

Original Article

lncRNA SNHG17 promotes pancreatic carcinoma progression via cross-talking with miR-942

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Abstract: Objective: Long non-coding RNA (lncRNA) SNHG17 has been shown to modulate the biological behavior of multiple cancers (e.g., colorectal and lung cancers). However, its involvement in pancreatic cancer (PC) has not been explored; therefore, in the present study, we sought to examine this involvement. Methods: First, the mRNA expression levels of various genes were quantified in PC tissues and cell lines using quantitative reverse-transcription PCR (qRT-PCR). The interaction between SNHG17 and miR-942 was explored by bioinformatics prediction as well as a dual luciferase reporter assay. The proliferation and viability of pancreatic carcinoma cells were examined using cell counting kit-8 and MTT assays, respectively. Cellular migratory and invasive properties were evaluated using transwell migration and wound healing assays. Cell death was measured using flow cytometry. Protein expression was quantified by western blotting. Results: SNHG17 expression was markedly higher in human PC specimens and cell lines than in normal healthy tissues and pancreatic epithelial cells. MiR-942 expression displayed the opposite trend. Bioinformatics prediction and a dual luciferase reporter assay confirmed that SNHG17 serves as a sponge for miR-942. Loss-of-function assay revealed that SNHG17 silencing reduced the proliferation and viability of PC cells, impaired their migratory and invasive capacities, and led to their apoptosis. All these changes could be reversed by miR-942 inhibition. Further mechanical studies showed that SNHG17 silencing decreased the expression of several tumor modulators, including XXX, and this decrease was countered by miR-942 inhibition. Conclusion: Our study provides experimental evidence for an interaction between SNHG17 and miR-942, which may unveil a new approach for PC pharmacotherapy.

Keywords: PC, SNHG17, miR-942, tumor phenotypes, proliferation, invasion

Introduction

Pancreatic cancer (PC) has a five-year survival rate of approximately 2-9% and is recognized worldwide as a fatal cancer, ranking the 7th most common cause of cancer-associated deaths [1, 2]. China has witnessed a rapid growth in PC incidence over the past few decades [3, 4]. Although the pathogenesis of PC has been extensively studied, its prognosis remains poor, mainly because of the lack of specific symptoms and curative methods and delayed diagnosis [5, 6]. Therefore, identifying new PC biomarkers could facilitate the establishment of more accurate and reliable PC therapeutic options.

Long non-coding RNAs (lncRNAs) influence tumorigenesis through various strategies [7]. lncRNA SNHG17 may be involved in the regula-

tion of many malignant neoplasms [8-10]. In human glioma specimens and glioma cell lines, the SNHG17 expression level is increased. SNHG17 knockdown attenuates cellular proliferation, promotes apoptosis, and represses tumor growth [11]. SNHG17 expression is upregulated in PC, and its knockdown reduces the proliferation, invasion, epithelial-to-mesenchymal transition (EMT), and migration of cancer cells, while increasing their apoptosis [12]. This lncRNA is also upregulated by STAT3 in ovarian cancer, in which a positive link between high SNHG17 expression and poor prognosis has been established. Notably, functional analysis has shown that SNHG17 silencing can inhibit ovarian cancer growth [13]. Nonetheless, the role of SNHG17 in PC remains unknown.

lncRNAs can modify the expression of microRNAs (miRNAs) by acting as competing endoge-

nous RNAs (ceRNAs) and further influence the pathogenesis of many disorders [14]. In the present study, we searched for predicted sponging miRNAs of SNHG17 using the Starbase database. One such predicted miRNA was miR-942, which may be associated with many cancers, such as lung cancer [15], gastric cancer [16], breast carcinoma [17], prostate carcinoma [18], and hepatocellular carcinoma [19]. Notably, cancer growth may be suppressed by miR-942. We, therefore, hypothesized that the interaction between SNHG17 and miR-942 may influence PC development.

To this end we sought to elucidate the mechanism through which SNHG17 and miR-942 contribute to PC progression. We thus aimed to determine whether SNHG17 and miR-942 can modulate the viability, apoptosis, migration, and invasion of PC cells. Our results could benefit the search for potential PC biomarkers.

Materials and methods

Ethical statement

This study was approved by the Ethics Committee of Shanghai Ruijin Hospital and conducted in accordance with the Declaration of Helsinki. All patients provided written informed consent before the study was conducted. Animal experiments complied with the guidelines formulated by the Animal Ethics Committee. All possible measures were taken to minimize animal suffering.

Clinical samples

Subjects (n = 30; 12 men and 18 women; age range, 45.5-71.1 years; median age, 58.9 years) were selected from pathologically and clinically diagnosed PC patients who underwent primary resection at the Ruijin Hospital between November 2015 and December 2019. Patients who were treated with radiotherapy or chemotherapy before surgery were excluded. Relevant clinical data were collected. Healthy non-cancerous pancreatic tissue was obtained from volunteers (n = 30, normal group) for use in this study. Clinicopathological classification and stage of tissues were determined according to the current National Cancer Center Network (NCCN), Union for International Cancer Control (UICC), and Tumor-Node-Metastasis (TNM) classification.

Cell culture and transfection

PANC-1 (ATCC) and AsPC-1 (ATCC) cells were cultured in Dulbecco's modified Eagle's medium (DMEM, Gibco) supplemented with penicillin/streptomycin (1%, Gibco), glutamine (1%), and fetal bovine serum (FBS, 10%, Gibco). HPDE6-E6E7 clone 6 cells (HPDE6) are human pancreatic duct epithelial cells that were used as a control. HPDE6 cells were also cultured in DMEM supplemented with penicillin/streptomycin (1%, Gibco), glutamine (1%), and fetal bovine serum (FBS, 10%, Gibco).

According to the manufacturer's instructions, SNHG17 shRNA, shRNA negative control (NC), miR-942 inhibitor, and inhibitor NC were used to transfect each cell type using Lipofectamine RNAiMAX (Thermo Fisher).

Quantitative reverse-transcription PCR (qRT-PCR)

Total RNA was extracted from cells or specimens (100 mg) using TRIzol reagent (Invitrogen), and the concentration of the extracted RNA was determined using Nanodrop2000 (OD260) (Thermo Fisher Scientific). RNA was reverse transcribed to complementary DNA (cDNA) using a Hieff First Strand cDNA Synthesis Super Mix for qRT-PCR kit (Yeasen Biotech, Shanghai, China) for miRNA and mRNA detection. Gene qPCR detection was performed using relevant kits, and all procedures were conducted in accordance with the manufacturer's instructions. The reaction procedure was as follows: denaturation for 10 min at 95°C, and 40 cycles of denaturation for 15 s at 95°C, annealing for 15 s at 37°C, and extension for 40 s at 60°C. Glyceraldehyde-3-phosphate dehydrogenase or U6 mRNA served as internal controls. The mRNA expression levels of the genes in this study were calculated using the $2^{-\Delta\Delta CT}$ method. All reactions were set in triplicate.

Western blotting

To extract total protein, cells were lysed in radioimmunoprecipitation assay buffer supplemented with protease inhibitor cocktail (Roche Applied Science, Penzberg, Germany). The protein concentration was quantified using BCA kits. Proteins were subsequently loaded onto sodium dodecyl sulfate-polyacrylamide gels for

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electrophoresis and transferred to polyvinylidene fluoride membranes (Millipore, MA). The membranes were then incubated overnight at 4°C with primary antibodies. Bands were probed immunologically using PPAR gamma antibody (ab59256, Abcam), VEGF antibody (ab46154, Abcam), ZEB1 antibody (ab124512, Abcam), BMP antibody (ab14933, Abcam), ISG12a antibody (ab171919, Abcam), GDNF antibody (ab18956, Abcam), and Actin antibody (ab8227, Abcam), and then washed with tris-buffered saline. Next, the membranes were incubated for 60 min at room temperature with the secondary antibodies. After washing with tris-buffered saline multiple times, the bands on the membranes were visualized using a Maximum Sensitivity Substrate Kit (Thermo Fisher Scientific).

MTT assay

The viability of pancreatic carcinoma cells was assessed using an MTT assay. MTT was added to each sample (0.5 mg/mL, 20 µL, Sigma) and gently rotated for 10 min. Absorbance was measured at 490 nm using a microplate reader.

Cell counting kit-8 (CCK-8) assay

PC cell proliferation was measured using a CCK-8 assay. Cells were seeded into 96-well plates. CCK-8 reagent (0.01 mL) was added to each well at different time points post transfection and incubated for 2 h at 37°C. Absorbance was measured at 450 nm using an Infinite M200 plate reader (Tecan, Männedorf, Switzerland).

Flow cytometry

An Annexin V-FITC apoptosis detection kit (BD, San Jose, CA) was used to examine cell apoptosis using flow cytometry according to the manufacturer's protocol. Cells were rinsed three times with cold phosphate buffered saline and then stained with Annexin V-FITC and PI. Cells were analyzed using a BD FACSCanto II flow cytometer (BD Biosciences) and the BD FACSDiva (BD Biosciences) software.

Transwell assay

Cells were trypsinized and rinsed once with D-Hanks solution. To determine cell migration and invasion, pore size (8 µm) culture and

Matrigel inserts, respectively, were placed in 24-well plates. Four hundred microliters of F-12 with hepatocyte growth factor (0.02 µg/mL) and fetal bovine serum (10%) was added to the lower chamber of the inserts. Approximately 1×10^5 cells were placed into the upper chamber and incubated for 20 h. Cells that traversed the pores were stained with crystal violet and observed under a microscope.

Wound healing assay

Confluent cells were scraped using a sterile pipette tip (10 µL). Then, the scratched area and cells that migrated into the wound area were observed under a microscope. The distance between the two edges of the scratch was measured and calculated. The migration percentage was obtained using the following formula: width at 0 h-width at 36 h/width at 0 h.

Bioinformatics prediction

Starbase (<http://starbase.sysu.edu.cn/>) was used to identify targets of lncRNA SNHG17. Prediction results were listed as targeting prediction efficacies. Prediction results were also listed based on conserved targets.

Dual-luciferase reporter assay (DLRA)

SNHG17 promoter-WT/MU was inserted into the pGL3-basic vector (Promega, Madison, WI, USA) to construct pGL3-SNHG17 promoter-WT/MU, which was then co-transfected with miR-942 mimics or NC mimics into HEK293T cells. The relative luciferase activity of each sample was measured using a dual-luciferase reporter assay system, while Renilla luciferase was served as the internal control (Promega, USA).

Data analysis

Each experiment was performed three times independently, and the data are shown as the mean \pm standard deviation. One-way analysis of variance and student's test were used for data analysis. Two-sided $P < 0.05$ was considered statistically significant.

Results

SNHG17 acts as the ceRNA of miR-942

Several studies have demonstrated the roles of lncRNA SNHG17 and miR-942 in cancer cells

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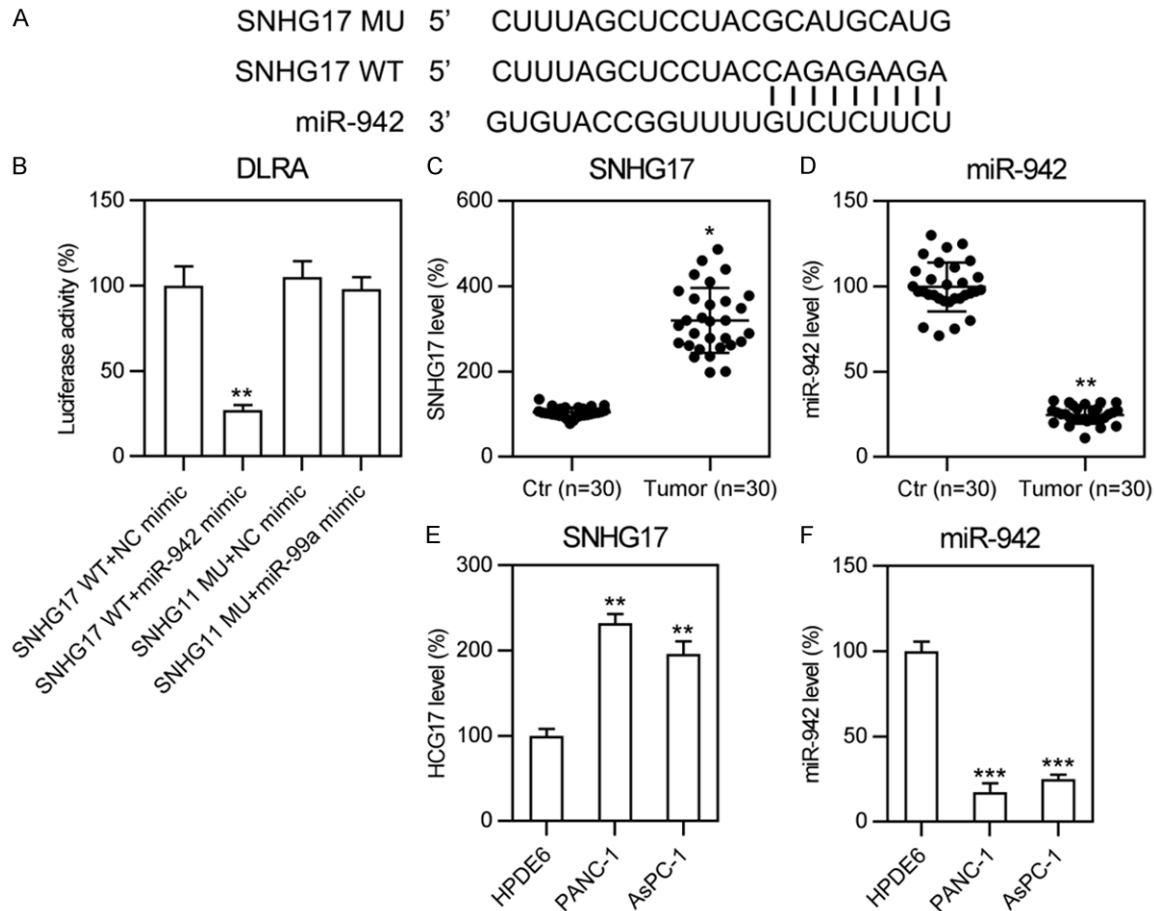


Figure 1. lncRNA SNHG17 targets miR-942. **A.** Illustration of the conserved SNHG17-binding motifs in miR-942. **B.** Luciferase functions assessed with luciferase reporter products consisting of WT or MU copy of SNHG17 after miR-942 mimic transfection in HEK293T cells. Renilla luciferase served as a reference. **C, D.** qRT-PCR analysis showing the expression of SNHG17 and miR-942 in PC specimens (n = 30) and in specimens from normal healthy tissues (n = 30). **E, F.** qRT-PCR analysis showing the expression levels of SNHG17 and miR-942 in PC (PANC-1 and AsPC-1) and HPDE6 cell lines. Data are given as the mean \pm SD. *P < 0.05, **P < 0.01, ***P < 0.001 versus the indicated groups.

[8-10, 15-18]. Therefore, we suspected that SNHG17 may act as the ceRNA of miR-942 and speculated that the interaction between SNHG17 and miR-942 may be involved in the establishment of PC cell phenotypes and tumorigenesis. Bioinformatics analysis was conducted to identify targets of SNHG17, and we found that SNHG17 targeted miR-942 (**Figure 1A**). Dual luciferase reporter assay demonstrated that SNHG17 negatively modulated miR-942 in HEK293T cells (**Figure 1B**).

To further reveal the role of SNHG17 and miR-942 during PC development, we first used qRT-PCR to analyze SNHG17 and miR-942 expression in 30 PC tissue samples and 30 paired adjacent pancreatic tissues. Compared with

that in normal healthy controls, SNHG17 was upregulated, while miR-942 was downregulated in PC samples (**Figure 1C, 1D**). Furthermore, SNHG17 expression was higher, whereas miR-942 expression was lower, in PC cell lines than in HPDE6 cells (**Figure 1E, 1F**), indicating that SNHG17 and miR-942 are negatively correlated in non-small cell lung cancer (NSCLC) cell lines.

Silencing of SNHG17 and miR-942 in PC cells

To further investigate the relationship between SNHG17 and miR-942, shRNA-NC and shRNA-SNHG17 were transfected into PANC-1 and AsPC-1 cells, respectively. The PANC-1 and AsPC-1 cells were co-transfected with shRNA-SNHG17 and miR-942 inhibitor or inhibitor NC,

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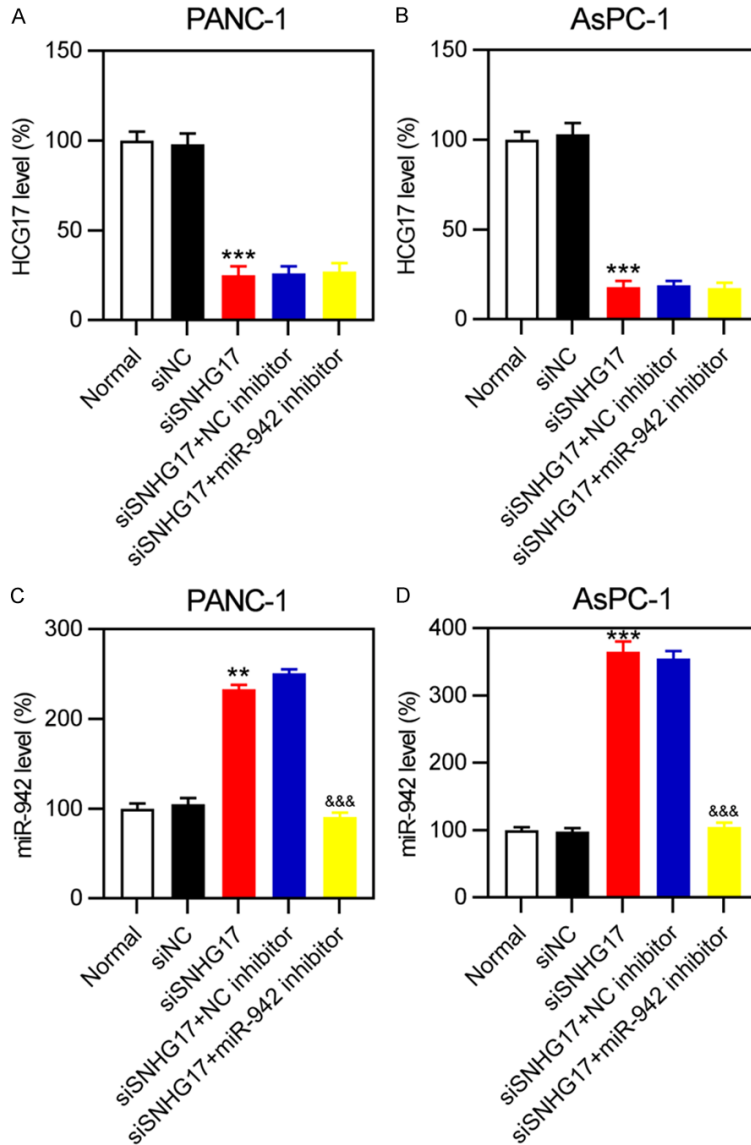


Figure 2. Downregulation of SNHG17 and miR-942 expression in PC cells. PANC-1 and AsPC-1 cells were subjected to shRNA-NC, shRNA-SNHG17, shRNA-SNHG17+NC inhibitor, and shRNA-SNHG17+miR-942 inhibitor for 36 h. qRT-PCR assays showing the expression of (A, B) SNHG17 and (C, D) miR-942 in PANC-1 and AsPC-1 cells. Data are presented as the mean \pm SD. ** $P < 0.01$, *** $P < 0.001$ vs. siNC groups; &&& $P < 0.001$ vs. siSNHG17+NC inhibitor groups.

respectively. qRT-PCR data showed that SNHG17 expression levels were significantly reduced after shRNA-SNHG17 transfection (Figure 2A, 2B). Furthermore, transfection with shRNA-SNHG17 caused a significant increase in miR-942 expression (Figure 2C, 2D). PANC-1 and AsPC-1 cells were then co-transfected with shRNA-SNHG17 and miR-942 inhibitor, which caused a downregulation in miR-942 expression in SNHG17-silenced cells without altering

SNHG17 expression (Figure 2C, 2D). These data further confirm that miR-942 expression is negatively modulated by SNHG17 in PC cells.

Effect of SNHG17 and miR-942 interaction on oncogenic phenotypes of PC cells

The viability and proliferation of PANC-1 and AsPC-1 cells were investigated via MTT and CCK-8 assays, respectively. SNHG17 silencing reduced the viability of both PANC-1 and AsPC-1 cells, and this was recovered following miR-942 inhibition (Figure 3A, 3B). Cell proliferation was examined using a CCK-8 assay and was found to be suppressed by SNHG17 silencing compared with the shNC groups. Down-regulation of miR-942 restored cell proliferation to normal levels in PC cells after SNHG17 silencing (Figure 3C, 3D). These data suggest that the SNHG17-miR-942 interaction is essential for maintaining the viability and proliferation of PC cells.

We then evaluated the effects of SNHG17 and miR-942 on cell apoptosis (Figure 4A, 4B). Our results revealed significant apoptosis in PC cells transfected with shRNA-SNHG17 compared to that in the shNC group, and this effect was countered in both PC cell lines by inhibiting miR-942 (Figure 4A, 4B). To determine which apoptotic pathway (extrinsic or intrinsic) was involved in SNHG17 silencing-induced apoptosis, we examined the expression of caspase-8 (extrinsic) and Bak (intrinsic) in PANC-1 and AsPC-1 cells with or without SNHG17 knockdown. The results showed that both caspase-8 and Bak expression was induced after SNHG17 silencing, while inhibition of miR-942 abolished the effect of SNHG17 on the expression of both the proteins (Figure 4C, 4D), sug-

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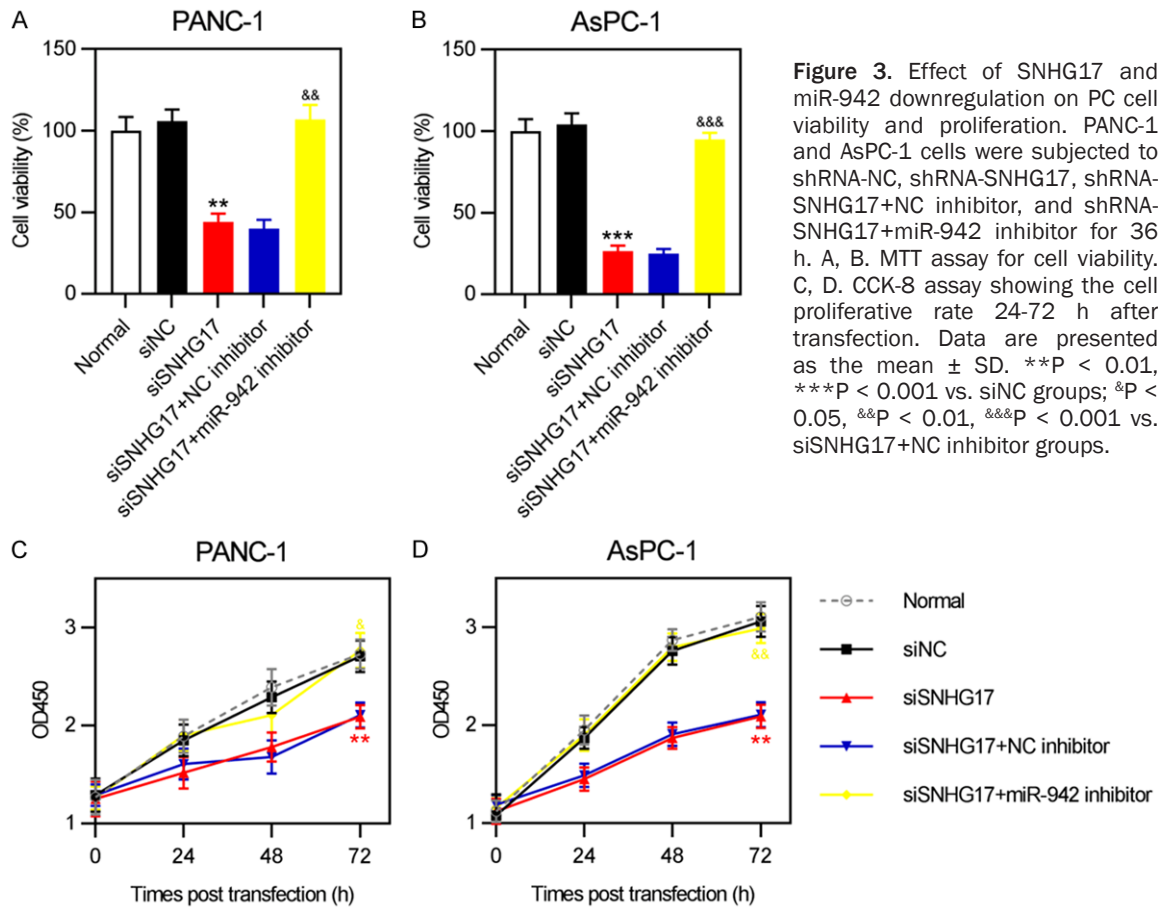


Figure 3. Effect of SNHG17 and miR-942 downregulation on PC cell viability and proliferation. PANC-1 and AsPC-1 cells were subjected to shRNA-NC, shRNA-SNHG17, shRNA-SNHG17+NC inhibitor, and shRNA-SNHG17+miR-942 inhibitor for 36 h. A, B. MTT assay for cell viability. C, D. CCK-8 assay showing the cell proliferative rate 24-72 h after transfection. Data are presented as the mean \pm SD. ** $P < 0.01$, *** $P < 0.001$ vs. siNC groups; &P < 0.05 , &&P < 0.01 , &&&P < 0.001 vs. siSNHG17+NC inhibitor groups.

gesting that both extrinsic and intrinsic pathways were activated due to SNHG17 silencing.

We then tested whether cell invasion and migration were regulated by SNHG17 and miR-942. SNHG17 silencing considerably impaired the migratory rate (**Figure 5A** and **5B**) and suppressed the invasion ability (**Figure 6A** and **6B**). The inhibition of miR-942 also restored the number of migrated and invasive PC cells, as shown in **Figures 5A, 5B, 6A** and **6B**. Thus, these data suggested that SNHG17-miR-942 interaction regulates PC cell migration and invasion.

SNHG17-miR-942 interaction regulates oncogene expression in PC cells

Previous studies have indicated that miR-942 suppresses cancer progression and cell phenotypes by downregulating PPAR γ [20], VEGF [21], ZEB1 [22], BMP [23], ISG12a [24], and GDNF [25]. Thus, we examined whether these oncogenes are also the targets of

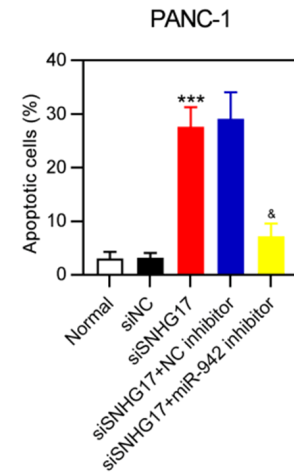
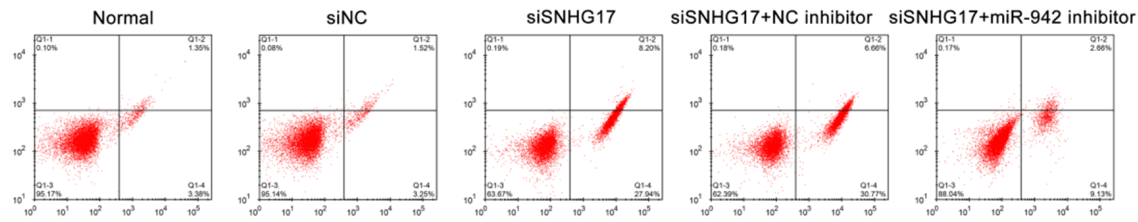
SNHG17 and miR-942 in PC cells. qRT-PCR data confirmed that SNHG17 silencing significantly reduced the expression of PPAR γ , VEGF, ZEB1, BMP, ISG12a, and GDNF at the mRNA and protein levels in PC cells, compared with that in the shNC group. These changes in expression were restored following miR-942 inhibition (**Figure 7A-N**). These data clearly suggest that the expression of these oncogenes is negatively regulated by miR-942, whereas the latter was sponged by SNHG17. Therefore, there is a positive correlation between oncogene expression and SNHG17 expression in PC cells.

SNHG17 silencing suppresses PC proliferation in vivo

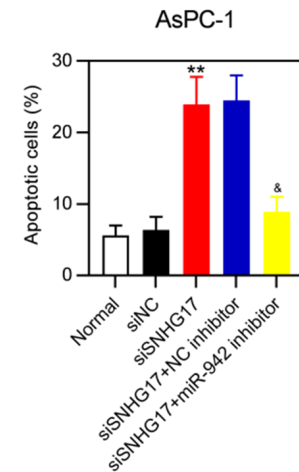
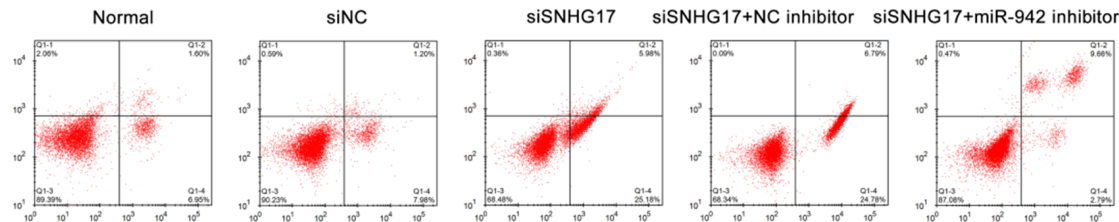
To assess the influence of SNHG17 on the tumorigenesis of pancreatic carcinoma cells, BALB/c nude mice were subcutaneously injected with AsPC-1 cells transfected with si-SNHG17 or si-NC, and were observed daily. As illustrated in **Figure 8A**, tumor growth was sig-

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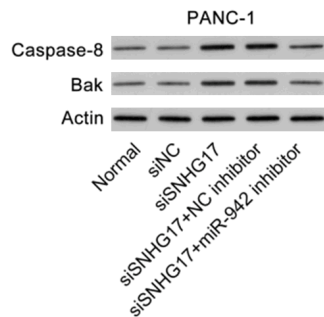
A



B



C



D

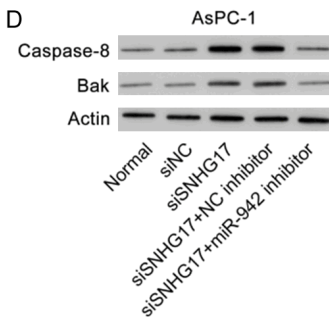


Figure 4. Effect of SNHG17 and miR-942 downregulation on PC cell death. PANC-1 and AsPC-1 cells were subjected to shRNA-NC, shRNA-SNHG17, shRNA-SNHG17+NC inhibitor, and shRNA-SNHG17+miR-942 inhibitor for 36 h. A, B. Flow cytometry analysis examining the apoptosis of PANC-1 and AsPC-1 cells. C, D. Western blotting showing protein expression of Caspase-3 and Bak in PANC-1 and AsPC-1 cells in each group. Data are presented as the mean \pm SD. * $P < 0.05$ vs. siNC groups; &P < 0.05 , &&P < 0.01 vs. siSNHG17+NC inhibitor groups.

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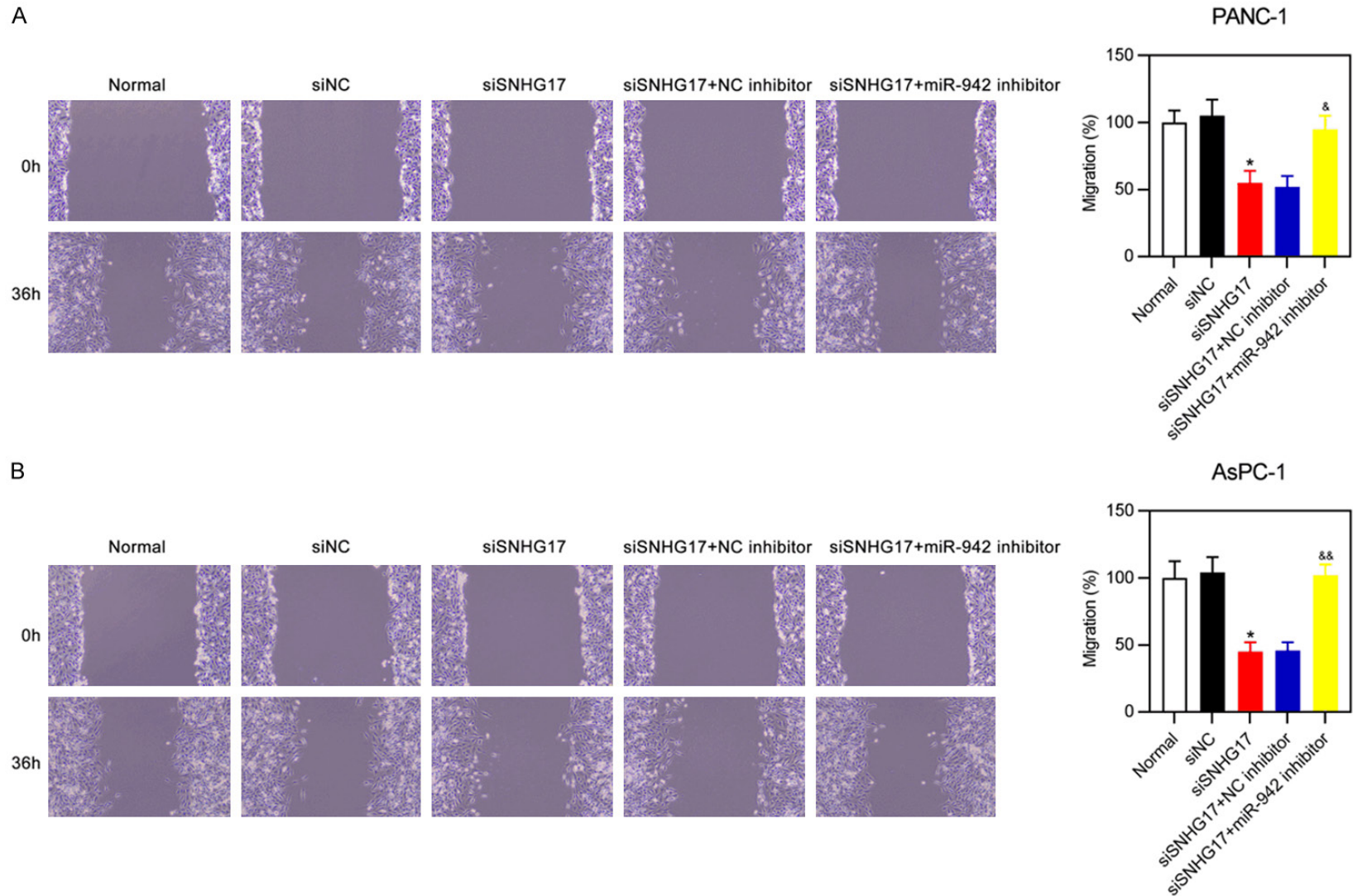
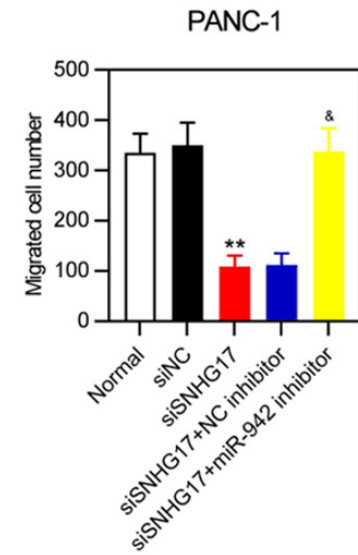
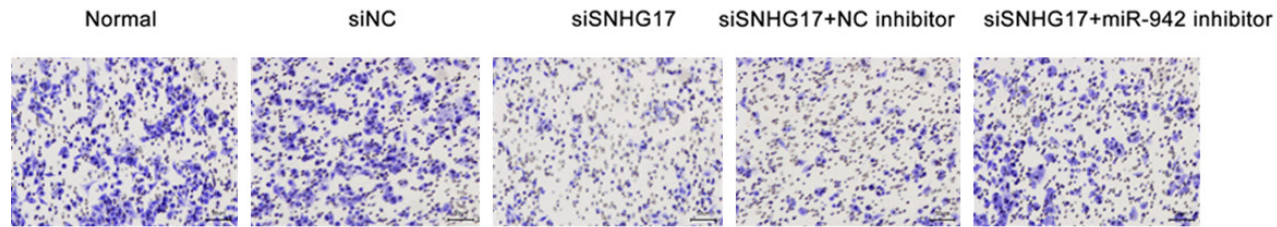


Figure 5. Effect of SNHG17 and miR-942 downregulation on the migration of PC cells. PANC-1 and AsPC-1 cells were subjected to shRNA-NC, shRNA-SNHG17, shRNA-SNHG17+NC inhibitor, and shRNA-SNHG17+miR-942 inhibitor for 36 h. A, B. Wound healing assay showing the migratory rate of PANC-1 and AsPC-1 cells. Data are presented as the mean \pm SD. *P < 0.05, **P < 0.01 vs. siNC groups; [&]P < 0.05 vs. siSNHG17+NC inhibitor groups.

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A



B

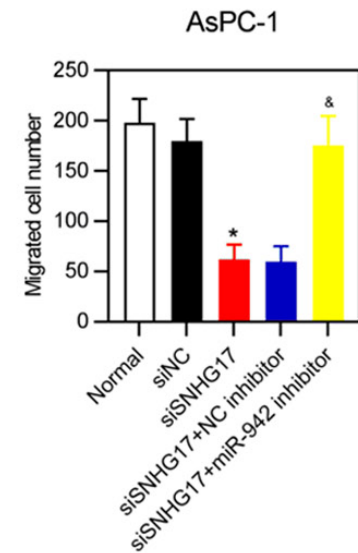
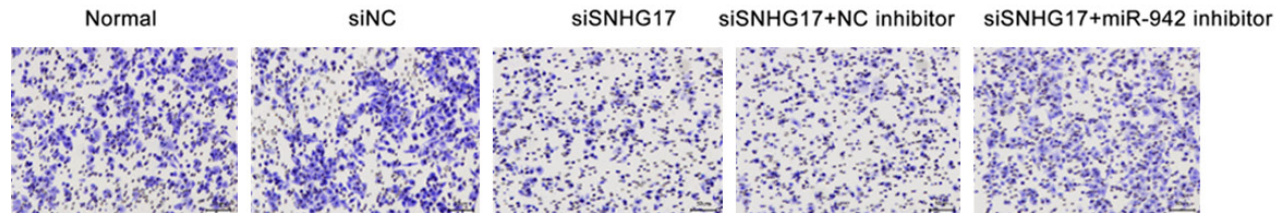
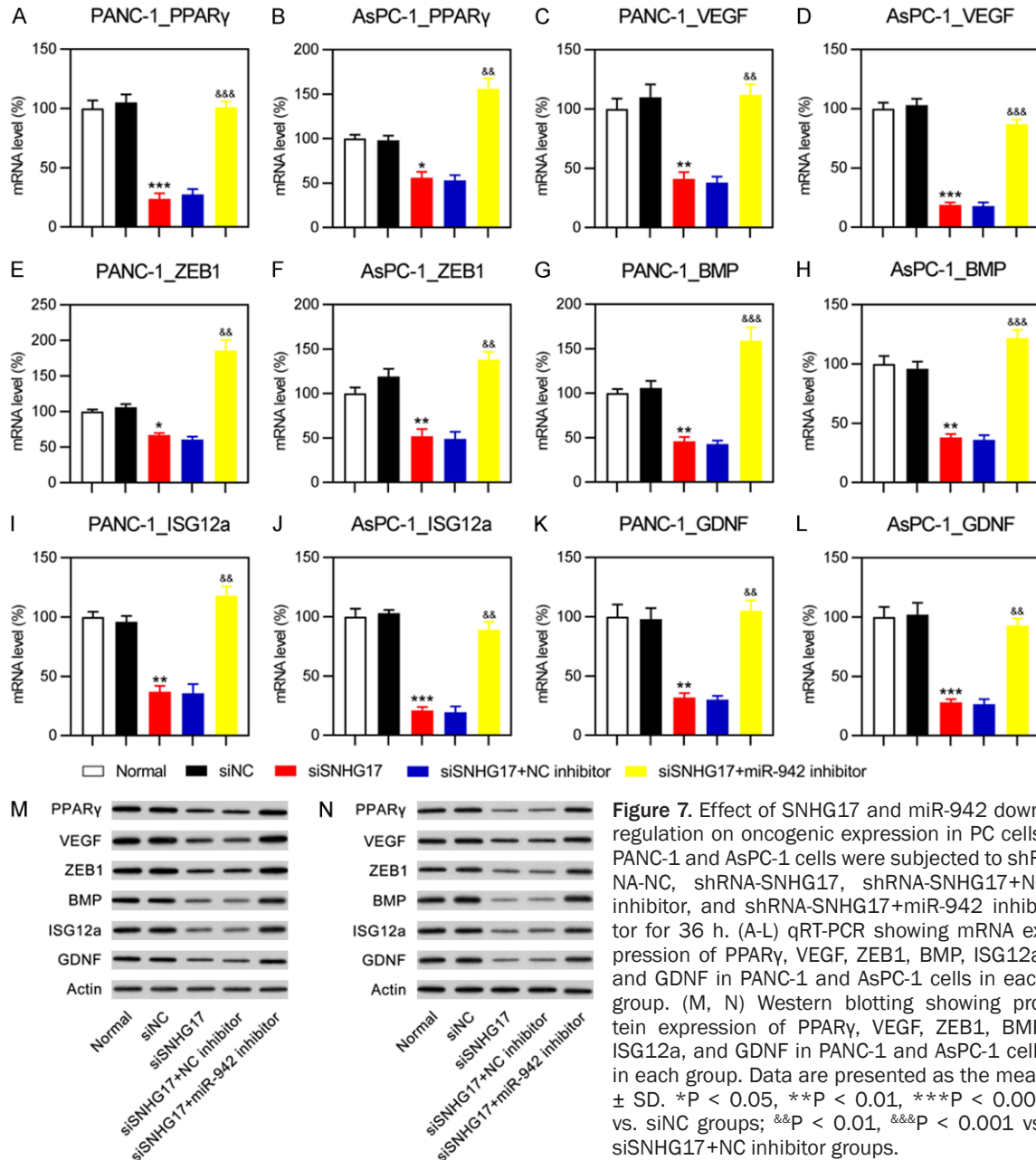


Figure 6. Effect of SNHG17 and miR-942 downregulation on the invasion of PC cells. PANC-1 and AsPC-1 cells were subjected to shRNA-NC, shRNA-SNHG17, shRNA-SNHG17+NC inhibitor, and shRNA-SNHG17+miR-942 inhibitor for 36 h. A, B. Transwell assay showing the invasion ability of PANC-1 and AsPC-1 cells. Data are presented as the mean \pm SD. ** $P < 0.01$, *** $P < 0.001$ vs. siNC groups; & $P < 0.05$ vs. siSNHG17+NC inhibitor groups.

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nificantly inhibited in the si-SNHG17 group as compared to that in the control group. Mice were euthanized 28 days later and the pancreas was harvested and weighed (**Figure 8B**). Based on average tumor weights, tumor growth was much slower in the si-SNHG17 group than in the si-NC group (**Figure 8C**). Furthermore, SNHG17 and miR-942 expression in pancreatic tissue was tested on day 28 post-injection. SNHG17 downregulation and miR-942 upregulation were confirmed in the siSNHG17 group (**Figure 8D, 8E**), suggesting that SNHG17 silencing inhibits PC tumorigenesis *in vivo*.

Discussion

Exploring PC pathogenesis to identify potent markers of early diagnosis and therapeutic targets is essential. Aberrant expression of lncRNAs in PC has been reported. Notably, SNHG17 has been found to have diagnostic and prognostic value in many human cancers, such as PC [26-28]. Our experiments revealed that lncRNA SNHG17 expression is higher in PC specimens and cell lines than in normal healthy pancreatic tissue. We found that SNHG17 is pivotal in maintaining the viability, invasion,

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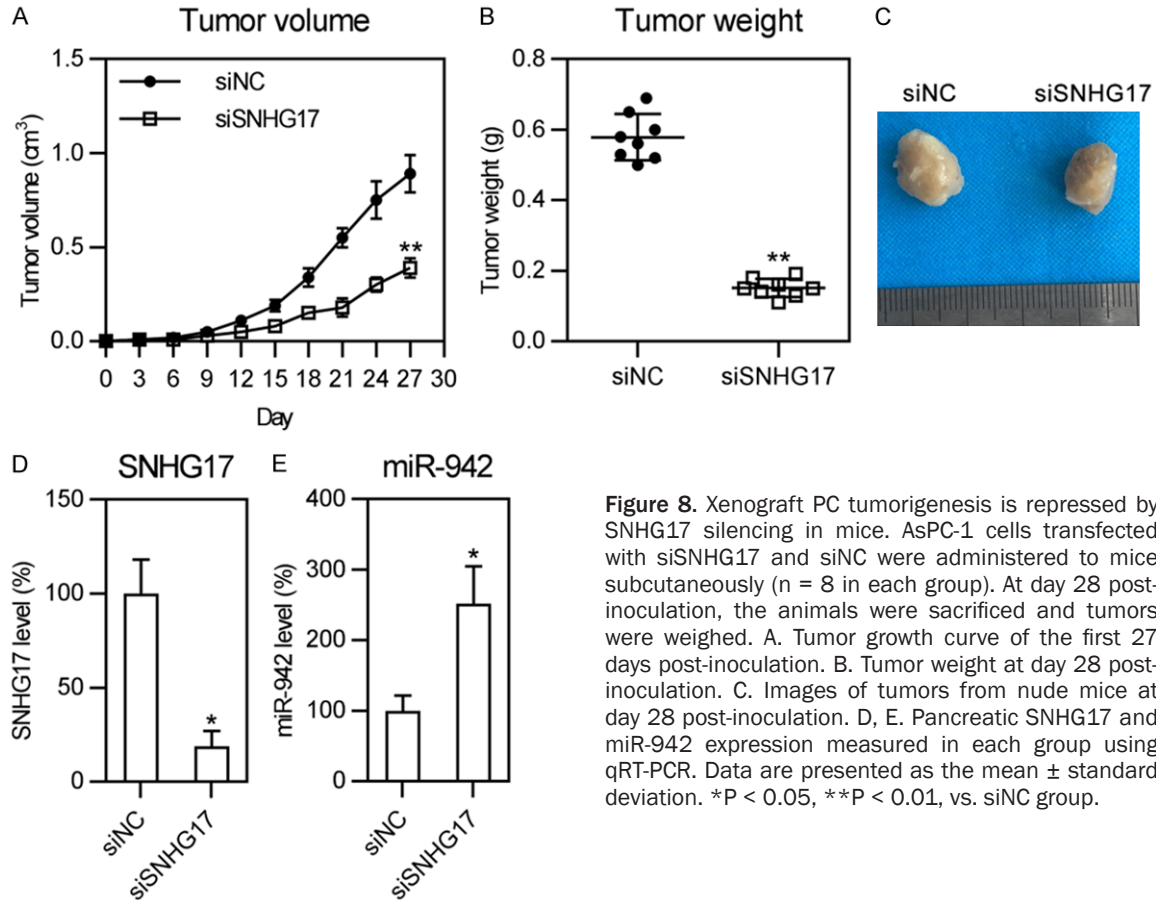


Figure 8. Xenograft PC tumorigenesis is repressed by SNHG17 silencing in mice. AsPC-1 cells transfected with siSNHG17 and siNC were administered to mice subcutaneously (n = 8 in each group). At day 28 post-inoculation, the animals were sacrificed and tumors were weighed. A. Tumor growth curve of the first 27 days post-inoculation. B. Tumor weight at day 28 post-inoculation. C. Images of tumors from nude mice at day 28 post-inoculation. D, E. Pancreatic SNHG17 and miR-942 expression measured in each group using qRT-PCR. Data are presented as the mean \pm standard deviation. *P < 0.05, **P < 0.01, vs. siNC group.

and migration of PC cells and can prevent apoptosis by competitively interacting with miR-942, which subsequently upregulates the expression of downstream oncogenes.

lncRNAs can modulate the expression of genes by interacting with miRNAs. Recently, several studies have shown that lncRNAs can regulate cancer proliferation, metastasis, angiogenesis, lipid metabolism, EMT, and apoptosis. In addition, they have been proven to impact tumorigenesis and the prognosis of patients [29-31].

A few studies have demonstrated that SNHG17 may play a key role in many medical disorders. For example, lower SNHG17 may correlate with the progression of type II diabetes mellitus [32]. Moreover, modulation of FOXA1 and BIK activities by SNHG17 is essential for cancer cell proliferation and migration in NSCLC [33]. Wu et al. showed that SNHG17 could be used to monitor prostate cancer progression [12], while Pan et al. reported that SNHG17 may be an oncogene that mediates CDK6 expression in

ovarian cancer [13]. The present study revealed for the first time that SNHG17 may promote malignant biological behaviors in PC. We discovered that SNHG17 expression levels are increased in human PC specimens as well as in PC cell lines compared to those in normal healthy controls. Additionally, through loss-of-function assays, we demonstrated that SNHG17 silencing results in attenuated cell proliferation rate, increased cell death rate, and impaired migratory and invasive capacity of PANC-1 and AsPC-1 cells. An in vitro xenograft study also suggested that SNHG17 silencing slows down the growth rate of tumors in nude mice, suggesting that SNHG17 functions as an oncogene in PC.

miR-942 has been recognized as an essential modulator of malignancy progression. Upregulation of miR-942 increases metastasis in hepatocellular carcinoma cells through RRM2B [19], and miR-942 has been shown to promote EMT in NSCLC [15]. Accumulating evidence indicates that BAMBI [34], PPAR γ [20], ISG12a

[24], RRM2B [19], FOXA2 [17], ZNF471 [35], BMP, activin membrane-bound inhibitor (BAMBI) [23], ZEB1, VEGF [21], sFRP4, GSK3, TLE1 [22], EPSTI1 [36], BARX2 [15], ANK1, GDNF, PAX6 [25], and NFKBIA [16] are targets of miR-942 and act as intermediators during tumor development. Here, we performed loss-of-function experiments, and found that inhibiting miR-942 could restore the growth and invasion of PC cells. PPAR γ , VEGF, ZEB1, BMP, ISG12a, and GDNF were also confirmed to be targets of miR-942, and therefore, controlled by SNHG17. The expression levels of PPAR γ , VEGF, ZEB1, BMP, ISG12a, and GDNF, which are considered to be oncogenes involved in PC, were positively correlated with the expression of SNHG17. Consistent with previous studies suggesting an oncogenic role of PPAR γ [37], VEGF [38], ZEB1 [39], BMP [40], and GDNF [41], the interaction between SNHG17 and miR-942 might play a role in PC pathogenesis and tumorigenesis through PPAR γ , VEGF, ZEB1, BMP, ISG12a, and GDNF expression.

In conclusion, our findings indicate that the expression of lncRNA SNHG17 is upregulated in PC, while that of miR-942 is decreased. Moreover, we found that SNHG17 directly modulates miR-942 expression, thereby regulating the expression of many oncogenes, in addition to influencing proliferation and migration of PC cells. Thus, lncRNA SNHG17 may be used as a new indicator for monitoring PC progression.

Disclosure of conflict of interest

None.

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