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miR-150-5p promotes the proliferation and epithelial-mesenchymal transition of cervical carcinoma cells *via* targeting SRCIN1

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ABSTRACT

Cervical carcinoma is one of the most universal cancers among women. Recent researches have reported that microRNA-150-5p (miR-150-5p) is up-regulated in diverse carcinomas containing cervical carcinoma. The purpose of this study was to further investigate the potential role of miR-150-5p in the progress of cervical carcinoma cells including proliferation and epithelial-mesenchymal transition (EMT).The ability of miR-150-5p to promote carcinogenesis was analyzed using quantitative reverse transcription polymerase chain reaction (qRT-PCR) and western blot assays, respectively. Bioinformatics analyses predicted and identified whether SRC kinase signaling inhibitor 1 (SRCIN1) was served as a potential target of miR-150-5p. C-33A and HeLa cells were utilized to determine the function of miR-150-5p through targeting SRCIN1. Among the aberrantly expressed miRNAs, miR-150-5p was significantly revealed differential expression in cervical carcinoma cell lines and was closely relevant to cell growth regulation. Furthermore, we found that SRCIN1 overexpression could obviously inhibit the proliferation and EMT of cervical cancer cells triggered by miR-150-5p mimics as well as accelerated the apoptosis of cervical carcinoma cells. In conclusion, our data demonstrated that miR-150-5p could promote the proliferation and EMT of cervical carcinoma cells *via* targeting SRCIN1. Thus, miR-150-5p may hold a promise as a prognostic biomarker and potential therapeutic target for cervical carcinoma.

1. Introduction

Cervical carcinoma is a prevalent cancer in worldwide which has a high mortality rate in women [1–3]. Several strategies, such as surgery, chemotherapy, radiotherapy and combined radio-chemotherapy, were utilized in the clinical treatment which depending on the stage of carcinomas, and overall survival rate was improved. However, the treatment failure or poor prognosis were occurred, especially in advanced stage of cervical carcinoma, owing to the development of resistance to radiotherapy and chemotherapy [4,5]. Thus, it is necessary to identify the new potential therapeutic targets for improving the treatment and prognosis of cervical carcinoma.

microRNAs (miRNAs), 19–25 nucleotides in length, are small non-coding RNA molecules that act a key part in regulating the gene expression *via* binding target mRNA at the partial sequence homology to the 3′-untranslated regions (3′-UTRs) [6,7]. These were illustrated that miRNAs took an important effect on different carcinomas, such as lung carcinoma [8,9], breast carcinoma [10,11], colorectal carcinoma [12,13] and cervical carcinoma [14–16]. microRNA-150-5p (miR-150-5p) locating in chromosome 19q13, is acted as a primary oncomiR in a large mass of carcinomas, such as leukemia [17–19] and cervical carcinomas [20]. The aberrant expression of miR-150-5p is associated with the development of carcinomas, predicting that it may be served as a valuable biomarker in the diagnosis and prognosis of cervical carcinoma.

SRC kinase signalling inhibitor 1 (SRCIN1), named as p140 Cas-associated protein (p140CAP), containing two regions of highly charged amino acids, two proline-rich regions and two coiled-coil domains [21–24], is a newly identified tumor suppressor gene that plays a vital role in tumor cells [25,26]. The silencing of SRCIN1 favors cell proliferation, motility and invasion *in vitro* as well as tumor growth *in vivo* [26]. However, the explicit molecular mechanism through which SRCIN1 contributes to the progression of cervical carcinoma remains largely unknown.

In this study, human cervical carcinoma C-33A and HeLa cells wereutilized to verify the role of miR-150-5p in cell proliferation and epithelial-mesenchymal transition (EMT) in cervical carcinoma and investigate its potential mechanism. Our results showed that miR-150-

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5p significantly promoted cervical carcinoma cell proliferation and EMT *via* targeting SRCIN1.

2. Materials and methods

2.1. Cell culture

The normal cervical carcinoma epithelial cell line End1/E6E and cervical carcinoma cell lines (C-33A, SiHa, CaSki and HeLa cell lines) were purchased from Shanghai Institutes of Biological Sciences (SIBS, China) and stored in liquid nitrogen. The cells were cultured in Dulbecco's Modified Eagle Medium (DMEM, Invitrogen, USA) supplement with fetal bovine serum (10%, FBS, Life Technologies, UK), penicillin (100 U/mL) and streptomycin (100 μg/mL) in an incubator containing 5% CO_2 in a humid atmosphere at 37 °C.

2.2. Cell transfection

The miR-150-5p mimic and miR-150-5p mimic negative control (miR-NC) were synthesized by GenePharma Co., Ltd. (Shanghai, China). SRCIN1 plasmid (EX-Y4423-M68) was purchased from GeneCopoeia (Germantown, MD, USA). The cells (3×10^5 cells/well) were inoculated into 6-well plates at 60%–70% confluence in the transfection assays, collected at 48 h post-transfection and grouped as follows: control, miR-NC and miR-150-5p mimic. The transfection was performed using Lipofectamine 2000 (Invitrogen, USA) according to the manufacturer's instruction.

2.3. Quantitative reverse transcription polymerase chain reaction (qRT-PCR)

Total RNA was extracted from the cells using TRIzol reagent with the manufacturer's protocol in this study. The RNA concentration was qualitied by Uv–vis at 280 nm. The reverse transcription (RT) and polymerase chain reaction (PCR) primers for miR-150-5p were obtained from Shanghai Sangon Biotech Co. (China), Ltd. qRT-PCR was performed with iCycler thermal cycler (Bio-Rad) using iQ SYBR Green supermix. U6 was used as an endogenous control gene for miR-150, and the primer sequences were used as follows: (1) miR-150-5p, RT: 5′-CTCAACTGGTGTCGTGGAGTCGGCAATTCAGTTGAGCACTGGTA-3′, PCR: forward: 5′-ACACTCCAGCTGGGTCTCCCAACCCTTGTACCA-3′, reserve: 5′-CTCAACTGGTGTCGTGGA-3′. (2) U6, forward: 5′-CTCGCT TCGGCAGCACA-3′, reserve: 5′-AACGCTTCACGAATTTGCGT-3′. The expression levels of miR-150 were analyzed by the $2^{-\Delta\Delta CT}$ method with GAPDH for normalization.

2.4. Western blot analysis

Total given cells were lysed in SDS sample buffer and the lysates were resolved by 10% SDS-PAGE and transferred to polyvinyldifluoride (PVDF) membranes (Millipore, USA). After blocking in PBS solution containing 0.2% Tween20 (TBS-T) and 5% nonfat dry milk for 30 min, the membranes were washed 4 times in TBS-T and incubated with primary antibodies overnight at 4 °C, including E-cadherin (1:500, Santa Cruz), ZO-1 (1:500, Cell signaling Technology, CST), vimentin (1:1000, CST), N-cadherin (1:500, CST), β-catenin (1:500, CST) and GAPDH (1:2000, CST), followed by incubation with an HRP-conjugated anti-mouse or anti-rabbit secondary antibody separately. The membranes were extensive washed and the bound antibodies were visualized using enhanced chemiluminescence (ECL kit, Pierce Biotechnology, USA) and exposed to radiography film.

2.5. Luciferase assay

The target gene of miR-150-5p was predicted with TargetScan (www.targetscan.org). Luciferase activities were investigated using the 293T cells *via* a double luciferase reporter assay system (Promega, USA) according to the manufacture's instruction with Envision Multilabel Plate Reader (Perkin-Elmer Life Sciences, USA). After seeding in 24-well plates at 2×10^4 cells/well and incubating overnight, the cells were cotransfected with miR-NC or miR-150-5p mimic using Lipofectamine 2000. Then transfecting for 48 h, the cells were obtained. All experiments of luciferase assay were repeated three times.

2.6. Cell proliferation assay

Equal numbers of the transfected human C-33A and HeLa cells were plated in a 96-well plate, and the cell proliferation *via* Colony formation assays was measured with Cell Counting Kit-8 (CCK-8, Dojindo, Japan) at 0, 24, 48 and 72 h. C-33A and HeLa cells-transfected were plated in a 6-well plate and cultured in DMEM, including in 5% CO_2 and 10% fetal bovine serum (FBS) at 37 °C for 10 days, then fixing and staining the cells by 0.1% crystal violet. The absorbance at 450 nm (A450) was measured using a microplate reader.

2.7. Transwell assay

The cell migration was investigated using transwell assay. The transfected cells were cultured in a serum-free medium at the cell density of 1×10^6 cells/mL. The upper chamber was pre-coated with 100 μL of the cell suspension, and the lower chamber was loaded with DMEM containing 10% FBS. After placing at 37 °C of 5% CO_2 for 24 h, the cells on the upper surface which did not get through the polycarbonate membrane was removed. In the upper chamber, the cells were immobilized with 4% paraformaldehyde for 30 min, stained with 1% Giemsa for 15 min, rinsed using PBS and dried in air.

2.8. Statistical analysis

In this work, the data were showed as the mean ± standard deviation (SD). The comparison of groups in differences was applied the student's *t*-test. All statistical analyses were performed with SPSS 20.0 software. $P < 0.05$ was considered a statistically significant event. Statistical analysis was performed using GraphPad Prism 6.0 (La Jolla, CA, USA).

3. Results

3.1. The characterization of miR-150-5p in cervical carcinoma cell lines

The expression levels of miR-150-5p in cervical carcinoma cell lines were analyzed by qRT-PCR in this work. As shown in Fig. 1A, the expression levels were significantly increased in human cervical carcinoma cell lines (C-33A, HeLa, SiHa and CaSki) compared with the normal cervical cancer epithelial cell line End1/E6E. It was indicated that miR-150-5p is closely correlated with cell growth in C-33A and HeLa cells. In addition, the transfection efficiency of miR-150-5p in C-33A and HeLa cells was detected using qRT-PCR as shown in Fig. 1B and C. The expression levels of miR-150-5p were increased with time-dependent behaviors at 0 h, 24 h, 48 h and 72 h post-transfection in C-33A and HeLa cells, respectively. It was the fact that miR-150-5p could play a major role in cell growth.

3.2. The effect of miR-150-5p in the proliferation and migration of C-33A and HeLa cells

The proliferation of C-33A and HeLa cells treated with miR-NC or miR-150-5p mimic were determined by CCK-8 assay. As shown in Fig. 2A and B, the results indicated that the expression of miR-150-5p increased distinctly compared with that in control and miR-NC groups in C-33A and HeLa cells, respectively. It was demonstrated that the proliferation activity of cells were increased after transfecting miR-150-

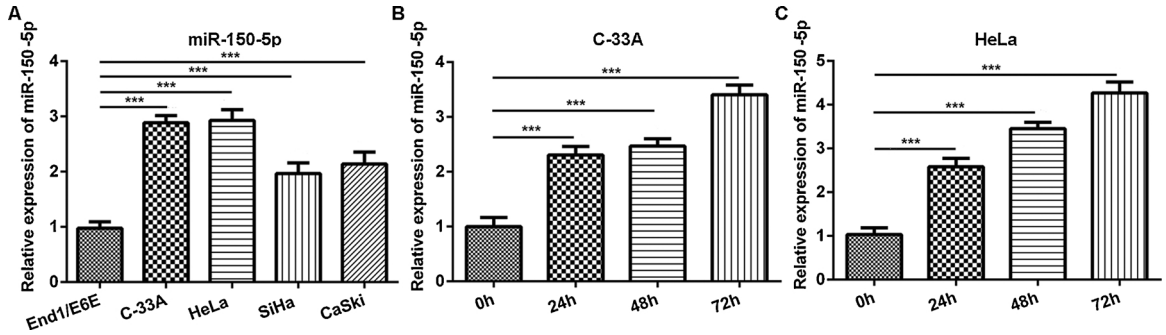

Fig. 1. The characterization of miR-150-5p in cervical carcinoma cells. (A) The expression of miR-150-5p in cervical carcinoma cell lines were analyzed by qRT-PCR, $^{***}P < 0.005$ vs. End1/E6E. C-33A (B) and HeLa (C) cells treated with miR-150-5p mimic were assessed at 0 h, 24 h, 48 h and 72 h post-transfection as follows. The levels of miR-150-5p in transfected C-33A and HeLa cells were measured at various time points using qRT-PCR. The data were presented as the means ± SD in three independent experiments, $^{***}P < 0.005$ vs. 0 h.

5p mimic. The migration of C-33A and HeLa cells were investigated using Transwell assay. These results suggested that the number of C-33A (Fig. 2C) and HeLa (Fig. 2D) cells passing through the membranes in miR-150-5p mimic group were obviously more than that in miR-NC group.

3.3. The expression of EMT markers in C-33A and HeLa cells treated with miR-150-5p

Since miR-150-5p was associated with epithelial markers in cervical carcinoma cells [27], the role of miR-150-5p were investigated in the EMT process using western blot. As shown in Fig. 3, the effect of miR-150-5p in ETM was analyzed with mesenchymal cell markers (vimentin, N-cadherin, β-catenin) epithelial cell markers (E-cadherin, ZO-1) in C-33A (Fig. 3A) and HeLa (Fig. 3B) cells. It was observed that vimentin, N-cadherin and β-catenin expression were significantly elevated after transfecting miR-150-5p, while the expression of E-cadherin and ZO-1 were blocked due to miR-150-5p post-transfection in C-33A and Hela cells, respectively. These results suggested that miR-150-5p could participate in EMT process and play a pivotal role in regulating EMT in C-33A and HeLa cells.

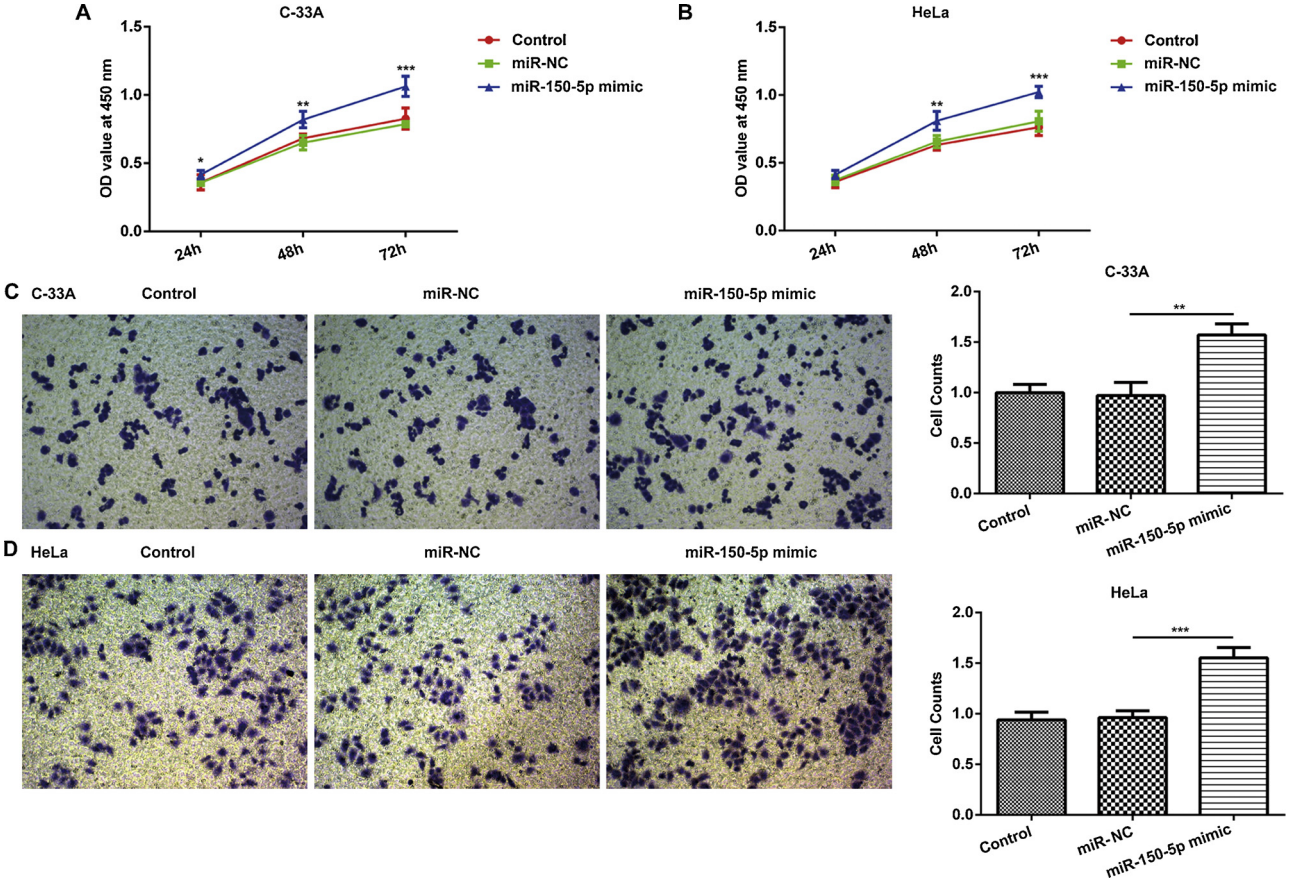

Fig. 2. The effect of miR-150-5p in the proliferation and migration of HeLa and C-33A cells. The proliferation in C-33A (A) and HeLa (B) cells treated with miR-150 mimic were measured using CCK-8 assay at 24 h, 48 h and 72 h post-transfection. Transwell assay was used to assess the migration of C-33A (C) and HeLa (D) cells. The data were presented as the means ± SD in three independent experiments, $^{*}P < 0.05$ vs. miR-NC, $^{**}P < 0.01$ vs. miR-NC, $^{***}P < 0.005$ vs. miR-NC.

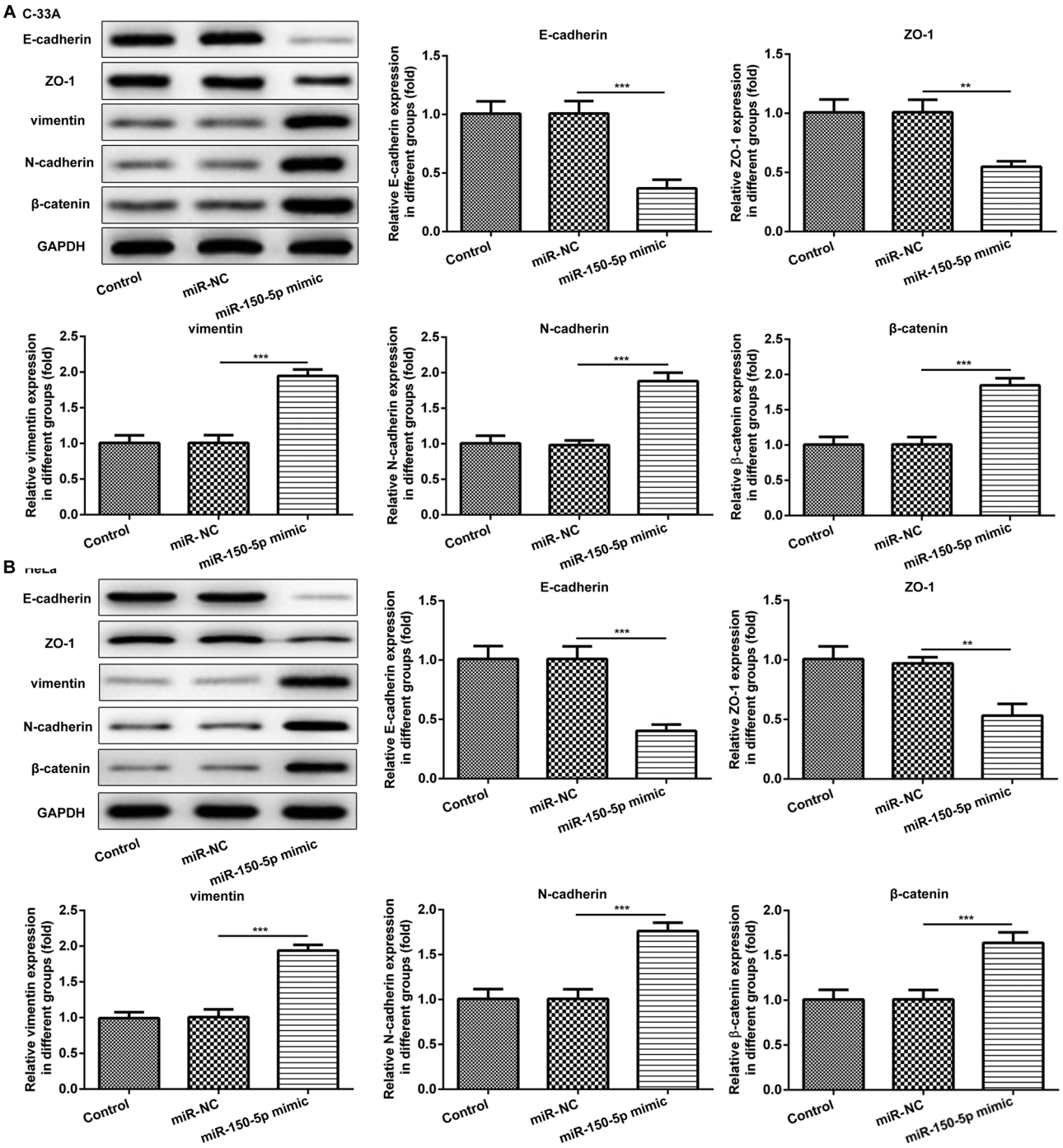

Fig. 3. The expression of EMT markers in C-33A and HeLa cells treated with miR-150-5p. (A) The expression of vimentin, N-cadherin, β-catenin, E-cadherin and ZO-1 in C-33A cells treated with SRCIN1 as determined by western blot. (B) The expression of vimentin, N-cadherin, β-catenin, E-cadherin and ZO-1 in HeLa cells treated with miR-150-5p as detected using western blot. Error bars indicate the mean ± SD. $^{**}P < 0.01$ vs. miR-NC, $^{***}P < 0.005$ vs. miR-NC.

3.4. The verification of SRCIN1 as a target gene of miR-150-5p

As the expression of target genes were often regulated by miRNAs, the target of miR-150-5p was predicted using TargetScan (Fig. 4A). In order to confirm whether SRCIN1 was a direct target gene of miR-150-5p, 3′UTR WT and 3′UTR MUT of SRCIN1 were co-transfected into 293T cells along with miR-NC or miR-150-5p mimic. As shown in Fig. 4B, the relative luciferase activity of 3′UTR WT and 3′UTR MUT of SRCIN1 were reduced in the presence of miR-150-5p, whereas the luciferase activity was distinctly decreased in the MUT binding sites. It was explained that SRCIN1 is a target gene of miR-150-5p. The transfection efficiency was investigated as shown in Fig. 4C and D. When miR-150-5p inhibitor post-transfection, the expression levels of miR-150-5p were restrained in C-33A (Fig. 4C) and HeLa (Fig. 4D) cells compared with miR-NC group. In addition, the effect of miR-150-5p mimic transfection on SRCIN1 expression in C-33A and HeLa cells was determined *via* western blot (Fig. 4E and G) and qRT-PCR (Fig. 4F and H) analysis. The results indicated that the expression of SRCIN1 with a significant decrease was attributed to miR-150-5p mimic post-transfection, and that obviously increased due to miR-150-5p inhibitor post-transfection in the C-33A and HeLa cells.

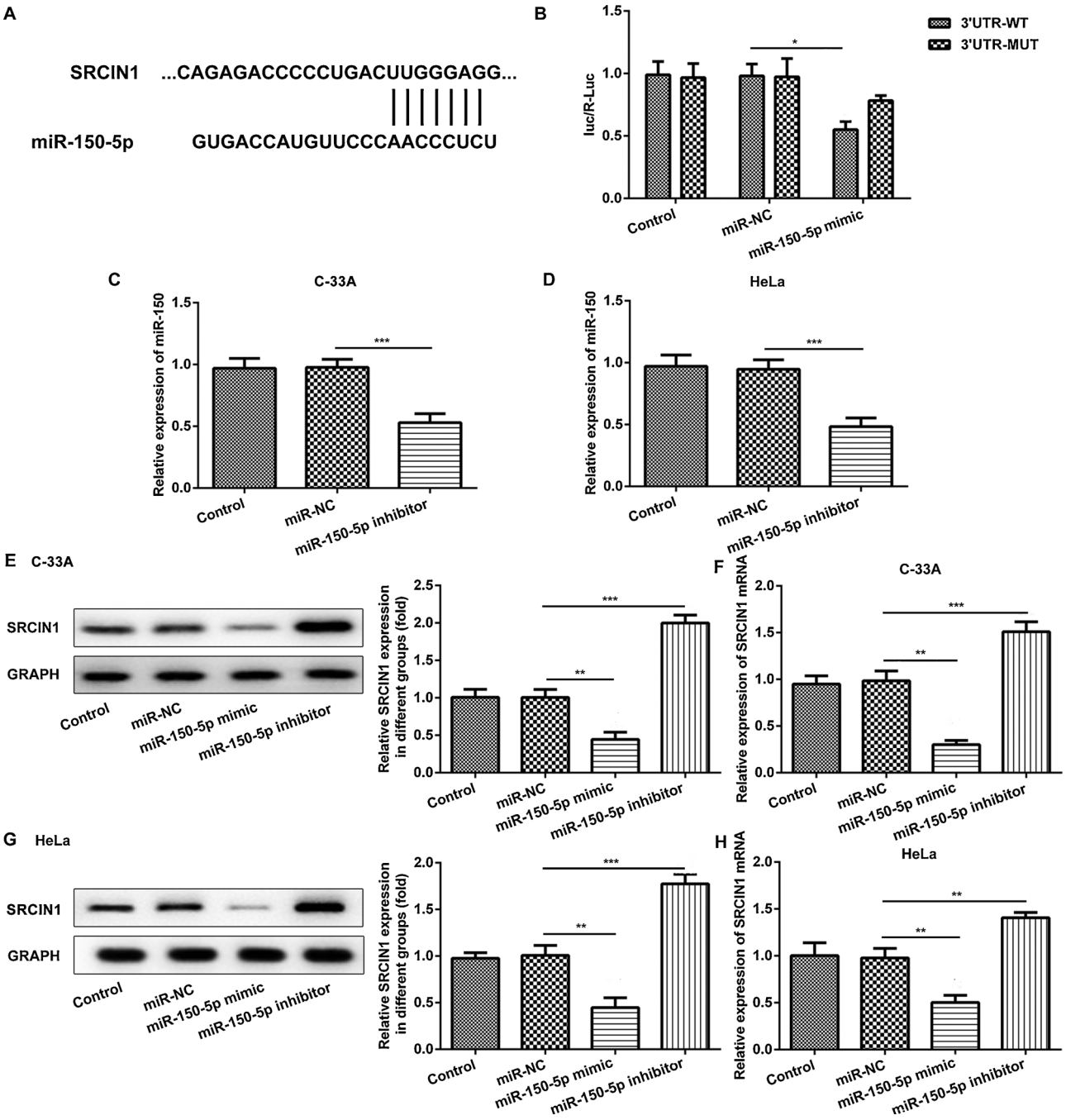

Fig. 4. The verification of SRCIN1 as a target gene of miR-150-5p. SRCIN1was predicted as a target gene of miR-150-5p using TargetScan (A) and Dual-Luciferase Reporter assay (B). The expression levels of miR-150 for transfection efficiency were analyzed by qRT-PCR in C-33A (C) and HeLa (D) cells. The expression levels of miR-150 were detected using western blot (E) and qRT-PCR (F) in C-33A cell. The expression levels of miR-150 were detected using western blot (G) and qRT-PCR (H) in HeLa cell, $^{*}P < 0.05$ vs. miR-NC, $^{**}P < 0.01$ vs. miR-NC, $^{***}P < 0.005$ vs. miR-NC.

3.5. The role of SRCIN1 in cervical carcinoma cells

To investigate the role of SRCIN1 in cervical carcinoma cells, the expression levels were detected using western blot and qRT-PCR as presented in Fig. 5. The results shown in Fig. 5A and B indicated that the expression levels of SRCIN1 were low in cervical carcinoma cell lines (C-33 A, HeLa, SiHa and CaSki) compared with the normal cervical carcinoma epithelial cell line End1/E6E. In addition, we investigated the transfection efficiency of SRCIN1 in C-33 A (Fig. 5C and D) and HeLa (Fig. 5E and F) cells utilizing western blot and qRT-PCR. Compared with miR-NC group, the expression levels of SRCIN1 were obviously increased after transfecting with SRCIN1 over-expressed plasmid. Meanwhile, we further evaluated whether the overexpression of SRCIN1 could reverse the effects of miR-150-5p using western blot and qRT-PCR. C-33 A and HeLa cells were transfected with miR-NC, miR-150 mimic, or cotransfected with miR-150-5p mimic and SRCIN1 over-expressed plasmid. It was demonstrated that the expression of SRCIN1 was significantly decreased after transfecting with miR-NC or miR-150-5p mimic. Compared with miR-150-5p mimic groups, SRCIN1 expression revealed at a high levels when the cells were cotransfected with miR-150-5p mimic and SRCIN1 over-expressed plasmid in Fig. 5G–J.

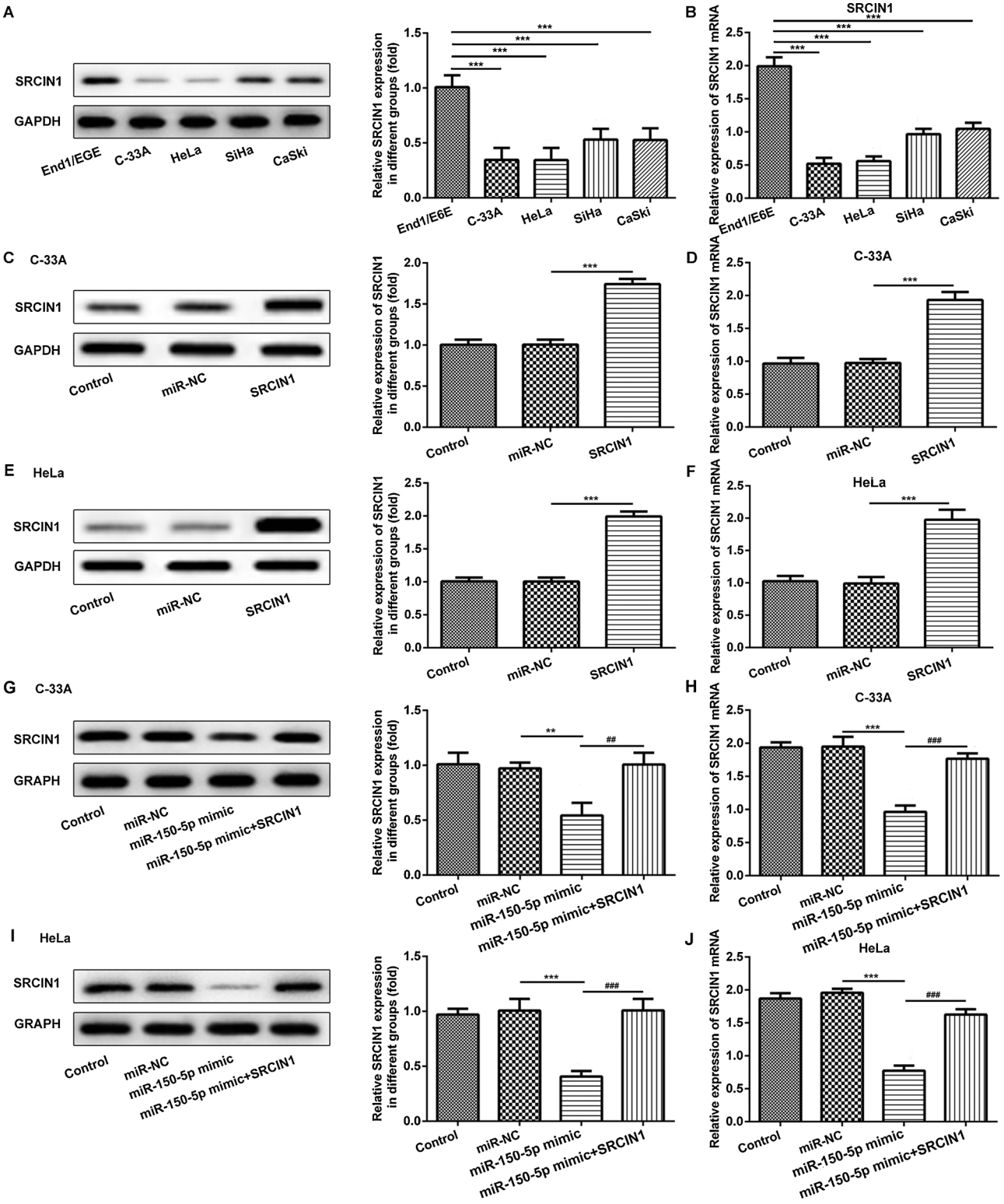

Fig. 5. The role of SRCIN1 in cervical cancer cells. The expression of SRCIN1 in cervical cancer cell lines were analyzed by western blot (A) and qRT-PCR (B), $^{***}P < 0.005$ vs. End1/E6E. The expression levels of SRCIN1 for transfection efficiency were detected using western blot (C) and qRT-PCR (D) in C-33A cell, $^{***}P < 0.005$ vs. miR-NC. The expression levels of SRCIN1 for transfection efficiency were detected using western blot (E) and qRT-PCR (F) in C-33A cell, $^{***}P < 0.005$ vs. miR-NC. The expression levels of SRCIN1 were determined *via* western blot (G) and qRT-PCR (H) in C-33A cell, $^{**}P < 0.01$ vs. miR-NC, $^{**}P < 0.005$ vs. miR-NC, $^{\#\#}P < 0.01$ vs. miR-150-5p mimic, $^{\#\#\#}P < 0.005$ vs. miR-150-5p mimic. The expression levels of SRCIN1 were determined *via* western blot (I) and qRT-PCR (J) in HeLa cell, $^{***}P < 0.005$ vs. miR-NC, $^{\#\#\#}P < 0.005$ vs. miR-150-5p mimic. The data were represented as the means ± SD in three independent experiments.

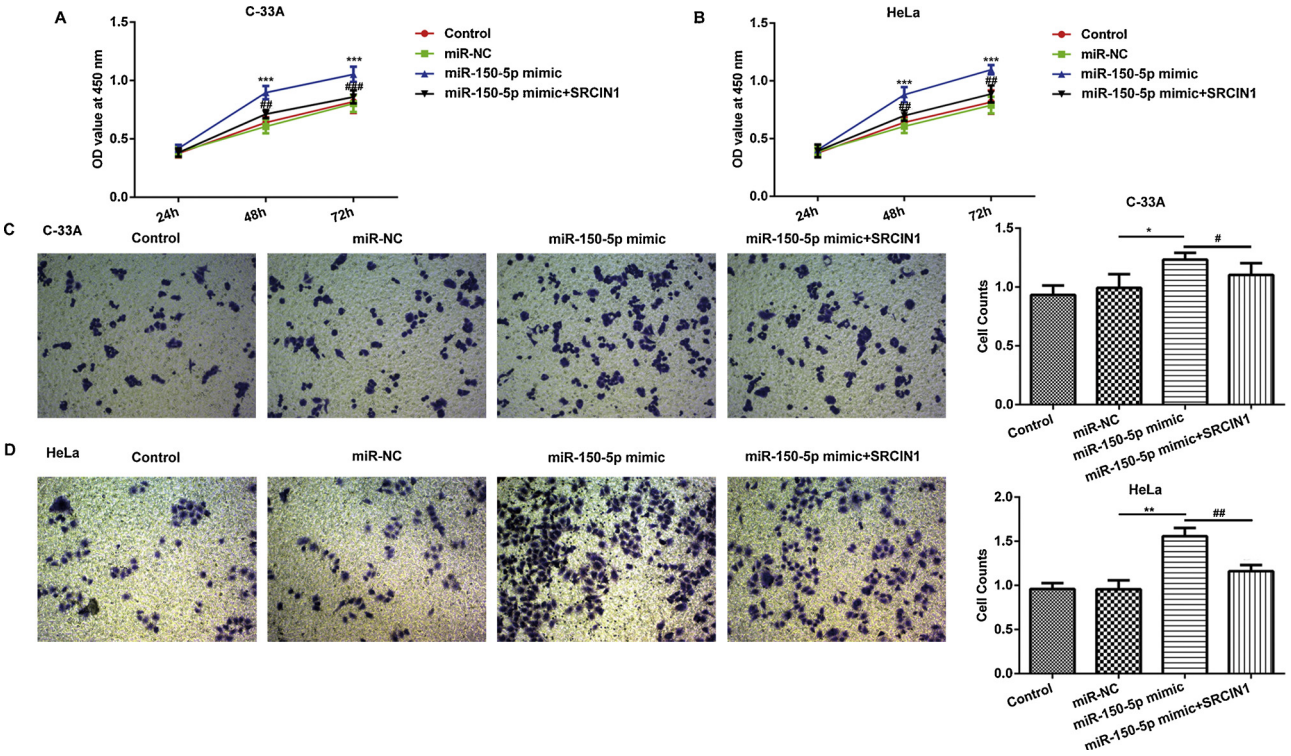

Fig. 6. The effect of SRCIN1 in the proliferation and migration of C-33A and HeLa cells. The proliferation in C-33A (A) and HeLa (B) cells treated with miR-150 mimic and SRCIN1 were measured using CCK-8 assay at 24 h, 48 h and 72 h post-transfection. Transwell assay was used to assess the migration of C-33A (C) and HeLa (D) cells. The data were presented as the means ± SD in three independent experiments. $^{*}P < 0.05$ vs. miR-NC, $^{**}P < 0.01$ vs. miR-NC, $^{***}P < 0.005$ vs. miR-NC, $^{\#}P < 0.05$ vs. miR-150-5p mimic, $^{\#\#}P < 0.01$ vs. miR-150-5p mimic, $^{\#\#\#}P < 0.005$ vs. miR-150-5p mimic.

3.6. The effect of SRCIN1 in the proliferation and migration of C-33 A and HeLa cells

The previous research has been reported that SRCIN1 is essential for the regulation of cell proliferation and migration [27]. The proliferation activity of C-33A and HeLa cells treated with miR-NC, miR-150-5p mimic, or cotransfected with miR-150-5p mimic and SRCIN1 over-expressed plasmid were determined using CCK-8 assay. The datum explained that the expression of miR-150-5p were inhibited due to SRCIN1 over-expressed plasmid post-transfection in C-33A (Fig. 6A) and HeLa (Fig. 6B) cells, respectively. As shown in Fig. 6C and D, the migration of C-33A and HeLa cells were analyzed using Transwell assay. Compared with miR-150-5p mimic group, the number of C-33A and HeLa cells passing through the membranes were visibly reduced by transfecting SRCIN1 over-expressed plasmid. Hence, the proliferation and migration of cells were inhibited because of SRCIN1 post-transfection.

3.7. The expression of EMT markers in C-33A and HeLa cells treated with SRCIN1

To investigate the effect of SRCIN1 in cervical carcinoma cells, the expression levels of EMT markers were measured *via* western blot. As shown in Fig. 7, the results were analyzed with mesenchymal cell markers (vimentin, N-cadherin, β-catenin) epithelial cell markers (E-cadherin, ZO-1) in C-33A (Fig. 7A) and Hela (Fig. 7B) cells. It was observed that vimentin, N-cadherin and β-catenin expression were obviously reduced after transfected with SRCIN1 over-expressed plasmid, while the expression of E-cadherin and ZO-1 were distinctly elevated due to SRCIN1 over-expressed plasmid post-transfection compared with miR-150-5p mimic groups in C-33A and HeLa cells, respectively. These results demonstrated that EMT in C-33A and HeLa cells were inhibited after transfecting with SRCIN1 over-expressed plasmid.

4. Discussion

Cervical carcinoma is one of the most common malignancies that seriously threatens women's life [28,29]. Latest research reported that the number of cervical cancer in female approximately amounted to 135,000 per year in China [29]. It remains a severe challenge for researchers how to identify novel and efficacious early diagnosis and treatment strategies. In this study, we analyzed a new potential therapeutic target for improving the treatment and prognosis of cervical cancer.

miRNAs, as an important post-transcriptional regulator of gene expression, play a crucial role in carcinogenic processes [30]. The related study has reported that miR-150-5p served as an important oncomiR in cervical carcinoma [31–33], and the functional studies have explained that miR-150-5p was served as an oncogene or a tumor promoter of relevant behaviors of tumors in biology [20,34]. However, the potential effects of miR-150-5p in the carcinogenesis of cervical carcinoma remain largely unknown. In consequence, the purpose of this study was to further investigate the potential roles of miR-150-5p in cervical carcinoma cell proliferation and EMT. The results showed that the expression of miR-150 in cervical carcinoma cells (C-33A and HeLa) was increased remarkably. The previous literature has presented that the growth and survival of cervical carcinoma cells were promoted in the presence of miR150-5p through targeting FOXO4 [34]. The latest protocol has presented that the proliferation, migration and invasion of cervical carcinoma were promoted with miRNA-150-5p and its target gene (PDCD4) [20]. These are consistent with our data that miR-150-5p may act a significant role in cervical carcinoma, nevertheless, the effect of miR-150-5p on cell EMT was not mentioned. The Cap family protein p140CAP, encoded by SRCIN1, functions as a tumor suppressor protein, the silencing of which promotes anchorage-independent growth of

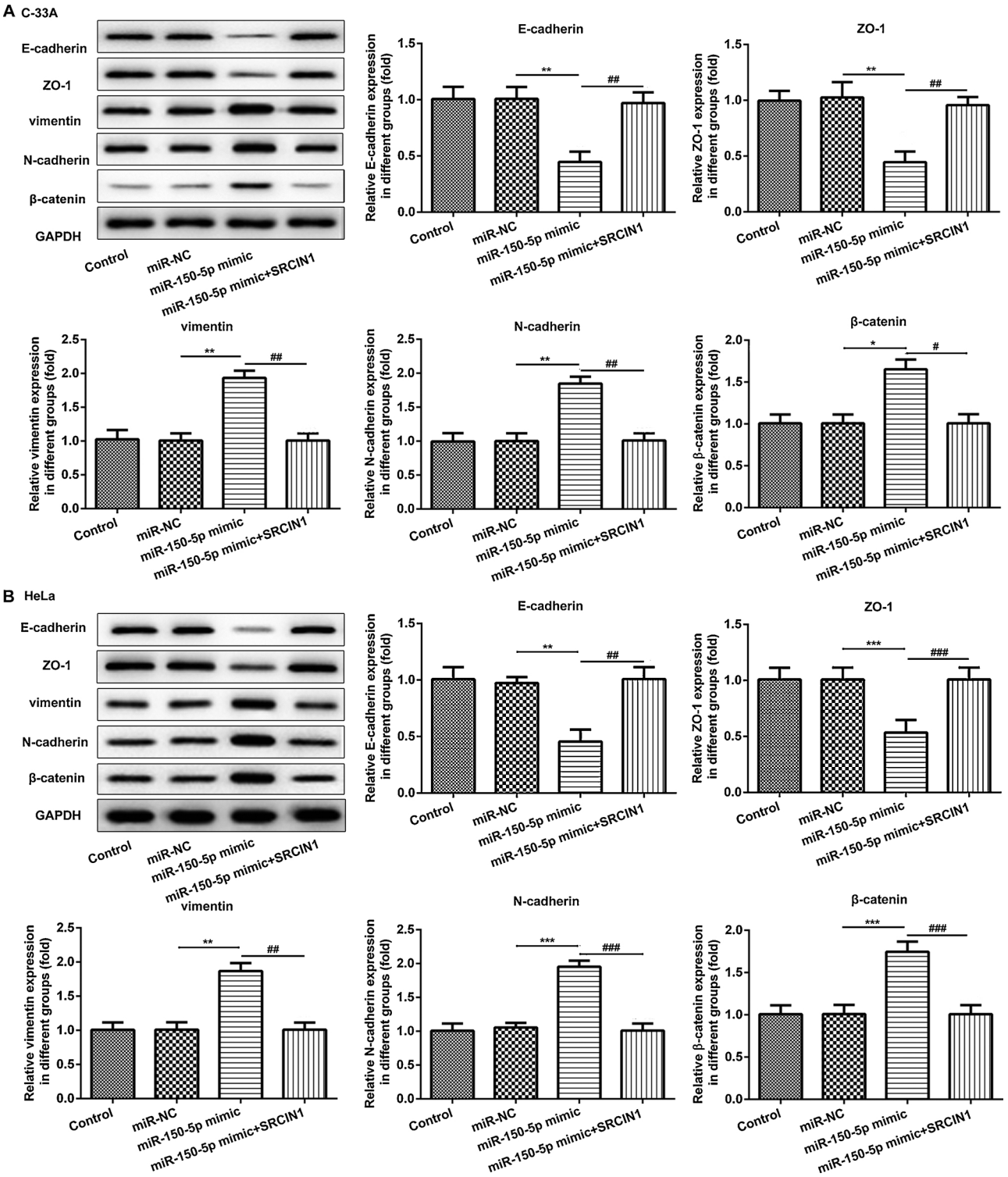

Fig. 7. The expression of EMT markers in C-33A and HeLa cells treated with SRCIN1. (A) The expression of vimentin, N-cadherin, β-catenin, E-cadherin and ZO-1 in C-33A cells treated with SRCIN1 as analyzed using western blot. (B) The expression of vimentin, N-cadherin, β-catenin, E-cadherin and ZO-1 in HeLa cells treated with SRCIN1 as detected by western blot. Error bars indicate the mean ± SD. $^{*}P < 0.05$ vs. miR-NC, $^{**}P < 0.01$ vs. miR-NC, $^{***}P < 0.005$ vs. miR-NC, $^{\#}P < 0.05$ vs. miR-150-5p mimic, $^{\#\#}P < 0.01$ vs. miR-150-5p mimic, $^{\#\#\#}P < 0.005$ vs. miR-150-5p mimic.

cancer cells and tumor development and growth. The CAP protein p140CAP can inhibit Src signaling pathways and downstream focal adhesion kinase, epidermal growth factor receptor and Ras/extracellular signal-related kinase activity, and functions as a potent tumor suppressor. Src is a cellular tyrosine kinase that is frequently overexpressed or aberrantly activated in cancer cells. Researchers have reported high levels of Src activity in cervical carcinoma cell. Src is normally maintained in an inactive state *via* the phosphorylation of Tyr527 in the carboxy-terminal of the protein. P140CAP activates the Csk kinase, which phosphorylates the negative-regulatory Tyr527 and induces a conformational change in the structure of Src. miR-150-5p, therefore, may exert its oncogenic function by targeting SRCIN1, a

concept which has been evaluated [35]. The recent study has verified that miR-150-5p, as a tumor promoter, can predict prognosis for early-stage non-small cell lung carcinoma and promote tumor cell proliferation by targeting tumor suppressor gene SRCIN1 [36], meanwhile, also promote the proliferation and migration of lung carcinoma cells *via* targeting SRCIN1 [35]. In this study, the roles of miR-150-5p and SRCIN1 in cervical carcinoma were investigated to fill the blank. However, to our best knowledge, it is unclear whether miR-150-5p and SRCIN1 are relational on cervical carcinoma cell proliferation and EMT. It is valuable to illuminate the role of miR-150-5p and SRCIN1 which are served as high sensitive and specific noninvasive biomarkers in cervical carcinoma.

The wide studies have revealed that there are multiple target genes of miR-150-5p in cancers [37–41]. It is crucial to verify the role of miR-150-5p in the pathogenesis and progress of cervical carcinoma *via* detecting target genes, which is also necessary for identifying initial targeted treatments of cervical carcinoma. In this work, we predicted and determined SRCIN1 as one of the target genes of miR-150-5p, and the overexpression of SRCIN1 could blocked the effects of miR-150-5p in cervical carcinoma. The previous studies have confirmed that SRCIN1 as a tumor suppressor gene was involved in the growth, proliferation, migration, invasion and apoptosis of tumor cells [42–44]. EMT is a biological process that the polarized epithelial cells usually interact with the basilemma through the basal surface and experience a series of biochemical changes to the mesenchymal cell phenotype. It is contributed to elevate the migratory capacity, invasiveness, resistance to apoptosis and production of extracellular matrix components [45]. It was the fact that SRCIN1 could inhibit the EMT of cells through regulating the expression of E-cadherin, ZO-1, vimentin, N-cadherin and β-catenin which are the major EMT-related genes in various cancers [46].

Herein, the effects of miR-150-5p on the proliferation and epithelial-mesenchymal transition of cervical carcinoma cells were investigated in this present study. The results illustrated that miR-150-5p was able to promote the proliferation and epithelial-mesenchymal transition of cervical cancer cells by targeting SRCIN1. Hence, miR-150-5p and its target gene (SRCIN1) may be served as a therapeutic target to improve the clinical treatment of cervical carcinoma.

Acknowledgements

Not applicable.

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