RETRACTED ARTICLE: MiR-142-3p targeting NUCKS1 inhibits proliferation and invasion of pancreatic cancer cells

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ABSTRACT

Objective: To investigate the effects of microRNA-142-3p (miR-142-3p) on the biological characteristics of pancreatic cancer cells and its mechanism.

Methods: The expression of miR-142-3p and nuclear casein kinase and cyclin-dependent kinase substrate 1 (NUCKS1) in pancreatic tissues and four cancer cell lines (Panc-1, BxPC-3, AsPC-1, MIA-PaCa2) were detected by Quantitative PCR (qPCR) or Western blot. The cell viability of pancreatic cancer cells was examined by MTT assay. The apoptosis of pancreatic cancer cells was measured by flow cytometry. Transwell assay was utilized to test the migration and invasion of pancreatic cancer cells. Bioinformatics analysis for miR-142-3p was conducted and the dual luciferase reporter gene assay was utilized to further validate the predicted target relationship. The protein levels of PI3K, p-AKT and T-AKT were analyzed by Western blot.

Results: The expression of miR-142-3p was down-regulated, while the expression of NUCKS1 was significantly up-regulated in pancreatic tissues and four cancer cell lines. The expression of miR-142-3p in pancreatic tissues was inversely correlated with NUCKS1 expression. Overexpression of miR-142-3p inhibited the cell viability, cell migration, and invasion, while promoted cell apoptosis of AsPC-1 and MIA-PaCa2 cells. MiR-142-3p targeted NUCKS1 and negatively regulated NUCKS1. Overexpression of miR-142-3p decreased PI3K and p-AKT expression. Up-regulation of NUCKS1 partially reversed the effects of the overexpression of miR-142-3p on the cell viability, cell apoptosis, migration and invasion, as well as PI3K and p-AKT expression in AsPC-1 and MIA-PaCa2 cells.

Conclusion: MiR-142-3p regulated the biological characteristics of pancreatic cancer cells by directly targeting NUCKS1.

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Introduction

Pancreatic cancer is a common malignant tumor with high invasive ability. Most of the patients are diagnosed in advanced stage and the 5-year survival rate is less than 5% [1]. Globally, the incidence of pancreatic cancer is increasing year by year, which is one of the leading causes of death in cancer patients [2–3]. However, the regulatory mechanism of pancreatic cancer progression has not been completely elucidated. Therefore, identification of more novel molecules, specifically expressed in pancreatic cancer, is very important for the early diagnosis and study on molecular mechanism involved.

MicroRNAs (miRNAs) are a class of non-coding small RNAs that are highly conserved and widely expressed in various tissues and cells of the body. They are involved in the regulation of tumor cell proliferation, apoptosis, invasion and other processes in tumorigenesis and development. They function as oncogenes or proto-oncogenes and may serve as a molecular target for early diagnosis and treatment of tumors [4]. Previous studies have shown that the miRNAs were abnormally expressed in pancreatic cancer, which regulated the occurrence and development of pancreatic cancer by mediating the expression level of downstream target genes [5]. Studies indicated that miR-142-3p was aberrantly expressed in pancreatic cancer [6] and served as a negative regulator of cell proliferation in pancreatic cancer [7]. However, the specific mechanism of miR-142-3p in pancreatic cancer progression is still unclear.

The nuclear casein kinase and cyclin-dependent kinase substrate 1 (NUCKS1) is a 27 kD highly phosphorylated nuclear protein and participates in DNA damage response and correlates with microRNA [8]. Previous researches have demonstrated that NUCKS1 was overexpressed in various cancers and acted as an oncogene. For example, NUCKS1 was upregulated in cervical squamous cell carcinoma (CSCC) and may be related to tumor progression [9]. Furthermore, NUCKS1 was overexpressed in gastric cancer (GC) and the proliferation, migration and invasion capacities of GC cells could be enhanced by NUCKS1 through activating the...
PI3K/Akt/mTOR signaling pathway [10]. However, the role of NUCKS1 in pancreatic cancer remains unknown.

In the current study, the expression of miR-142-3p and NUCKS1 in pancreatic cancer tissues and cells was detected and their effects on cell viability, apoptosis, migration and invasion were explored by function experiments. This study provides potential targets for the therapy of pancreatic cancer and demonstrates the underlying molecular mechanisms of pancreatic cancer.

Materials and methods

Materials

Forty one pairs of human pancreatic tissues and adjacent normal tissues were obtained postoperatively from First Hospital of Lanzhou University. The current study was approved by the Ethics Committee of the First Hospital of Lanzhou University. No patient had received radiotherapy, chemotherapy or other systemic treatments before surgical resection.

Human pancreatic cancer cells Panc-1, BxPC-3, AsPC-1, MIA-PaCa2 were purchased from American type culture collection (ATCC, Manassas, VA, USA) and human normal pancreatic ductal epithelial cell line HPDE cells were purchased from Shanghai Library of Chinese Academy of Sciences. DMEM Medium, RPMI 1640 medium, and fetal bovine serum were purchased from Gibco (Grand Island, NY, USA). TRIzol reagent, reverse transcription kit, Quantitative PCR (qPCR) kit, and apoptosis detection kit were purchased from Beyotime Biotechnology Co., Ltd (Shanghai, China). Antibodies used in this study were purchased from Abcam, (Cambridge, MA, USA). Lipofectamine 2000, MTT kit and the flow cytometer were purchased from Sigma (Saint Louis, MO, USA). MiR-142-3p mimics, pcDNA-NUCKS1 and their negative controls (miR-NC, pcDNA-NC) as well as cell culture incubator and the microplate reader were purchased from Thermo Fisher Scientific (Waltham, MA, USA). The Transwell chamber was purchased from Corning Inc. (Corning, NY, USA).

Methods

Cell culture

Pancreatic cancer cells Panc-1, BxPC-3, AsPC-1, MIA-PaCa2 and human normal pancreatic ductal epithelial cells HPDE were cultured in a medium containing 10% fetal bovine serum and 1% penicillin-streptomycin at 37 °C with an atmosphere of 5% CO2. Fresh medium was replaced every 2–3 days and cells were subcultured in a ratio of 1:3.

QPCR assay

About 5 × 10^5 cells were collected and total RNA was extracted from cells in each group according to the instructions of the TRIzol kit. About 1 μg RNA was incubated at 60 °C for 5 min and reverse transcription reagent was added to synthesize cDNA. Using U6 as a reference, the PCR mixed enzyme was dissolved in ice and used to detect the expression level of miR-142-3p and NUCKS1 according to the manufacturer’s instructions. The relative expression was calculated using the 2^ΔΔACT method. The primer for miR-142-3p was 5'-AGGTATTTCATCTTTGTGATGT-3'; the primer for NUCKS1 was 5'-TGCCCCAACCAGACTAAAG-3' (sense), and 5'-GACCCCTCATCCCCAGATT-3' (anti-sense); the primer for U6 was 5'-TGTAGGTGTTTCTACTTTAT-3' (sense), and 5'-GTCGTATCCAGTGCGGG-3' (anti-sense).

Western blot

Protein was extracted from pancreatic tissues or cell using a Protein Extraction Kit (Bio-Rad, Hercules, CA, USA). The concentration of the protein samples was determined by bicinchoninic acid assay (BCA) protein assay kit (Pierce Biotechnology, Rockford, IL, USA). Equal amount of protein samples (30 μg) were separated on 12% sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and electro-transferred to polyvinylidene fluoride (PVDF) membranes (Millipore, Billerica, MA, USA). The membranes were blocked in 5% non-fat milk at room temperature for 2 h, then incubated with primary antibodies against NUCKS1 (1:1000 dilution) or β-actin (1:2000 dilution) at 4 °C overnight. Then, the membranes were incubated with corresponding secondary antibody (1:1000 dilution) at room temperature for 2 h. The enhanced chemiluminescence system (Thermo Fisher Scientific) was utilized to detect immunoreactive bands. Image Lab Software (Bio-Rad) was used to analyze protein expression.

Cells transfection

Each well in 6-well plate was seeded with 1 × 10^5 MIA-PaCa2 or AsPC-1 cells and cells that confluence reached 70–80% were used for transfection. MiR-142-3p mimics (miR-142-3p), miR-142-3p inhibitors (anti-miR-142-3p), pcDNA-NUCKS1 (NUCKS1) and their negative controls (miR-NC, anti-NC and pcDNA-NC) were transfected into MIA-PaCa2 or AsPC-1 cells according to the Lipofectamine 2000 instruction manual, respectively.

MTT assay

Cells in each group were seeded into 96-well plate with 5 × 10^3 cells per well and inoculated for 0 h, 24 h, 48 h, 72 h, 96 h. Then 20 μL MTT was added into each well and cells were incubated at 37 °C for another 4 h, and 200 μL DMSO was added into each well. The absorbance of each well at 490 nm was measured.

Flow cytometry

According to the kit, the density of MIA-PaCa2 or AsPC-1 cells was adjusted to 1 × 10^6 cells/mL. After incubation for 24 h, 10 μL of Annexin V-FITC and 5 μL of propidium iodide (PI) were added to each well. The apoptosis rates of MIA-PaCa2 or AsPC-1 cells were analyzed by flow cytometry after staining at 4 °C for 15 min.
Transwell assay

Transwell chamber was placed above the sterile 24-well plate. Cells in each group were collected and digested with trypsin. The cell density was adjusted to $1 \times 10^5$ cells/mL. 100 μL cell suspension was added to the upper well. About 600 μL medium containing 10% fetal bovine serum was added to the lower chamber and incubated at 37°C for 48 h. Then, the cells on the lower surface of the membrane were washed twice with PBS and the remaining cells on the upper surface of the membrane were wiped off with wet cotton swab. Cells were immobilized with 4% paraformaldehyde for 30 min, stained with 0.5% crystal violet for 15 min, photographed and counted under a microscope. For invasion detection, Matrigel gel was removed. The rest of the procedures were same as migration detection.

Target gene prediction and dual luciferase reporter gene assay

The target genes of miR-142-3p were predicted using the online database TargetScan. The wild-type of NUCKS1 and its mutant were synthesized by Guangzhou Ribo Bio Co., Ltd. A fragment of 3'-untranslated region (3'-UTR) of NUCKS1 containing the predicted binding site for miR-142-3p or its mutant was amplified and cloned into psiCHECK-2 luciferase reporter vector (Promega Corp., Madison, WI, USA) to synthesis NUCKS1-WT or NUCKS1-MUT. MIA-PaCa2 or AsPC-1 cells at logarithmic phase were co-transfected with NUCKS1-WT or NUCKS1-MUT and miR-142-3p mimics or anti-miR-142-3p using Lipofectamine 2000. The dual-luciferase activity of the samples was evaluated utilizing the dual-luciferase reporter detection system (Promega) according to the instructions of the manufacturer.

Statistical analysis

SPSS 22.0 software (IBM, Armonk, NY, USA) was used for statistical processing. The results were exhibited as mean ± SD (standard deviation). The difference among groups was analyzed by ANOVA (one-way analysis of variance) or SNK-q test. $p < .05$ was considered statistically significant.

Results

Expression of miR-142-3p and NUCKS1 in pancreatic cancer tissues and cell lines

The expression levels of miR-142-3p and NUCKS1 in pancreatic cancer tissues and cells were detected by qPCR or Western blot assay. MiR-142-3p expression was significantly decreased in pancreatic cancer tissues and four cancer cell lines (Figure 1(A,B)). MIA-PaCa2 or AsPC-1 cells with the lower expression level of miR-142-3p were used for the subsequent study. As it was exhibited in Figure 1(C–F), NUCKS1 was obviously up-regulated in pancreatic cancer tissues and four cancer cell lines at both mRNA level and protein level. Person correlation analysis suggested that the expression level of miR-142-3p was inversely correlated with NUCKS1 expression in pancreatic cancer tissues (Figure 1(G)).

![Figure 1](https://example.com/figure1.png)

Figure 1. Expression of miR-142-3p and NUCKS1 in pancreatic cancer tissues and cell lines. (A and C) QPCR was conducted to measure the miR-142-3p and NUCKS1 expression in pancreatic cancer tissues. *$p < .05$ compared with normal tissues. (B and E) QPCR assay for the expression of miR-142-3p and NUCKS1 in pancreatic cancer cell lines. *$p < .05$ compared with HPDE cells. (D) Western blot assay for the protein level of NUCKS1 in pancreatic cancer tissues. *$p < .05$ compared with normal tissues. (F) Western blot assay for the protein level of NUCKS1 in pancreatic cancer cell lines. *$p < .05$ compared with HPDE cells. (G) Correlation analysis between miR-142-3p and NUCKS1 in pancreatic cancer tissues. *$p < .05$. 


The association between miR-142-3p expression and clinicopathological features of pancreatic cancer

To evaluate the correlation between miR-142-3p expression and the clinicopathological features, 41 patients were divided into high miR-142-3p expression group (higher than the median level, \( n = 19 \)) and low miR-142-3p expression group (lower than the median level, \( n = 22 \)) according to the median level of miR-142-3p expression (0.295). Comparisons were performed between the two groups (Table 1). The results showed that high miR-142-3p expression group was significantly correlated with the clinical stage (\( p = .002 \)) and distant metastasis (\( p = .000 \)), not with other clinical characteristics, including gender, age, tumor size and differentiation, clinical stage, and lymph node status (Table 1).

Table 1. The association between miR-142-3p expression and clinicopathological features of pancreatic cancer.

| Characteristic          | Low (\( n = 22 \)) | High (\( n = 19 \)) | \( p \) |
|-------------------------|--------------------|---------------------|--------|
| Gender                  |                    |                     | .707   |
| Male                    | 25                 | 14 (34.2%)          | 11 (26.8%) |
| Female                  | 16                 | 8 (19.5%)           | 8 (19.5%) |
| Age (years)             |                    |                     | .577   |
| \(<60\)                 | 24                 | 12 (29.2%)          | 12 (29.2%) |
| \(>60\)                 | 17                 | 10 (24.4%)          | 7 (17.2%) |
| Tumor size (cm)         |                    |                     | .850   |
| \(0–2\)                 | 9                  | 5 (12.2%)           | 4 (9.8%) |
| \(2–5\)                 | 27                 | 13 (31.7%)          | 14 (34.1%) |
| \(>5\)                  | 5                  | 2 (4.9%)            | 3 (7.3%) |
| Tumor differentiation   |                    |                     | .812   |
| Well                    | 6                  | 4 (9.8%)            | 2 (4.9%) |
| Moderate                | 25                 | 12 (29.2%)          | 13 (31.7%) |
| Poor                    | 10                 | 6 (14.6%)           | 4 (9.8%) |
| Clinical stage          |                    |                     | .002   |
| I–II                    | 24                 | 8 (19.5%)           | 16 (39.0%) |
| III–IV                  | 17                 | 14 (34.1%)          | 3 (7.4%) |
| Lymph node status       |                    |                     | .257   |
| N0                      | 22                 | 10 (24.5%)          | 12 (29.2%) |
| N1                      | 19                 | 12 (29.2%)          | 7 (17.1%) |
| Distant metastasis      |                    |                     | .000   |
| M0                      | 24                 | 7 (17.1%)           | 17 (41.4%) |
| M1                      | 17                 | 15 (36.6%)          | 2 (4.9%) |

Effect of up-regulation of miR-142-3p on cell viability and apoptosis of pancreatic cancer cells

MIA-PaCa2 or AsPC-1 cells were transiently transfected with miR-142-3p mimics or its negative control miR-NC. QPCR was employed to confirm transfection efficiency. MiR-142-3p expression was apparently up-regulated in MIA-PaCa2 or AsPC-1 cells after transfection with miR-142-3p mimics, compared with that in cells transfected with miR-NC (Figure 2(A,B)). Introduction of miR-142-3p significantly inhibited the cell viability of MIA-PaCa2 or AsPC-1 cells (Figure 2(C,D)). Up-regulation of miR-142-3p facilitated the apoptosis of MIA-PaCa2 or AsPC-1 cells (Figure 2(E,F)).

Effects of up-regulation of miR-142-3p on cell migration and invasion of pancreatic cancer cells

Transwell assay was applied to examine the migration and invasion capacities of MIA-PaCa2 or AsPC-1 cells after...
transfection. Gain of miR-142-3p significantly repressed cell migration of MIA-PaCa2 or AsPC-1 cells (Figure 3(A,B)). Introduction of miR-142-3p also inhibited cell invasion of MIA-PaCa2 or AsPC-1 cells (Figure 3(C,D)).

NUCKS1 was a target of miR-142-3p

Online database TargetScan was used to predict the downstream target genes of miR-142-3p. The predicted binding site for miR-142-3p or its mutant site on the 3′-UTR of NUCKS1 was exhibited in Figure 4(A). The luciferase activity of MIA-PaCa2 or AsPC-1 cells co-transfected with miR-142-3p mimics and NUCKS1-WT was notably lower than that of cells co-transfected with miR-NC and NUCKS1-WT (Figure 4(B,D)). Evidently, the luciferase activity of MIA-PaCa2 or AsPC-1 cells co-transfected with anti-miR-142-3p and NUCKS1-WT was elevated, compared with cells co-transfected with anti-NC and NUCKS1-WT (Figure 4(C,E)), while no significant change was

Figure 3. Effect of up-regulation of miR-142-3p on migration and invasion of pancreatic cancer cells. (A,B) Transwell migration assay for MIA-PaCa2 and AsPC-1 cells. (C,D) Transwell invasion assay for MIA-PaCa2 and AsPC-1 cells. *p<0.05 compared with miR-NC group.
detected in the luciferase activity of cells co-transfected with NUCKS1-MUT and miR-142-3p or anti-miR-142-3p (Figure 4(B–E)).

Effects of combination of miR-142-3p and NUCKS1 on cell viability and apoptosis of pancreatic cancer cells

Transfection with miR-142-3p mimics effectively inhibited NUCKS1 expression at both mRNA level and protein level in MIA-PaCa2 or AsPC-1 cells. While overexpression of NUCKS1 partially rescued the inhibitory effects on NUCKS1 expression at both mRNA level and protein level in MIA-PaCa2 or AsPC-1 cells induced by miR-142-3p (Figure 5(A–D)). MTT assay indicated that NUCKS1 also reversed the miR-142-3p-mediated inhibitory effects on cell viability of MIA-PaCa2 or AsPC-1 cells in part (Figure 5(E,F)). As is shown in Figure 5(G–J), up-regulation of miR-142-3p facilitated the apoptosis of MIA-PaCa2 or AsPC-1 cells, whereas overexpression of NUCKS1 abolished almost all miR-142-3p-induced promoted impact on the apoptosis of the two cell lines.

Effects of combination of miR-142-3p and NUCKS1 on migration and invasion of pancreatic cancer cells

Transwell assay manifested that up-regulation of NUCKS1 reverted the repressive impact on the migration and invasion
Figure 5. Effects of combination of miR-142-3p and NUCKS1 on cell viability and apoptosis of pancreatic cancer cells. (A and B) QPCR assay for miR-142-3p expression in MIA-PaCa2 and AsPC-1 cells after transfection or co-transfection. (C and D) Western blot assay for the protein level of NUCKS1 in MIA-PaCa2 and AsPC-1 cells after transfection or co-transfection. (E and F) MTT assay for the cell viability of MIA-PaCa2 and AsPC-1 cells after transfection or co-transfection. (G and H) Flow cytometry assay for cell apoptosis in MIA-PaCa2 and AsPC-1 cells after transfection or co-transfection. *p<0.05 compared with miR-NC group. **p<0.05 compared with miR-142-3p group.
Effects of combination of miR-142-3p and NUCKS1 on migration and invasion of pancreatic cancer cells. (A and B) Transwell migration assay for MIA-PaCa2 and AsPC-1 cells after transfection or co-transfection. (C and D) Transwell invasion assay for MIA-PaCa2 and AsPC-1 cells. *p<.05 compared with miR-NC group. #p<.05 compared with miR-142-3p + pcDNA group.

Discussion
Pancreatic cancer is a lethal disease without effective treatments until now and the early symptoms of it are unconspicuous [11]. In recent years, the morbidity and mortality of pancreatic cancer have gradually increased. The mortality of patients with pancreatic cancer is high, which is closely related to the ability of tumor tissue metastasis. Studies have shown that about 80% of the patients with pancreatic cancer have lost the chance of surgery at the time of diagnosis because the tumor has metastasized [12]. Therefore, it is very crucial to search for novel biomarkers which contribute to the early diagnosis of pancreatic cancer.

MiRNAs is a group of short non-coding RNAs and the abnormal expression of miRNAs plays an important role in the occurrence and development of tumors, including pancreatic cancer [13]. Although miRNAs only account for 1–3% of the human genome, they regulate 30% of the human genome and participate in the growth and development of the body, the occurrence and development of diseases, as well as cell proliferation, apoptosis and other processes [14,15]. Previous studies revealed that the expression level of miR-142-3p in tumor tissues was significantly ectopic and it participated in the occurrence and development of tumors by regulating various physiological and pathological processes of cells. For instance, the expression level of miR-142-3p in renal carcinoma was significantly increased and down-regulation of miR-142-3p expression significantly inhibited the proliferation, migration and promoted apoptosis of renal carcinoma cells, thus affecting the occurrence and development of renal carcinoma [16]. MiR-142-3p played an important role in the growth and development of breast cancer by targeting the activity of Wnt/β-catenin signaling pathway.
Meanwhile, the expression of miR-142 was obviously inhibited in pancreatic cancer samples and miR-142 modulated cell proliferation, migration and invasion by targeting hypoxia-inducible factor 1 (HIF-1α) in the tumor microenvironments [19]. In line with previous studies, in this study, the expression level of miR-142-3p was significantly decreased in pancreatic tissues and four cancer cell lines, which was detected by qRT-PCR. We observed that up-regulation of miR-142-3p inhibited cell viability and promoted apoptosis of MIA-PaCa2 and AsPC-1 cells. In addition, up-regulation of miR-142-3p also suppressed migration and invasion of pancreatic cancer cells, suggesting that miR-142-3p may play an inhibitory role in the occurrence and development of pancreatic cancer, thus inhibiting the growth of the tumor.

To further explore the mechanism of regulation of miR-142-3p on pancreatic cancer, we predicted the target gene of miR-142-3p by utilizing online database TargetScan. The results showed that miR-142-3p and NUCKS1 have partial complementary sequences. Following dual-luciferase reporter, gene assay results showed that NUCKS1 was a target gene of miR-142-3p.

NUCKS1 is a member of the high migration protein family with a molecular weight of 27 kDa, which is located on human chromosome 1q32. NUCKS1 widely exists in various tissues of the human body and participates in the occurrence and development of tumors. Studies have shown that NUCKS1 can bind to insulin-like growth factor-1 receptors and participate in the evolution of many malignant tumors [20–22]. Studies revealed that the expression of NUCKS1 in a variety of tumor tissues is significantly up-regulated, suggesting that NUCKS1 plays a vital role in the progression of tumors. The expression of NUCKS1 in breast cancer was obviously increased and was significantly related to clinical-pathological parameters and NUCKS1 can be used as a molecular marker for poor prognosis of breast cancer [23]. It was reported that NUCKS1 was prominently up-regulated in skin squamous cell carcinoma and basal-cell epithelioma and was closely related to the expression of cell proliferation-related proteins, which could function as a potential marker for the malignant proliferation of tumor cells [24]. In addition, NUCKS1 was highly expressed in cervical cancer and colorectal cancer and was strongly associated with the survival rate and prognosis of patients [9,25]. However, the role of NUCKS1 in pancreatic cancer has not been reported. In our study, NUCKS1 was a target of miR-142-3p and was up-regulated in pancreatic cancer tissues and cell lines. To confirm whether miR-142-3p regulated the biological characteristics of pancreatic cancer cells by regulating NUCKS1, recovery experiment was conducted. Evidently, the exogenous introduction of NUCKS1 counteracted the biological characteristics of pancreatic cancer cells.
of pancreatic cancer cells mediated by miR-142-3p. A former literature indicated that NUCKS1 enhanced gastric cancer aggressiveness via the PI3K/Akt/mTOR signaling pathway in an IGF-1R-dependent manner [10]. In addition, we found that miR-142-3p repressed the expression of AKT signaling pathway-related protein, PI3K and p-AKT. NUCKS1 also reversed the inhibitory effects of miR-142-3p on AKT signaling pathway.

In conclusion, the expression of miR-142-3p was down-regulated in pancreatic cancer tissues and cell lines, whereas the expression of NUCKS1 was up-regulated. Up-regulation of miR-142-3p could significantly inhibit the proliferation, invasion and migration of pancreatic cancer cells and induce their apoptosis. Up-regulation of miR-142-3p also inhibited AKT signaling pathway. While the effects of miR-142-3p on pancreatic cancer cells and AKT signaling pathway could be reversed by NUCKS1 in part. Thus, miR-142-3p inhibited proliferation, migration and invasion of pancreatic cancer cells by targeting NUCKS1. This is the first report about the role of NUCKS1 in pancreatic cancer and the association between miR-142-3p and NUCKS1, moreover, this study provides a new target for pancreatic cancer treatment.

Disclosure statement

No potential conflict of interest was reported by the authors.

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