Functions and clinical significances of LncRNA SChLAP1 and UBE2E3 in prostate cancer

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Abstract

Backgrounds: Prostate cancer is the most common cancer in male with high morbidity and mortality. The long noncoding RNA second chromosome locus associated with prostate-1(SChLAP1) and ubiquitin conjugating enzyme(UBE2E3) have been reported to relate to prostate cancer. SCHLAP1-UBE2E3 was found to be the most frequent gene fusion in Chinese. However, the function and mechanism of SChLAP1-UBE2E3 axis in prostate cancer are not well known.

Methods: In this study, we investigated the relationship between SChLAP1 and UBE2E3 expression and development of prostate cancer in Chinese men who have undergone radical resection of prostate cancer. The effects of SChLAP1 and UBE2E3 on proliferation, migration and invasion of prostate cancer cells were respectively explored.

Results: We found that SChLAP1 and UBE2E3 were both over expressed in prostate cancer cells and tissues. There was a positive correlation between the expression of UBE2E3 and SCHLAP1. Overexpression of SChLAP1 and UBE2E3 both promoted cell proliferation. migration and invasion and inhibited apoptosis. SChLAP1 and UBE2E3 knocking down just had the opposite effects.

Conclusions: Our study proved that SChLAP1 and UBE2E3 influenced the progressive of prostate cancer and provided potential biomarkers or drug targets for prostate cancer.

Introduction

Prostate cancer is the most prevalent male malignancy in the world with high incidence and mortality, ranking second of the most frequent cancer and the fifth leading cause of death in male^[1-3]. Although the highest rates were found in western countries, the incidences of prostate cancer in developing countries were increasing year by year, including China^[4]. According to WHO, the incidence of prostate cancer in China was about 15.6 per 100,000 people in 2020, with more than 110,000 new cases and more than 50,000 deaths. Diagnoses and treatment of prostate cancer is an urgent public health problem worldwide. However, for the majority of prostate cancer patients, they have multiple primary tumor foci, and there was obvious molecular heterogeneity among different foci^[5-6]. The identification of molecular biomarkers to aid in diagnostics prognostics and treatment of prostate cancer is a difficulty, but need to overcome.

Long Noncoding RNA, LncRNA, is a kind of noncoding RNA that are longer than 200 nucleotides. Many lncRNAs have been shown to be specifically expressed in tumors and therefore considered to be potential biomarkers^[7-9]. Second chromosome locus associated with prostate-1(SChLAP1), one of the most important lncRNAs, has been demonstrated that it is highly expressed in a portion of prostate cancers and is closely related to tumourgenesis. An RNA in situ hybridization (ISH) assay proved that SChLAP1 expression increased with prostate cancer progression, and high SChLAP1 expression was related to poor outcome after radical prostatectomy^[10]. The mechanism of SChLAP1 in prostate cancer has been

reported in some studies. It was reported that knockdown of SCHLAP1 can promote apoptosis and inhibit proliferation and metastasis *in vitro* and *in vivo*, through interacting with miR-198 and subsequently modulated the MAPK1 signaling pathway^[11]. Some results suggested that SCHLAP1 promoted proliferation, migration, and tumor development of prostate cancer through directly binding with EZH2 and repressing miR-340-3p expression^[12]. SCHLAP1 may also antagonize the SWI/SNF-complex to coordinates cancer cell invasion and metastatic spread^[13]. However, studies are still required to investigate the clinical value and exact mechanism of SCHLAP1 in prostate cancer.

Interestingly, SCHLAP1-UBE2E3 was found to be the most frequent gene fusion in a large Chinese cohort, suggesting important diagnostic and therapeutic potential of SCHLAP1-UBE2E3 for prostate cancer^[14]. UBE2E3, also called UbcM2, is a ubiquitin conjugating enzyme. Cells depleted of UBE2E3 become senescent and the action of UBE2E3 can be partially compensate by co-depletion of either p53 or its cognate target gene, or by co-depleting the tumor suppressor^[15]. A genome-wide siRNA screen found UBE2E3 may regulate cell surface E-cadherin in the APC-defective colorectal cancer cell^[16]. A cDNA microarray identified that UBE2E3 was over-expressed in prostate cancer^[17]. UBE2E3 had also been reported that it may related to the invasiveness of non-functioning pituitary adenomas^[18]. All these previous studies indicated the possible effects of UBE2E3 on cancer, including prostate cancer. However, the exact function and mechanism of UBE2E3 in prostate cancer has not been reported.

In this study, we aim to explore the correlation between SCHLAP1-UBE2E3 and the physiological state of prostate cancer patients. The potential functions and mechanisms of SCHLAP1 and UBE2E3 in the progression of prostate cancer were also investigated. We revealed that the SCHLAP1 and UBE2E3 can both promote prostate cancer development and there was a positive correlation between the expression of UBE2E3 and SCHLAP1, providing promising biomarkers or drug targets for prostate cancer.

Materials And Methods

Reagents

RPMI 1640 was purchased from Beijing Solarbio. Fetal Bovine Serum was purchased from Invitrogen. NucleoZol was purchased from Gene. Antibodies for UBE2E3 and beta actin were purchased from abcam. Lipofectamine™ 2000 Transfection Reagent was purchased from Invitrogen. Cell Counting Kit-8 was from Dojindo. ANNEXIN V- FITC/PI Apoptosis kit was from Beijing solarbio.

Clinical samples

60 patients who have undergone radical resection of prostate cancer were collected with complete clinical data. All prostate cancer cases were diagnosed for the first time. All specimens were confirmed by pathology, and none of the patients received radiotherapy, chemotherapy, endocrine therapy, and androgen deprivation treatment before surgery. The corresponding para-cancerous specimens served as the control group. The study was approval of ethics committee.

Cell culture

RWPE-1 and PC-3 cells were purchased from Procell(China) and grown in RPMI 1640. All cells were cultured at 37°C in a 5% CO₂ incubator.

RNA extraction and quantitative reverse transcription(qRT-PCR)

RNA was extracted from tissues or cells using NucleoZol reagent, according to the manufacturers' protocols. Then RNA was reverse transcribed into cDNA using reverse transcriptase. Quantitative PCR was performed using SYBR Green qPCR Master Mix in a Fast Real-time PCR 7500 System(Applied Biosystems, USA). GAPDH served as the internal control for normalization. Relative expression levels were calculated using the $2^{-\Delta\Delta C_t}$ method.

Western-Blot: Cells were lysed with lysis buffer containing PMSF for 20 min on ice. The supernatant was collected by centrifugation at 12,000 rpm for 20 min at 4°C. Protein concentrations were determined using the BCA protein assay kit. Equivalent amounts of proteins samples were separated by SDS-PAGE and transferred to nitrocellulose membrane. After 2 h blocking with 5% bovine serum albumin, the membranes were incubated with primary antibodies overnight at 4°C. Wash the membranes with TBST three times and then incubated with HRP-conjugated secondary antibodies(1:5000 dilutions) at room temperature for 2 h. Membranes were extensively washed three times again. Finally, proteins were detected and analyzed using Versa Doc™ imaging system. β -actin was used as an endogenous reference.

Establishment of transient transfection cell lines

pcDNA3.1-SCHLAP1 and pcDNA3.1-UBE2E3 were constructed for gene over expression. shRNA for SCHLAP1 and UBE2E3 were designed and synthesized for gene silencing. PC-3 cells in logarithmic growth phase were harvested and inoculated into well plates before transfection. Transfection was performed using Lipofectamine™ 2000 according to the instructions. The expression levels of SCHLAP1 and UBE2E3 were detected by qRT-PCR or western bolt respectively after 24 h transfection.

Cell viability assay

Transfected cells were harvested after 24 h culturing and seeded in 96-well plates. Cell viability was measured by the CCK-8 Kit according to the instructions. Briefly, 100 μ l 10% CCK-8 solution was added to each well at 0, 24, 48 and 72 h respectively and incubated for another 1–2 h at 37°C after adding. The absorbance at 450 nm was then measured and inhibitory rate of cell proliferation was calculated.

Flow cytometry analysis of apoptosis

PC-3 cells were inoculated in 6-well plates and cultured overnight, transfected when the convergence degree reached 80%-90%. Transfected cells were harvested after 24 h. The cells were washed with ice-cold PBS twice and then resuspended in annexin V binding buffer. FITC-conjugated annexin V antibody

was added and incubated at room temperature for 15 min. Propidium iodide was added 5 min before detection. The cells were then analyzed with FACSVerse Flow Cytometer(BD,USA).

Transwell immigration and invasion assay

For immigration assay, cells were harvested and resuspend in serum-free medium. The concentration of cells was adjusted to 1×10^5 cell/ml. 100 μ l cells were seeded into the upper chamber and the lower chamber contained medium with 20% FBS. After 24 h incubation at 37°C, the cells in the upper chamber were wiped with a cotton swab and discard the medium in both chamber. The cells in the lower chamber were then fixed with 4% paraformaldehyde at 4°C for 30 min and stained with crystal violet for 15 min. Observe the cells under a microscope and count the cells.

For invasion assay, the upper chambers were pre-coated with Matrigel matrix. Briefly, the Martrigel matrix was melted at 4°C and diluted 1:8 on ice in serum-free medium. 50 μ l diluted Martrigel matrix was added into the chamber, air-dried at 4°C and solidified at 37°C, then placed in 24-well plate. The cell suspension was added into the upper chambers. After 24 incubation, take out the transwell chamber, wipe the cells and the remaining Matrigel in the chamber, and wash with PBS. The cells passing through the bottom side of the chamber were fixed by paraformaldehyde and stained with crystal violet. Images of the stained cells were captured and the number of cells were counted.

Statistical analysis

All experiments in this study were performed independently with three biological replicates. Data are presented as means $\pm$ standard deviation. The statistical significance was analyzed using Univariate anOVA for comparison between multiple groups, and Student's t-test for comparison between two groups. A value of $P < 0.05$ was indicated as statistically significant difference.

Results

SChLAP1 and UBE2E3 are highly expressed in prostate cancer

To explore the functions of SChLAP1 and UBE2E3 in prostate cancer, the mRNA levels of SChLAP1 and UBE2E3 were examined in prostate cancer tissues and their corresponding noncancerous tissues from 60 patients. The results showed that the expressions of UBE2E3 and SCHLAP1 were both significantly upregulated in prostate cancer tissues compared with normal tissues(Fig. 1A). Spearman correlate analysis showed that there was a positive correlation between the expression of UBE2E3 and SCHLAP1(Fig 1B). The expressions of SChLAP1 and UBE2E3 were subsequently detected in normal prostatic epithelial cells RWPE-1 and prostate cancer cells PC-3. The expression level of SChLAP1 was detected by RT-PCR, while the expression level of UBE2E3 was determined by western blot. Likewise, the expression levels of SChLAP1 and UBE2E3 were both remarkably higher in prostate cancer cells than

normal cells(Fig. 1C,1D). All these results indicated that both SChLAP1 and UBE2E3 were highly expressed in prostate cancer, and they may work together in the progression of prostate cancer.

Relationships between SChLAP1-UBE2E3 and the progression of prostate cancer

In order to clarify the association between the expression of SChLAP1-UBE2E3 and the factors associated with pathological features of patients, we took χ^2 test. As a result, no matter SChLAP1 expression level or UBE2E3 expression level showed no significant association with patients' ages($p>0.05$). But, the results revealed that high SChLAP1 and UBE2E3 expressions were associated with lymph node metastasis, tumor size, PSA expression level, PSAD, Gleason score and seminal vesicle invasion($p<0.05$) (Table 1, 2). Specifically, the expression of SChLAP1 in patients with high Gleason score was remarkably higher than those with low Gleason score(Table 1). The expression of SChLAP1 was also associated with positive margin, clinical stage and capsule invasion($p<0.05$) but the expression of UBE2E3 wasn't($p>0.05$)(Table 1, 2). The above results indicated that SChLAP1 and UBE2E3 may have certain effect on development and metastasis of prostate cancer.

SChLAP1 promotes proliferation and inhibits apoptosis of prostate cancer cells

Based on the above clinical observation, SChLAP1 indeed has effect in prostate cancer. To further investigate the function of SChLAP1 in prostate cancer cells, we constructed pcDNA3.1-SCHLAP1 for gene over expression and knocked down SCHLAP1 by shRNA respectively. All constructed plasmids were transfected into PC-3 cells and the efficiencies of transfection were verified by qRT-PCR. The results showed that the relative mRNA level of SChLAP1 was significantly decreased after the transfection of SChLAP1-shRNA and the level was increased after the transfection of pcDNA3.1-SCHLAP1(Fig 2A). This revealed the successfully transfection in PC-3 cells in the present study. Next, we determined the role of SChLAP1 in prostate cancer cell growth. The cells proliferation was detected by CCK-8 kit assay. The results showed that overexpression of SCHLAP1 promoted PC-3 cell proliferation, while knocking down of SChLAP1 evidently inhibited cell viability(Fig 2B). Flow cytometry analysis was also taken out to detect cell apoptosis. The results showed that SChLAP1 over expression significantly decreased the percentage of annexin V+ apoptotic cells, while SChLAP1 knock down had the opposite effect(Fig 2C,2D). All these results indicated that SChLAP1 may promote proliferation and inhibit apoptosis in prostate cancer cells.

SChLAP1 promotes migration and invasion of prostate cancer cells

We also investigated the effects of SChLAP1 on cell migration and invasion using transwell assays. The results demonstrated that the number of cells migrated and invaded the lower chamber in SChLAP1 knockdown group was significantly lower than that in the control group, while the invasion and migration of SChLAP1 over expressed cells were significantly improved compared with that of control cells(Fig 3A,3B,3C,3D). Collectively, these results indicated that SCHLAP1 can significantly affect the migration and invasion of prostate cancer cells.

UBE2E3 promotes proliferation and inhibits apoptosis of prostate cancer cells

As UBE2E3 was over expressed in prostate cancer cells and the expression was positively correlated with SCHLAP1, UBE2E3 may also influence the progression of prostate cancer. Similarly, pcDNA3.1-UBE2E3 was constructed for gene over expression and shRNA specific for UBE2E3 was designed and transfected into pLVX-Puro for knocking down. The efficiencies of transfection were verified by western blot. The relative expression level of UBE2E3 was significantly higher in pcDNA3.1-UBE2E3 transfected cells and lower in UBE2E3-shRNA transfected cells compared with the control, revealing the success of transfection(Fig 4A). CCK-8 assay showed that overexpression of UBE2E3 promoted PC-3 cell proliferation, while knocking down of UBE2E3 evidently inhibited cell proliferation(Fig 4B). Flow cytometry analysis demonstrated that silencing of UBE2E3 promoted apoptosis, while over expression of UBE2E3 significantly inhibited apoptosis(Fig 4C,4D). These results illustrated that UBE2E3 may promote proliferation and inhibit apoptosis in prostate cancer cells.

UBE2E3 promotes migration and invasion of prostate cancer cells

Furthermore, to explore the effects of UBE2E3 on cell migration and invasion, transwell assays were carried out. The cells migrated to the lower chamber were significantly reduced in UBE2E3 knockdown cells, but increased when UBE2E3 was over expressed(Fig. 5A,5B). The capacity for cell invasion was obviously improved in pcDNA3.1-UBE2E3 transfected cells, while decreased after UBE2E3 suppression compared to the control(Fig. 5C,5D). Taken together, these results indicated that UBE2E3 can also promote the migration and invasion of prostate cancer cells.

Discussion

Long noncoding RNAs are often dysregulated in prostate cancer, playing important roles in proliferating and metastasis of prostate cancer^[19–20]. It has been reported that there were hundreds of prostate-cancer-associated lncRNAs, including PCAT1, PVT1, PCAN-R1, PCA3 and SChLAP1 etc^[21–25]. The possible mechanisms of lncRNAs were various, including oncogenic mutations, copy number variation, DNA methylation and histone modifications^[26–28]. These lncRNAs may be potential biomarkers or novel

therapeutic targets for prostate cancer. lncRNAs are promising candidates for liquid-biopsy-based biomarkers, because they can be detected in both plasma and urine.

In this study, we focused on lncRNA SChLAP1 and explored the function of SChLAP1 in prostate cancer. Previous studies have revealed that SChLAP1 may be a prognostic biomarker and SChLAP1 levels can independently predict poor outcomes of prostate cancer. SChLAP1 could take part in cell growth arrest, apoptosis induction, and migration inhibition. In the present work, we found that SChLAP1 expression was significantly increased in prostate cancer samples compared with their adjacent normal tissues, and similar results were observed in the corresponding cell lines. Then we over-expressed SChLAP1 and knocked down SChLAP1 in prostate cancer cells respectively, and found that SChLAP1 over expression could promote proliferation, migration and invasion of prostate cancer cells and inhibit apoptosis. Conversely, knockdown of SChLAP1 accelerated apoptosis and restrained the proliferation, migration and invasion. These results proved that SChLAP1 is cancerogenic in prostate cancer.

SCHLAP1-UBE2E3 was found to be the most frequent gene fusion in Chinese, however very few studies have investigated the role of UBE2E3 in prostate cancer. UBE2E3 is a ubiquitin conjugating enzyme and commonly expressed in cells. Studies have demonstrated that ubiquitination and deubiquitination were usually dysregulated in cancer cells, exerting oncogenic or anti-cancer roles^[29–30]. UBE2E3 may also affect the development of prostate cancer. Thus, in this study, we first detected the expression of UBE2E3 in prostate cancer and found UBE2E3 was highly expressed in prostate cancer tissues as well as prostate cancer cells compared with their adjacent normal tissues or normal cells. Spearman correlate analysis showed that there was a positive correlation between the expression of UBE2E3 and SCHLAP1. The gain-of-function and loss-of-function experiments were also adopted. The results showed that UBE2E3 promoted proliferation, migration and invasion of prostate cancer cells and inhibit apoptosis, and knockdown of UBE2E3 exerted opposite effects. These findings provided evidence that UBE2E3 played a role in prostate cancer.

To further explored the clinical value of SChLAP1-UBE2E3, the clinical significances of SChLAP1 and UBE2E3 in the progression of prostate cancer were investigated. SChLAP1 and UBE2E3 expressions were both correlated with tumor size, PSA level, PSAD, Gleason score, tumor metastasis and invasion. Positive margin and clinical stage were also confirmed to have some relationship with SChLAP1 expression. SChLAP1 and UBE2E3 expressions were remarkably increased in samples from tumors with high Gleason scores(≥ 8) compared to tumors with lower GSs, suggesting an association with aggressive disease.

In summary, SChLAP1 and UBE2E3 were both highly expressed in prostate cancer and there was a positive correlation between the expression of UBE2E3 and SCHLAP1. Both SChLAP1 and UBE2E3 can promote proliferation, migration and invasion of prostate cancer cells and inhibit apoptosis. Meanwhile, SChLAP1 and UBE2E3 were associated with pathological features of patients, indicating that they were predictive of disease recurrence after prostatectomy. Although the exact mechanism between UBE2E3 and SChLAP1 were not fully investigated in the current study, we believe that SChLAP1 is a promising

biomarker or novel target for prostate cancer and SChLAP1 may influence the development of prostate cancer through UBE2E3.

Declarations

Ethics approval and consent to participant:

This study was approved by the Ethics Committee of Fujian Medical University Union Hospital and the methods were carried out in accordance with the approved guidelines. Informed consent was obtained from participants

Consent for publication:

No applicable.

Availability of data and material:

All data generated or analysed during this study are included in this published article [and its supplementary information files]..

Competing interests:

No applicable.

Funding:

None.

Authors' contribution:

XRY and CXB investigated the effects of SChLAP1 and UBE2E3 on proliferation, migration and invasion of prostate cancer cells and they contributed equally to this work. HLJ and ZJK collected the patients' samples. JW was the corresponding author and a major contributor in writing the manuscript. All authors read and approved the final manuscript.

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Tables

Table 1

Relationships between SChLAP1 and the pathological features of patients

Variables	Total(n=60)		SCHLAP1		χ^2	P
		n	High level[%]	Low level[%]		
age	≤70	30	20(66.67)	10(33.33)	0.6345	0.4257
	≥70	30	17(56.67)	13(43.33)		
Gleason score	2-7	25	8(32.00)	17(68.00)	15.96	< 0.001
	8-10	35	29(82.86)	6(17.14)		
PSA	≤20	31	15(48.39)	16(51.61)	4.785	0.0287
	≥20	29	22(75.86)	7(24.14)		
PSAD	≤0.4	37	19(51.35)	18(48.62)	4.345	0.0371
	≥0.4	23	18(78.26)	5(21.74)		
Tumor size	<5cm	38	19(50.00)	19(50.00)	5.967	0.0146
	≥5cm	22	18(81.82)	4(18.18)		
Clinical stage	T1-T2	37	19(51.35)	18(48.65)	4.345	0.0371
	T3-T4	23	18(78.26)	5(21.74)		
Lymph node metastasis	yes	14	12(85.71)	2(14.29)	4.467	0.0346
	no	46	25(54.35)	21(46.65)		
Capsule invasion	yes	23	18(78.26)	5(21.74)	4.345	0.0371
	no	37	19(51.35)	18(48.65)		
Seminal vesicle invasion	yes	15	13(86.67)	2(13.33)	5.288	0.0215
	no	45	24(53.33)	23(46.67)		
Surgical margin	Negative	22	9(40.91)	13(59.09)	6.332	0.0119
	Positive	38	28(73.68)	10(26.32)		

Table 2

Relationships between UBE2E3 and the pathological features of patients

Variables		n	Total(n=60)	UBE2E3		
			High level%	High level%	χ^2	P
age	≤ 70	30	15 (50.00%)	15 (50.00%)	2.500	0.1138
	> 70	30	21 (70.00%)	9 (30.00%)		
Gleason score	2-7	25	11 (44.00%)	14 (56.00%)	4.571	0.0325
	8-10	35	25 (71.43%)	10 (28.57%)		
PSA	≤ 20	31	14 (45.16%)	17 (54.84%)	5.884	0.0153
	> 20	29	22 (75.86%)	7 (24.14%)		
PSAD	≤ 0.4	37	17 (45.95%)	20 (54.05%)	7.944	0.0048
	> 0.4	23	19 (82.61%)	4 (17.39%)		
Tumor size	$< 5\text{cm}$	38	19 (50.00%)	19 (50.00%)	4.318	0.0377
	$\geq 5\text{cm}$	38	17 (77.27%)	5 (22.73%)		
Clinical stage	T1-T2	37	19 (51.35%)	18 (48.65%)	3.008	0.0828
	T3-T4	23	17 (73.91%)	6 (26.09%)		
Lymph node metastasis	yes	14	12 (85.71%)	2 (14.29%)	5.031	0.0249
	no	46	24 (52.17%)	22 (47.83%)		
Capsule invasion	yes	23	17 (73.91%)	6 (26.09%)	3.008	0.0828
	no	37	19 (51.35%)	18 (48.65%)		
Seminal vesicle invasion	yes	15	13 (86.67%)	2 (13.33%)	5.926	0.0149
	no	45	23 (51.11%)	22 (48.89%)		
Surgical margin	Negative	22	12 (54.55%)	10 (45.45%)	0.431	0.5117
	Positive	38	24 (63.16%)	14 (36.84%)		

Figures

Figure 1

SchLAP1 and UBE2E3 were highly expressed in prostate cancer. (A) The mRNA levels of SchLAP1 and UBE2E3 in 60 prostate cancer tissues and adjacent normal tissues. (B) Spearman correlate analysis between UBE2E3 expression and SCHLAP1 expression. (C) The mRNA levels of SchLAP1 in RWPE-1 and PC-3 cells. (D) The expression of UBE2E3 in RWPE-1 and PC-3 cells measured by Western Blot. The blots were intact and from different parts of the same gel.

Figure 2

SChLAP1 promotes proliferation and inhibits apoptosis in prostate cancer cells. (A) PC-3 cells were transfected with SChLAP1-shRNA or pcDNA-SChLAP1, and the mRNA levels of SChLAP1 were measured by qRT-PCR. (B) Cell viability was assayed by the CCK-8 kit after 0, 24, 48 and 72 h. (C) The cell apoptosis was analyzed by annexin V flow cytometry. (D) Apoptotic cell quantification

Figure 3

SChLAP1 promotes migration and invasion of prostate cancer cells. (A) PC-3 cells were transfected with SChLAP1-shRNA or pcDNA-SChLAP1, and cell migrations were determined by transwell assays. (B) Quantification of (A). (C) Transwell invasion assay. (D) Quantification of (C).

Figure 4

UBE2E3 promotes proliferation and inhibits apoptosis in prostate cancer cells. (A) PC-3 cells were transfected with pcDNA-UBE2E3 or shRNA-UBE2E3, and the expressions of UBE2E3 were determined by Western Blot. The blots were intact and from different parts of the same gel. (B) Cell viability was assayed by the CCK-8 kit after 0, 24, 48 and 72 h. (C) The cell apoptosis was analyzed by annexin V flow cytometry. (D) Quantification of (C).

Figure 5

UBE2E3 promotes migration and invasion of prostate cancer cells. (A) PC-3 cells were transfected with pcDNA-UBE2E3 or shRNA-UBE2E3, and cell migrations were determined by transwell assays. (B) Quantification of (A). (C) Transwell invasion assay. (D) Quantification of (C).