 were shown in Figure 1C and 1D. It was found that linc01433 was mainly located in the nucleus.

Linc01433 Promoted Proliferation and Migration In Vitro

To enquiry the regulation of linc01433 on the biological function of ESCC cells, cell transfection was performed. Eca109 cells were transfected with Lv-linc01433 to increase the expression of linc01433 and TE-1 cells were transfected with sh-linc01433 to antagonize the expression of linc01433. The efficiency of transfection was verified by qRT-PCR (Figure 2A). To verify the effect of linc01433 on proliferation, CCK-8 and colony formation assays were conducted. As Figure 2B suggested, after overexpression of linc01433, the proliferation rate of Eca109 cells was significantly accelerated, but the proliferation rate of TE-1 cells was slowed down after knocking down linc01433. Furthermore, the colony forming ability of Eca109 cells was also dramatically enhanced by linc01433 overexpression, while the number of colonies formed by TE-1 cells with low expression of linc01433 was significantly reduced (Figure 2C). Furthermore, overexpression of linc01433 increased migration and invasion activities (Figure 2D and 2E). Thus, these results indicated that linc01433 may regulate the ability to proliferate and migrate *in vitro*.

MiR-1301 Was a Potential Target of Linc01433

LncRNA could perform as a competing endogenous RNA (ceRNA) and overexpression of it may reverse the negative regulation between miRNAs and their target genes[18]. A potential downstream miRNA was found through the bioinformatics analysis website: miR-1301 (Figure 3A). The expression levels of miR-1301 in ESCC tissues were notably lower than that of adjacent tissues (Figure 3B). Similarly, the expression level of miR-1301 in esophageal cancer cell lines was also significantly lower than that of HEEC cell lines (Figure 3C). Given the above results, we speculated that linc01433 could sponge miR-1301. Dual-Luciferase reporter assay showed a substantial reduction in luciferase activity in the linc01433-wt and miR-1301 mimics co-transfected groups. However, co-transfection of linc01433-mut and miR-1301 mimics had no significant effect on luciferase activity (Figure 3D and 3E). In addition, a negative correlation was found between linc01433 and miR-1301 expression in human ESCC tissues (Figure 3F). The results showed that miR-1301 may be the target of linc01433.

Linc01433 Accelerated Proliferation and Migration by Regulating MiR-1301

To better study the regulatory relationship between miR-1301 and linc01433, rescue experiments were chosen to perform. As shown in Figure 4A, miR-1301 mimics were transfected into Eca109 cells and miR-1301 inhibitor was

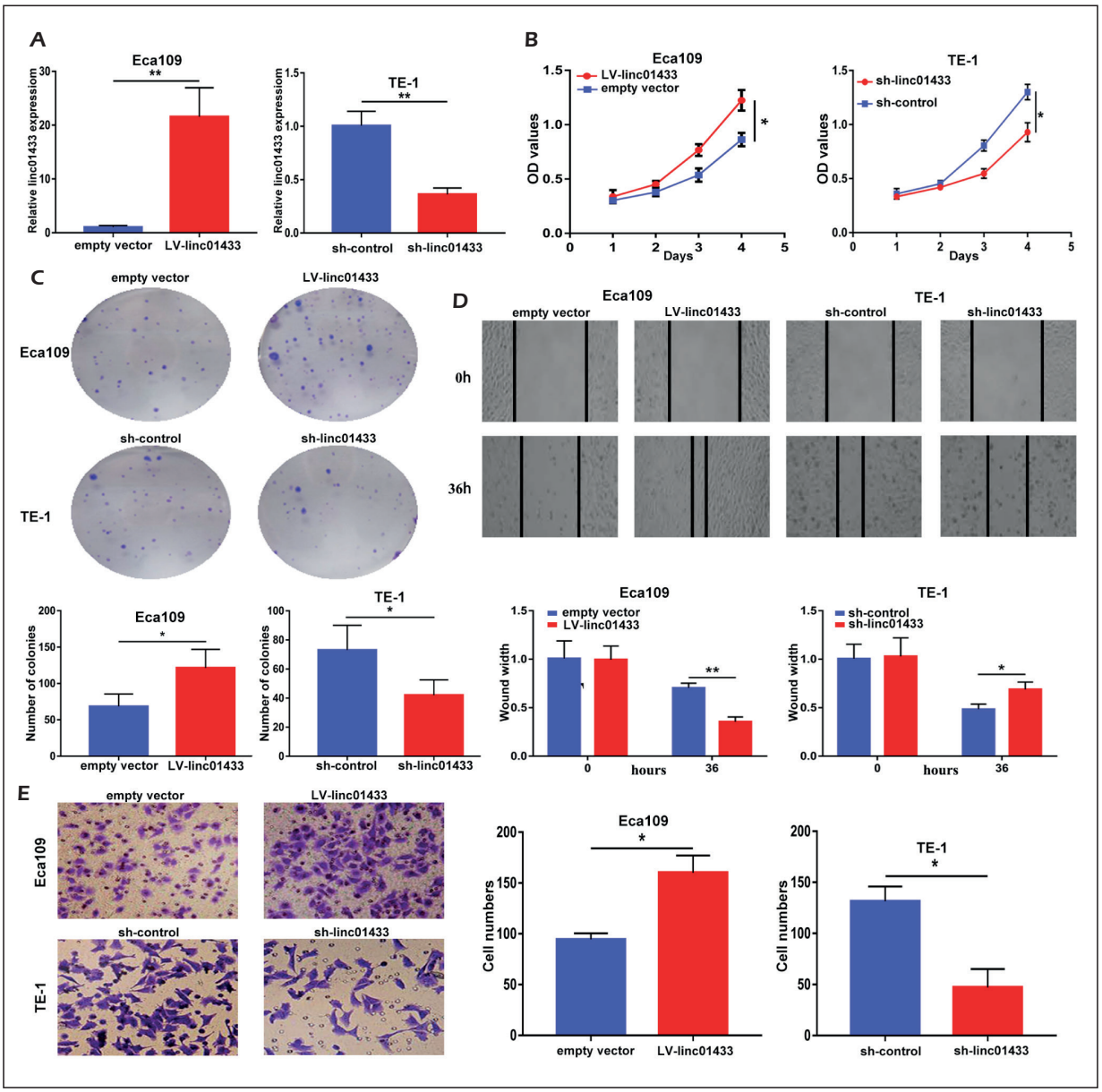

Figure 2. Linc01433 promoted cell proliferation and migration *in vitro*. **A**, Relative linc01433 expression in Eca109 and TE-1 cells after transfection. **B**, The effects of linc01433 on cell proliferation were estimated by CCK-8 assay. **C**, The effects of linc01433 on cell proliferation were detected by colony formation assay (original magnification, ×40). **D**, The effects of linc01433 on cell migration were analyzed by wound healing assay (original magnification, ×100). **E**, The effects of linc01433 on cell migration were measured by transwell assay (original magnification, ×100). *p<0.05, **p<0.01.

transfected into TE-1 cells. Through CCK-8 and wound healing assays, miR-1301 could reverse the phenomenon of enhanced proliferation and migration caused by linc01433 overexpression, and these functions were restored after knocking down miR-1301 (Figure 4B, 4C and 4D). Considering the aforementioned, it is likely that linc01433 promoted ESCC cell proliferation and migration by regulating miR-1301.

Discussion

Esophageal cancer is a serious malignancy with poor prognosis and high mortality rate[19]. Intervention strategies and efficacious therapeutic targets will be helpful to improve ESCC patients' prognosis[20]. Increasing evidence[21] has been provided to show the importance of lncRNAs in tumorigenesis and development of ESCC.

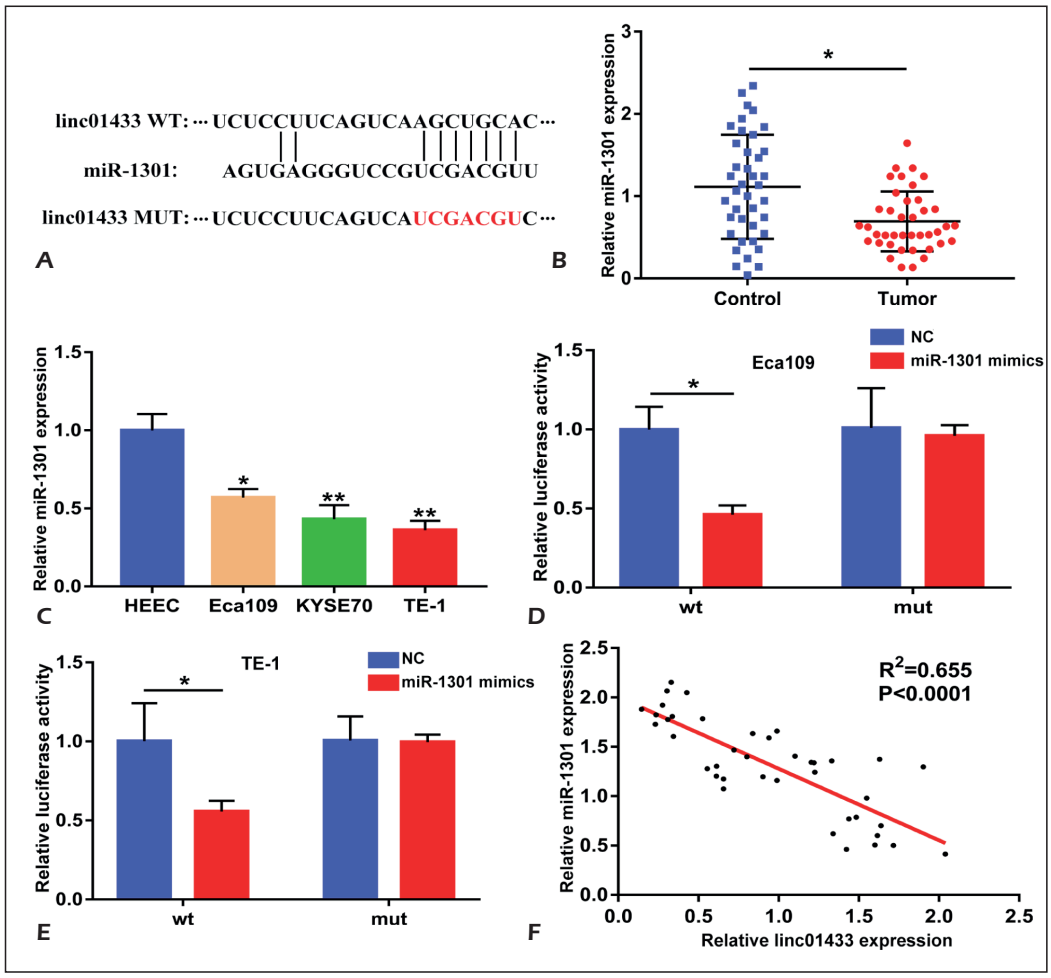

Figure 3. MiR-1301 may be a potential target of linc01433. **A**, The binding sites oflinc01433 and miR-1301. **B**, QRT-PCR analysis of miR-1301 expression in 40 ESCC tissues and corresponding esophageal tissues. **C**, QRT-PCR analysis of miR-1301 expression in ESCC cell lines and HEEC. **D**, Overexpression of miR-1301 decreased the luciferase activity of wt 3'-UTR of linc01433, while alteration of miR-1301 had no effect on the luciferase activity of mut 3'-UTR of linc01433 in Eca109 cells. **E**, Overexpression of miR-1301 decreased the luciferase activity of wt 3'-UTR of linc01433, while alteration of miR-1301 had no effect on the luciferase activity of mut 3'-UTR of linc01433 in TE-1 cells. **F**, Pearson correlation between linc01433 and miR-1301 levels. *p<0.05, **p<0.01.

LncRNA linc01433, the RNA transcripts with 682bp in length, is located on human chromosome 20p13[15]. From our current study, it was first discovered that linc01433 was abnormally up-regulated in ESCC tissues and cell lines of esophageal squamous cell carcinoma. There was a positive correlation among the high level of linc01433, ESCC tumor size, and lymph node metastasis. Therefore, we have determined the research value of linc01433 in ESCC. To find out the biological function of linc01433 in ESCC, we overexpressed and knocked down linc01433 in the ESCC cell lines. The experimental results showed that the up-regulation of linc01433 promoted cell proliferation and migration, which were weakened by knockdown of linc01433. In summary, linc01433 may be an oncogene in ESCC.

Subsequently, the downstream mechanism of linc01433 was analyzed and explored. It is well known that lncRNAs can post-transcriptionally sponge miRNAs by acting as ceRNAs[22,23]. Song et al[24] have found that lncRNA linc01535 promoted cervical cancer progression *via* targeting miR-214, while linc01551 promoted HCC cell proliferation, migration, and invasion by sponging miRNA-122-5p. MicroRNAs (miRNAs) are small noncoding RNAs, with about 22 nucleotides[25]. Recent studies have in-

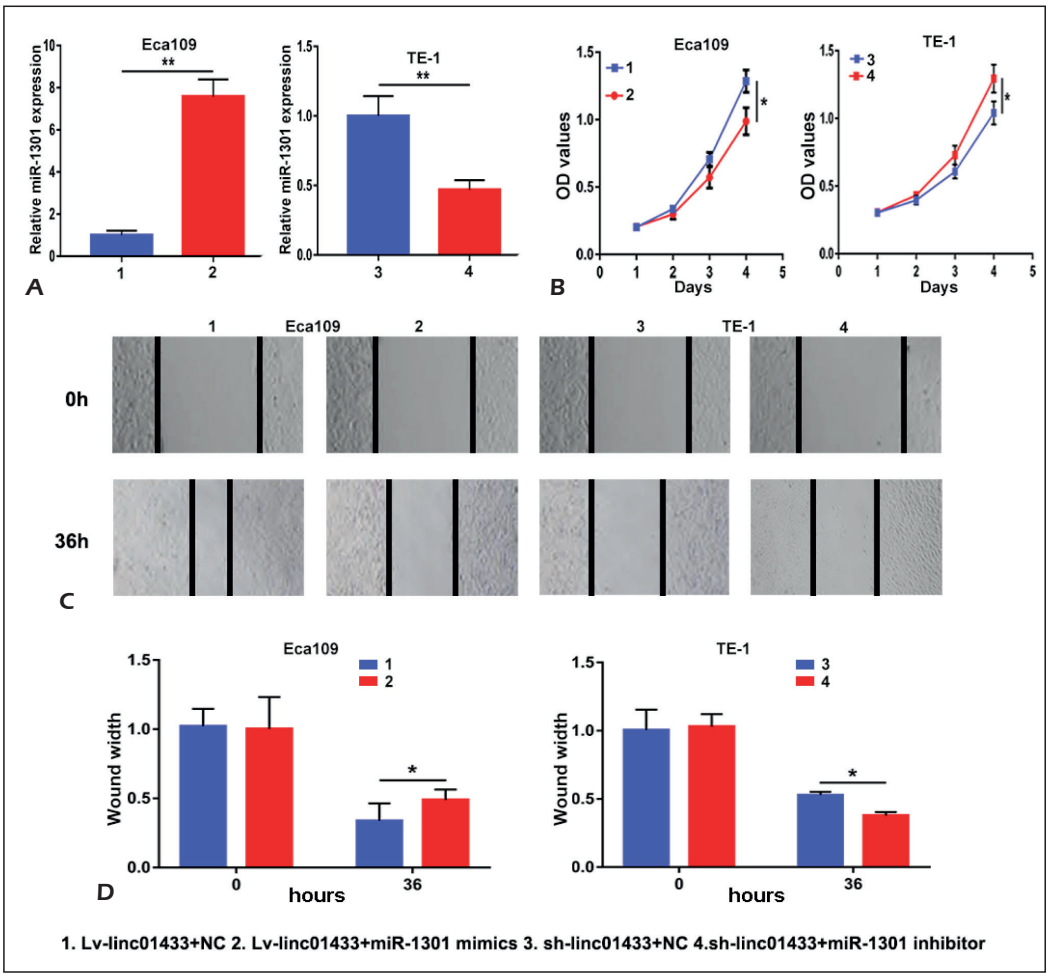

Figure 4. linc01433 promoted proliferation and migration by modulating miR-1301. **A**, Relative miR-1301 expression in Eca109 and TE-1 cells after transfection. **B**, Effects of linc01433 on cell proliferation were detected by CCK-8 assay. **C**, and **D**, The effects of linc01433 on cell migration were revealed by wound healing assay (original magnification, ×100). *p<0.05, **p<0.01.

dicated the association between MiR-1301 and tumor development. MiR-1301 could inhibit cell migration and invasion by targeting BCL9 in osteosarcoma[26]. MiR-1301 inhibits cell proliferation by regulating cell cycle and apoptosis by directly modulating ICT1 in breast cancer[27]. In this investigation, we first discovered that miR-1301 was lowly expressed in esophageal cancer tissues and cells. MiR-1301 could reverse the proliferation and migration ability caused by linc01433. These results revealed that linc01433 promoted the proliferation of ESCC cells by modulating the expression of miR-1301. Whether linc01433 could play a role in promoting cancer through other molecular mechanisms remains to be elucidated.

Conclusions

To sum up, our results demonstrated convincingly that the roles of lncRNA linc01433 in ESCC and linc01433 might function as a ceRNA to sponge miR-1301. Thus, linc01433 may serve as an innovative and prospective therapeutic target for ESCC.

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Conflict of Interests

The authors declare that they have no conflict of interests.

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