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RESEARCH

LncRNA LINC01128 promotes prostate cancer cell proliferation, metastasis, and epithelial-mesenchymal transition by modulating miR-27b-3p

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Abstract

Background Prostate cancer (PCa) is a prevalent malignancy within the male reproductive system that poses a significant threat to patients' lives. The function of long non-coding RNA LINC01128 in PCa progression remains to be elucidated.

Objective The objective was to evaluate the significance of LINC01128 in PCa and to elucidate the underlying mechanisms, thereby identifying a potential target for PCa treatment.

Methods The clinical significance of LINC01128 in PCa was investigated by bioinformatics methods and data analysis. The expression of LINC01128 was quantified using real-time quantitative PCR. The impact of LINC01128 on PCa cell viability and metastasis was evaluated through Cell Counting Kit-8 and Transwell assays. The expression of epithelial-mesenchymal transition markers was analyzed by Western blot analysis. Bioinformatics methods and dual-luciferase reporter assay were employed to explore the mechanisms underlying the role of LINC01128 in PCa progression.

Results LINC01128 demonstrated significant upregulation in PCa and exhibited a strong correlation with tumor-node-metastasis (TNM) stage, Gleason score, and lymph node metastasis. The upregulation of LINC01128 was found to be linked to a poorer prognosis for PCa. In PCa cells, silencing LINC01128 resulted in the suppression of cell proliferation, migration, and invasion. Furthermore, the knockdown of LINC01128 enhanced the expression of E-cadherin while concurrently repressing the expression of N-cadherin and Vimentin. Mechanistically, the negative regulation of miR-27b-3p by LINC01128 mediated the role of LINC01128 in PCa progression.

Conclusions In PCa, high expression of LINC01128 may predict patients' unfavorable prognosis. LINC01128 promoted PCa cellular processes by negatively regulating miR-27b-3p.

Keywords LINC01128 · Prostate cancer · Prognosis · miR-27b-3p · Progression

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Abbreviations

cDNA	Complementary DNA
CCK-8	Cell counting kit-8
CI	Confidence interval
DMEM	Dulbecco's modified eagle medium
EMT	Epithelial-mesenchymal transition
FBS	Fetal bovine serum
GAPDH	Glyceraldehyde-3-phosphate dehydrogenase
HR	Hazard ratio
lncRNA	Long non-coding RNA
miRNA	MicroRNA
MUT	Mutant
NC	Negative control
OD	Optical density
PCa	Prostate cancer

PVDF	Polyvinylidene fluoride
RT-qPCR	Real-time quantitative polymerase chain reaction
siRNA	Small interfering RNA
SDS-PAGE	Sodium dodecyl sulfate polyacrylamide gel electrophoresis
TNM	Tumor-node-metastasis
WT	Wild-type

Introduction

Prostate cancer (PCa) represents the most widespread malignant neoplasm of males, ranking second only to lung cancer in the incidence of male cancers (Bray et al. 2024). The prevalence of PCa increases with age, with higher incidences observed in Europe and the United States (Nelson et al. 2022). The pathological types of PCa include adenocarcinoma, ductal adenocarcinoma, intraductal carcinoma, squamous cell carcinoma, uroepithelial carcinoma, and basal cell carcinoma, among others. Of these, adenocarcinoma represents more than 95% of the total (Chinese guidelines for 2019). PCa exhibits a heightened association with an augmented risk of bladder outlet obstruction, lower urinary tract lesions, and distant metastases. These complications have profound consequences for patients' quality of life (Pan et al. 2023). The primary treatments for PCa are radical prostatectomy, radiotherapy, and chemotherapy. The efficacy of these treatments in improving outcomes for patients with early-stage PCa has been well-documented (Kanesvaran et al. 2022). The mortality rate of PCa has exhibited a downward trend over the past few decades, a development that can be attributed to significant advancements in treatment modalities. Nevertheless, there is currently no effective pharmacological agent or other therapeutic tool that can meaningfully enhance the prognosis of patients with metastatic PCa (Guevelou et al. 2024). The exploration of new biomarkers for predicting PCa progression and molecular therapeutic targets has become a hot topic of increasing interest (Rafikova et al. 2023).

Long non-coding RNAs (lncRNAs) are molecules comprising more than 200 nucleotides that do not encode proteins. Cytoplasmic lncRNAs frequently serve as "sponges," which are molecules that bind to and sequester microRNAs (miRNAs) from their usual targets, thereby modulating the activity of the miRNAs (Dinescu et al. 2019). A substantial body of research has demonstrated the involvement of lncRNAs in the onset and progression of PCa, exerting either tumor-suppressive or oncogenic effects (Taheri et al. 2023; Shree et al. 2023). For example, lncRNA AFAP1-AS1 has been demonstrated to facilitate PCa cell proliferation and invasion, thereby functioning as an oncogene (Liu et al. 2021). A recent study has identified LINC01128 as a tumor promoter in pancreatic cancer, suggesting a potential association with the disease's prognosis (Zhong et al. 2022). One study identified LINC01128 as a differentially expressed lncRNA in PCa by analyzing the GSE46602 dataset (Ye et al. 2018). Nevertheless, the biological function and mechanism of LINC01128 in PCa have not yet been reported by any study. Furthermore, LINC01128 has been demonstrated to affect cellular processes in glioma cells by modulating miR-27b-3p (Zhang et al. 2022). In a previous study, Chen et al. indicated that miR-27b-3p functions as a suppressor in PCa (Chen et al. 2019). It is therefore postulated that LINC01128 exerts biological functions in PCa by regulating miR-27b-3p.

In the present study, the clinical significance of LINC01128 in PCa was analyzed through the evaluation of relevant clinical data. Additionally, the mechanisms by which LINC01128 affects PCa cell growth and metastasis were investigated through in vitro experiments.

Materials and methods

Study subjects

Cancerous and paracancerous normal tissues were collected from 125 patients with PCa who underwent radical prostatectomy between 2018 and 2020 at Longhua Hospital Shanghai University of Traditional Chinese Medicine. All patients with PCa were included based on the results of pathological diagnosis. The study obtained ethical approval from the hospital ethics committee. Inclusion and exclusion criteria: (1) patients with complete clinical information; (2) the patient or their relatives must sign an informed consent form; (3) patients who have not undergone molecularly targeted therapy, neoadjuvant therapy, chemoradiotherapy, and so forth; (4) the patient does not have other malignant neoplasms; and (5) patients who are able to complete follow-up visits.

RT-qPCR

The extraction of total RNA was carried out using TRI reagent (Sigma-Aldrich, Germany). The cDNA was reverse-transcribed using the miRNA cDNA Synthesis Kit (Cwbio, Jiangsu, China) and amplified by RT-qPCR in accordance with the instructions provided with the MicroRNAs qPCR Kit (Sangon Biotech, Shanghai, China). GAPDH and U6 were utilized to normalize LINC01128 and miR-27b-3p, respectively. The relative level of LINC01128 and miR-27b-3p was calculated using the $2^{-\Delta\Delta Ct}$ method.

Cell lines and transfection

PCa cell lines (LNCaP, PC-3, 22Rv1, and DU145) and human normal prostate epithelial cells (RWPE-1) were procured from ATCC (USA). LNCaP and 22Rv1 were cultured in RPMI-1640 medium, PC-3 in Ham's F-12 K medium, DU145 in DMEM medium, and RWPE-1 in K-SFM medium. The medium was supplemented with 10% FBS and penicillin-streptomycin (Gibco, USA). The cells were cultivated in an incubator at 37 °C and 5% CO_2 atmosphere.

The siRNA targeting LINC01128 (si-LINC01128) and the negative control (si-NC) were synthesized by RiboBio (Guangzhou, China). The miR-27b-3p mimic, miR-27b-3p inhibitor, mimic NC, and inhibitor NC were procured from MedChemExpress (USA). Transfection was conducted using Lipofectamine RNAiMAX (Invitrogen, USA).

Western blot

Cells were lysed using Pierce RIPA buffer (Thermo Scientific, USA), which contains 1% cocktail. The protein samples were separated by SDS-PAGE. Subsequently, the proteins in the gel were transferred to a PVDF membrane (Millipore, USA). To prevent non-specific binding of the antibody, the membrane was incubated with 5% skim milk for 2 h. The primary antibodies utilized were procured from Cell Signaling Technology (USA), including N-cadherin (13116T), E-cadherin (3195T), and Vimentin (5741T), which were diluted at a ratio of 1:1000. The GAPDH antibody (ab9485, Abcam, UK) was diluted at a ratio of 1:2500. The membranes were incubated with primary antibodies at 4 °C overnight. Subsequently, the membranes were incubated with Rabbit IgG (H+L) Cross-Adsorbed Secondary Antibody (31212, Invitrogen, USA) diluted at 1:3000 for 1 h at room temperature. The blots were visualized using the Clarity Western ECL Substrate Kit (Bio-Rad, USA).

CCK-8 assay

Cells were inoculated into 96-well plates. The CCK-8 solution (Beyotime, Shanghai, China) was added to the wells at 24-hour intervals (0, 24, 48, and 72) for a two-hour incubation period. The absorbance values (OD_{450} nm) were determined using a Synergy HTX microplate spectrophotometer (BioTek, USA).

Transwell assay

The upper chamber of the Transwell inserts (Corning, USA) was prepared with matrix gel (which was not utilized in migration experiments), and the lower chamber was filled with complete medium. The transfected cells were seeded into the upper chamber as a cell suspension in FBS-free medium and cultured under routine conditions for 48 h. The residual cells on the membrane of the upper chamber were removed gently. The chambers were fixed with 4% paraformaldehyde and subsequently stained with 0.1% crystal violet. Five fields of view were selected for photographing and counting the migrated and invaded cells under an IX83 inverted microscope (Olympus, Japan).

Bioinformatics analysis

The differential expression of LINC01128 in PCa, as well as its association with different survival states, was analyzed using the online UALCAN database (https://ualcan.path.uab.edu/analysis.html). The relative expression of miR-27b-3p in PCa was analyzed using the online database starBase (https://rnasysu.com/encori/). Additionally, the complementary sites of LINC01128 and miR-27b-3p were predicted using this database.

Dual-luciferase reporter assay

The wild-type (WT) and mutant (MUT) vectors (WT-LINC01128, MUT-LINC01128) were constructed based on the complementary sites of LINC01128 and miR-27b-3p. Luciferase vectors were transfected into DU145 cells with either miR-27b-3p mimic, inhibitor, or corresponding negative controls. Following 48 h of cultivation, the luciferase activity was quantified.

Statistical analysis

The collected data were analyzed and presented in graphical form using GraphPad Prism 8.0 and SPSS 22.0. Discontinuous variables were analyzed by Chi-square test. Prognostic information was assessed using Cox regression analysis and Kaplan-Meier curve. The correlation between the expression levels of LINC01128 and miR-27b-3p in PCa tissues was evaluated using Pearson's method. Continuous variables were recorded as standard deviation of the means, and differences between groups were compared using Student's t-test or one-way ANOVA. There was a statistical difference at $P<0.05$.

Results

The upregulation of LINC01128 in PCa was related to prognosis

The analysis of the UALCAN database disclosed that the relative expression of LINC01128 was markedly elevated in PCa tissues (Fig. 1A). Furthermore, a notable distinction in the survival of patients with PCa exhibiting different levels of LINC01128 was also observed (Fig. 1B). The present study investigated the expression of LINC01128 in 125 PCa tissues and paracancerous normal tissues. Consequently, LINC01128 was found to be overexpressed in PCa (Fig. 1C).

The mean level of LINC01128 in PCa was employed as a threshold to categorize patients into high- and low-expression groups. Higher levels of LINC01128 were found to be associated with advanced tumor-node-metastasis (TNM) stage ($P=0.019$), lymph node metastasis ($P=0.001$), and higher Gleason score ($P=0.039$) in subjects (Table 1). Furthermore, patients with elevated LINC01128 levels exhibited a reduced overall survival rate ($P=0.021$, Fig. 1D). Cox regression analysis uncovered that LINC01128 [hazard ratio (HR) = 3.690, 95% confidence interval (CI) = 1.205–11.301], lymph node metastasis (HR = 2.945, 95% CI = 1.123–7.722), and Gleason score (HR = 3.550, 95% CI = 1.184–10.644) were independent prognostic factors for PCa (Fig. 1E).

Knockdown of LINC01128 suppressed the malignant phenotype of PCa cells

In comparison to normal prostate cells (RWPE-1), LINC01128 was also observed to be overexpressed in PCa cells (Fig. 2A). Subsequently, 22Rv1 and DU145 cells were selected for further in vitro experimentation. In comparison to si-NC, the transfection of si-LINC01128 reduced LINC01128 expression in both 22Rv1 and DU145 cells (Fig. 2B). The silencing of LINC01128 was observed to inhibit the viability of both 22Rv1 and DU145 cells (Fig. 2C). The knockdown of LINC01128 also demonstrated the attenuation of the migratory and invasive capabilities of 22Rv1 and DU145 cells (Fig. 2D **and E**). To elucidate the function

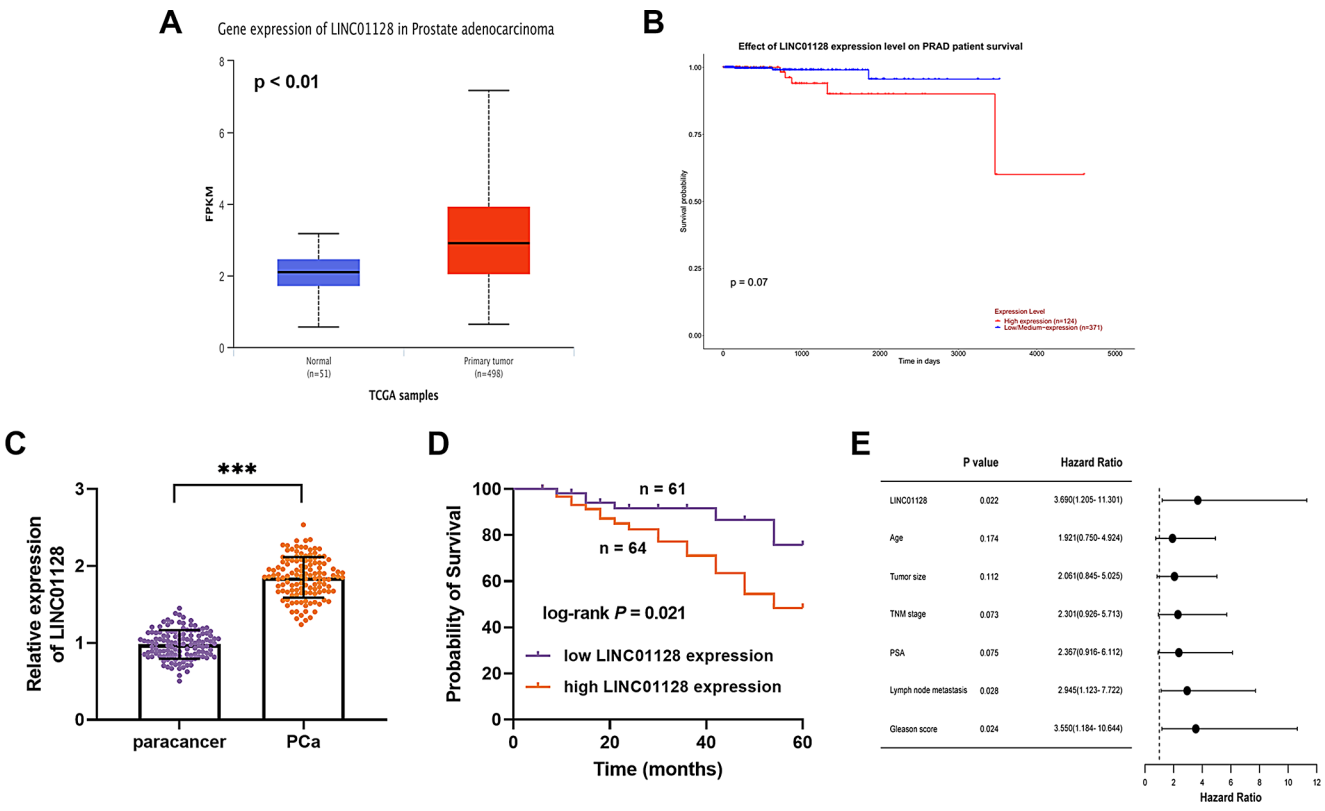

Fig. 1 The clinical significance of LINC01128 expression in PCa. **(A)** The UALCAN database revealed the upregulation of LINC01128 in PCa. FPKM: fragments per kilobase per million mapped reads exon model (a standardized metric utilized for the analysis of gene expression in RNA-seq data). **(B)** The UALCAN database revealed that patients with PCa who exhibited high levels of LINC01128 demonstrated a significantly reduced survival compared to those with low levels of LINC01128. **(C)** The expression of LINC01128 was markedly elevated in PCa tissues ($n=125$) relative to paracancerous normal tissues ($n=125$). **(D)** The survival of patients with PCa who exhibited high levels of LINC01128 ($n=64$) was less favorable than those with low LINC01128 expression ($n=61$). **(E)** Cox regression analysis revealed that LINC01128 serves as an independent prognostic indicator for PCa. ***$P<0.001$

Table 1 The association between LINC01128 expression and pathological features of PCa patients

Variables	Cases ($n=125$)	LINC01128 expression		P value
		Low ($n=61$)	High ($n=64$)	
Age (years)				0.312
≤60	57	25	32	
>60	68	36	32	
Tumor size (cm)				0.754
≤4	72	36	36	
>4	53	25	28	
TNM stage				0.019
I–II	75	43	32	
III	50	18	32	
PSA (ng/mL)				0.108
≤10	75	41	34	
>10	50	20	30	
Lymph node metastasis				0.001
Absence	85	50	35	
Presence	40	11	29	
Gleason score				0.039
≤7	64	37	27	
>7	61	24	37	

Abbreviations: PCa, prostate cancer; TNM, tumor-node-metastasis; PSA, prostate specific antigen

of LINC01128 in epithelial-mesenchymal transition (EMT), the expression of EMT markers was measured following the knockdown of LINC01128. In 22Rv1 and DU145 cells, the silencing of LINC01128 resulted in a notable elevation in the protein level of E-cadherin and a substantial reduction in the protein level of N-cadherin and Vimentin (Fig. 2F).

LINC01128 sponged miR-27b-3p in PCa cells

As stated in the preceding research, a targeting relationship was reported between LINC01128 and miR-27b-3p. The analysis of the starBase database demonstrated a notable decline in the relative expression of miR-27b-3p in PCa (Fig. 3A). Additionally, the level of miR-27b-3p was markedly diminished in the PCa tissues included in this study (Fig. 3B), exhibiting a negative correlation with LINC01128 (r = -0.757, $P<0.001$, Fig. 3C). Consistently, miR-27b-3p demonstrated decreased expression in PCa cells (Fig. 3D). Furthermore, a targeting relationship was confirmed in DU145 cells based on the complementary sites of miR-27b-3p and LINC01128. The miR-27b-3p mimic was observed to reduce the luciferase activity of LINC01128-WT, while the miR-27b-3p inhibitor displayed a contrary effect (Fig. 3E).

LINC01128 regulated cellular processes in PCa via miR-27b-3p

In DU145 cells, the levels of miR-27b-3p were effectively enhanced by the transfection of the miR-27b-3p mimic (Fig. 4A). Overexpressed miR-27b-3p markedly repressed the growth, migration, and invasion of DU145 cells (Fig. 4B-D). The silencing of LINC01128 resulted in an increased level of miR-27b-3p, which was reversed by miR-27b-3p inhibitor (Fig. 5A). The downregulation of miR-27b-3p markedly diminished the inhibitory effects of LINC01128 knockdown on cell growth, migration, and invasion (Fig. 5B-D). Furthermore, a reduction in miR-27b-3p expression mitigated the impact of LINC01128 silencing on EMT markers in DU145 cells (Fig. 5E).

Discussion

At present, the pathogenesis of PCa remains to be fully elucidated. The prevailing hypothesis suggests that PCa is mainly linked to the abnormalities of prostate cell proliferation and apoptosis, inflammatory stress, and tumor cell immune tolerance (Debelec Butuner et al. 2023). The exploration of novel biomarkers is not only advantageous for the monitoring of PCa progression, but also for the development of personalized treatments. It has been documented that lncRNAs possess the capacity to modulate the progression of PCa (Haghighi et al. 2023). For instance, lncRNA MAGI2-AS3 was identified as a suppressor in the progression of PCa (Yang et al. 2023). Additionally, several lncRNAs are implicated in the promotion of PCa progression, including TMPO-AS1, MNX1-AS1, and LINC01126 (Wang et al. 2023; Liang et al. 2022; Cai et al. 2024). According to a previous report, LINC01128 was shown to be overexpressed in PCa in the GSE46602 dataset (Ye et al. 2018). In this study, it was found that LINC01128 exhibited elevated levels in PCa tissues and was related to lymph node metastasis, higher Gleason score, and advanced TNM stage. Furthermore, an elevated expression of LINC01128 was observed to correlate with a reduced survival time in patients with PCa. LINC01128 was identified as an independent predictor of poor prognosis in PCa. Moreover, the analysis revealed that lymph node metastasis and Gleason score were risk factors for patient mortality, which are significant indicators for evaluating the degree of malignancy and prognosis of PCa (Ge et al. 2023a, b). These findings indicate that the upregulation of LINC01128 is potentially linked to malignant progression and poor prognosis in PCa.

It has been established that lncRNAs exert a pivotal regulatory influence over a series of vital cellular activities. Dysregulated expression of lncRNAs was observed to be

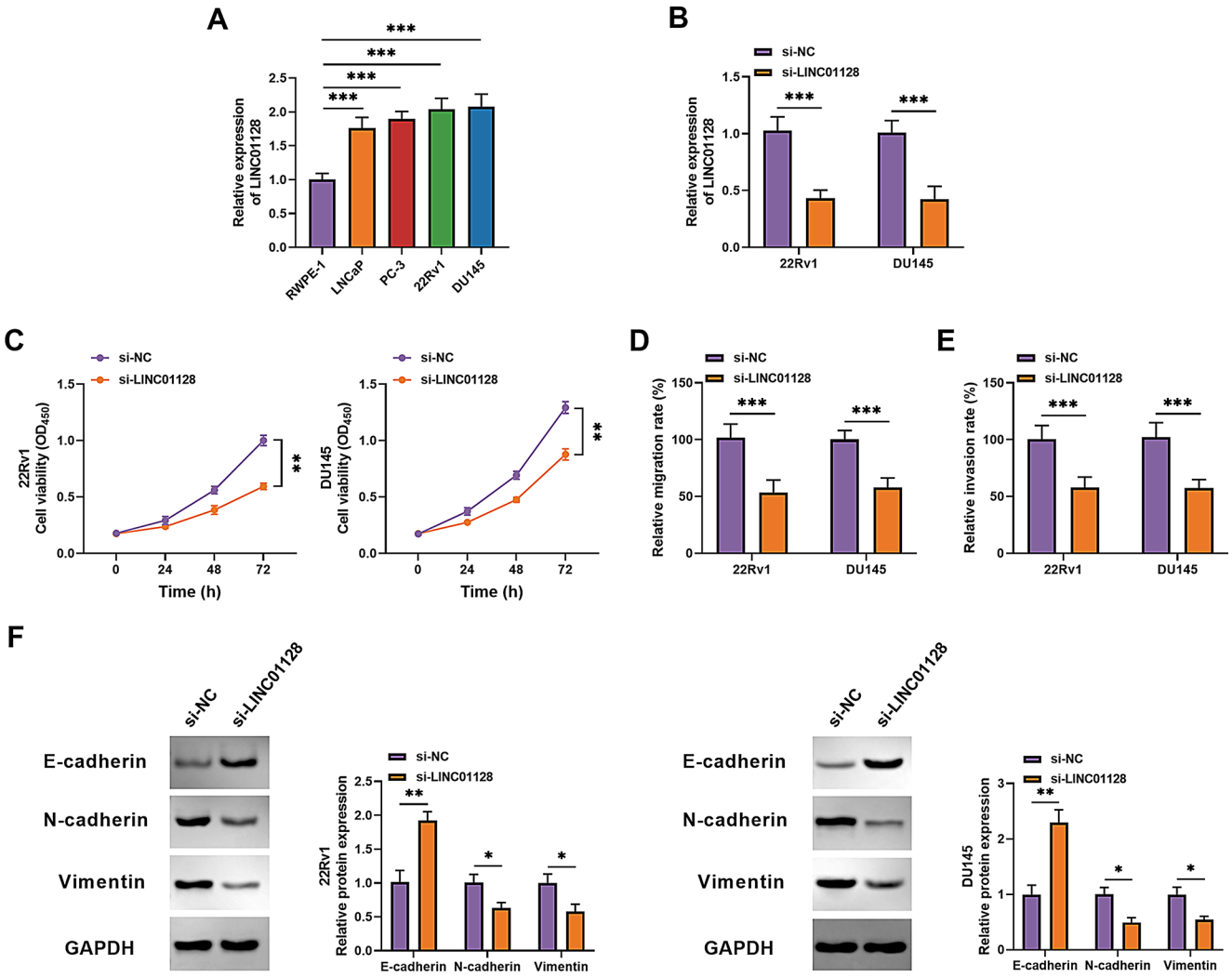

Fig. 2 Effects of LINC01128 on cellular processes in PCa. **(A)** In PCa cells, LINC01128 expression was found to be higher in comparison to normal prostate cells. **(B)** In 22Rv1 and DU145 cells, LINC01128 expression was found to be repressed by the transfection of si-LINC01128. **(C)** The silencing of LINC01128 markedly diminished the viability of 22Rv1 and DU145 cells as detected by CCK-8 assay at 0, 24, 48, and 72 h post-transfection. **D and E.** The knockdown of LINC01128 exhibited a substantial inhibitory effect on the migration (**D**) and invasion (**E**) of 22Rv1 and DU145 cells as detected by the Transwell assay at 48 h post-transfection. **F.** The silencing of LINC01128 demonstrated the ability to regulate EMT markers, including E-cadherin, N-cadherin, and Vimentin. $^{*}P<0.05$, $^{**}P<0.01$, $^{***}P<0.001$

related to the onset and progression of numerous diseases, including malignant tumors (Ge et al. 2023a, b; Park et al. 2022). In PCa, lncRNA VPS9D1-AS1 was discovered to be overexpressed, exerting a promotional effect on the phenotype of tumor cells (Wu et al. 2023). Increasing evidence indicates that LINC01128 modulates the malignant phenotype of tumor cells, including those associated with pancreatic, cervical, and colorectal cancers (Zhong et al. 2022; Hu et al. 2019; Zhou et al. 2024). At present, EMT is recognized as one of the key mechanisms of PCa invasion and metastasis (Gogola et al. 2023; Goncharov et al. 2024). Abnormalities in the structure and function of adhesion molecules in tumor cells during EMT result in a decrease in tumor cell-to-cell adhesion, accompanied by an increase in motility. The hallmark of EMT is the repression of epithelial cell markers and the activation of mesenchymal cell markers (Tomecka et al. 2024). Here, silencing of LINC01128 in PCa cells suppressed cell proliferation and metastasis, as well as an inhibitory effect on EMT. These findings suggest that LINC01128 might function as a promoter of PCa.

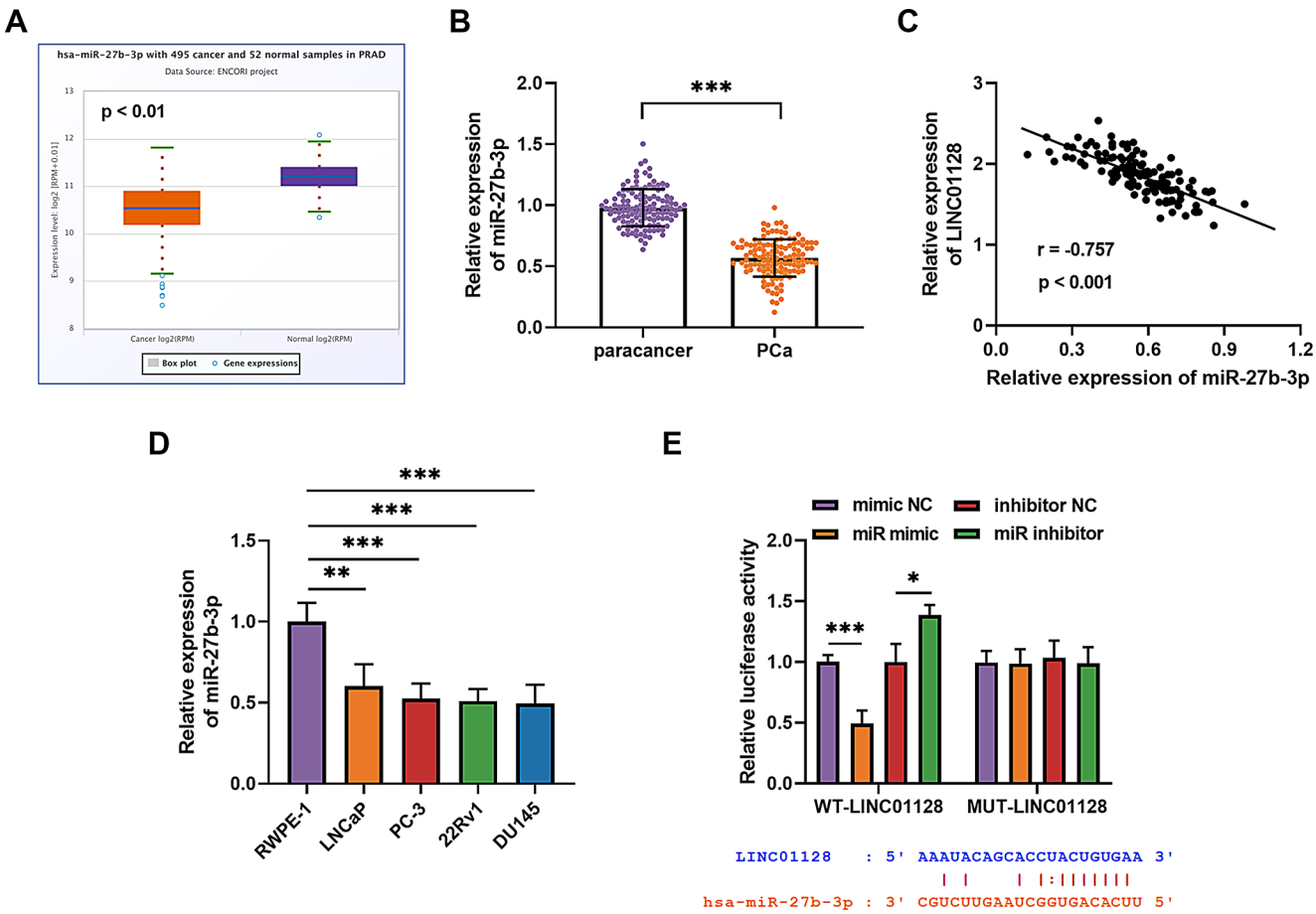

Fig. 3 The targeting relationship between LINC01128 and miR-27b-3p. **A.** The analysis of the starBase database disclosed that miR-27b-3p expression was reduced in PCa ($n=495$) compared to normal samples ($n=52$). **B and C.** miR-27b-3p was found to be downregulated in PCa tissues included in this study (**B**), exhibiting a negative correlation with LINC01128 (**C**). **D.** Low expression of miR-27b-3p was observed in PCa cells. **E.** The luciferase activity of LINC01128 in DU145 cells was inversely regulated by miR-27b-3p. $^{*}P<0.05$, $^{**}P<0.01$, $^{***}P<0.001$

Typically, lncRNAs bind to miRNAs in a complementary manner, exerting their effects by acting as molecular sponges to inhibit the function of miRNAs (Kazimierczyk and Wrzesinski 2021). For example, recent research has indicated that lncRNA HCG18 contributes to the progression of PCa by regulating miR-512-3p (Zhu et al. 2024). In gliomas, LINC01128 was found to facilitate tumor progression by targeting miR-27b-3p. In other neoplasms, such as colorectal cancer, esophageal squamous cell carcinoma, and hepatocellular carcinoma, miR-27b-3p was demonstrated to play a pivotal role during tumor progression (Gao et al. 2024; Zhu et al. 2022; Lin et al. 2022). Several studies have confirmed the regulatory role of miR-27b-3p in PCa progression (Yang et al. 2024; Chen et al. 2023). In the present study, miR-27b-3p suppressed the growth and metastasis of PCa cells. The knockdown of LINC01128 exhibited a considerable impact on miR-27b-3p expression. Concurrently, reducing the expression of miR-27b-3p could mitigate the inhibitory effect of silencing LINC01128 on the cellular process in PCa. Therefore, the mechanism by which LINC01128 exerts its tumor-promoting potential may be attributed to this regulation of miR-27b-3p.

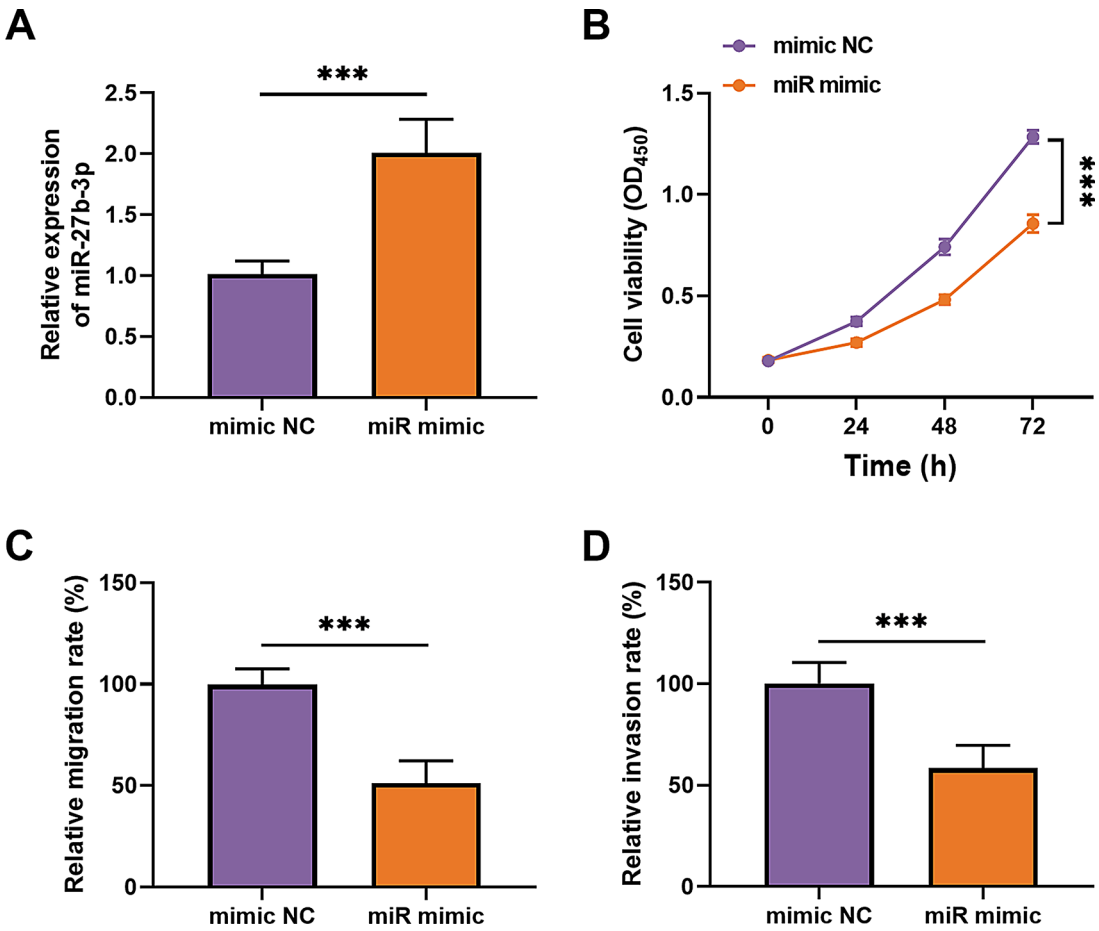

Fig. 4 Effects of miR-27b-3p on the function of PCa cells. **(A)** The expression of miR-27b-3p was markedly augmented in PCa cells following transfection with miR-27b-3p mimic. **(B)** Overexpression of miR-27b-3p remarkably reduced the viability of DU145 cells. **C and D.** Overexpression of miR-27b-3p showed marked suppression of migration (**C**) and invasion (**D**) of DU145 cells. ***$P<0.001$

However, some limitations of the study should be noted. Firstly, a relatively small sample size was employed, and the samples were obtained from a single center. To validate the clinical analysis results, future studies should include a greater number of PCa patients. Secondly, additional research is required to reveal the downstream genes and the signaling pathways involved in PCa progression. Finally, in vivo experiments are an essential tool for tumor research, and animal experiments are needed to verify the impact of LINC01128 on tumor progression in future studies.

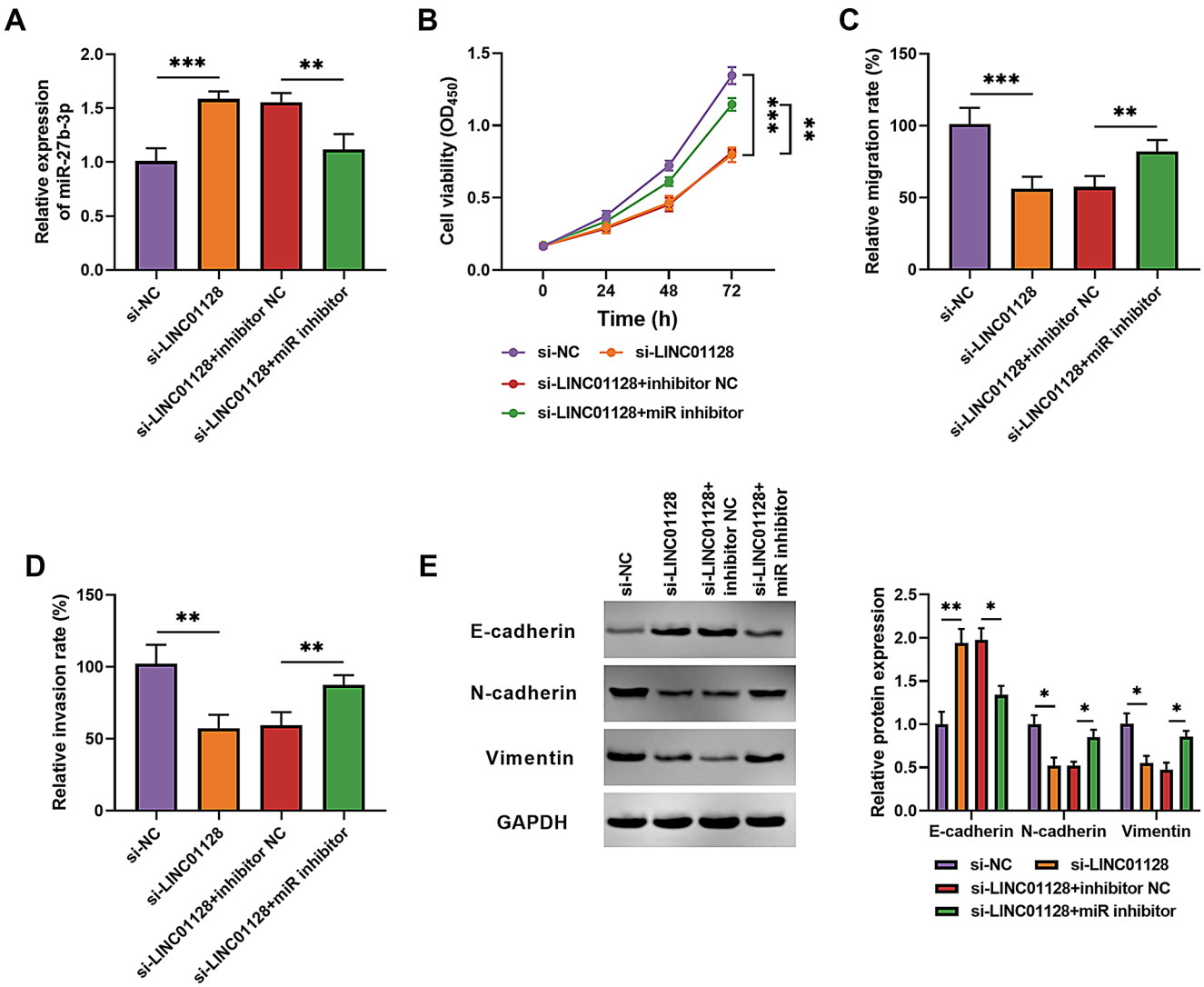

Fig. 5 miR-27b-3p mediated the function of LINC01128 in PCa. **A.** In DU145 cells, the silencing of LINC01128 enhanced the levels of miR-27b-3p, while this effect could be reversed by miR-27b-3p inhibitor. **B-D.** Decreased expression of miR-27b-3p mitigated the suppression of proliferation (**B**), migration (**C**), and invasion (**D**) of DU145 cells caused by LINC01128 silencing. **E.** Inhibition of miR-27b-3p was found to reverse the regulatory effects of LINC01128 silencing on E-cadherin, N-cadherin, and Vimentin. $^{*}P<0.05$, $^{**}P<0.01$, $^{***}P<0.001$

In conclusion, upregulation of LINC01128 was associated with the malignant progression of PCa and poor prognosis of patients. Moreover, LINC01128 may be involved in PCa progression as a tumor promoter mediated by miR-27b-3p.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00432-025-06153-6.

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Data availability The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethical approval This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Longhua Hospital Shanghai University of Traditional Chinese Medicine.

Consent to participate Informed consent was obtained from all individual participants included in the study.

Consent to publish Not applicable.

Competing interests The authors declare no competing interests.

References

Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I et al (2024) Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 74(3):229–263

Cai Y, Chen M, Gong Y, Tang G, Shu Z, Chen J et al (2024) Androgen-repressed LncRNA LINC01126 drives castration-resistant prostate cancer by regulating the switch between O-GlcNAcylation and phosphorylation of androgen receptor. Clin Transl Med 14(1):e1531

Chen Y, Zhao J, Duan Z, Gong T, Chen W, Wang S et al (2019) miR–27b–3p and miR–607 cooperatively regulate BLM gene expression by directly targeting the 3'–UTR in PC3 cells. Mol Med Rep 19(6):4819–4831

Chen B, Liu C, Long H, Bai G, Zhu Y, Xu H (2023) N(6)–methyladenosine–induced long non–coding RNA PVT1 regulates the miR–27b–3p/BLM axis to promote prostate cancer progression. Int J Oncol 62(1):16

Chinese guidelines for (2019) Diagnosis and treatment of prostate cancer 2018 (English version). Chin J Cancer Res 31(1):67–83

Debelec Butuner B, Ertunc Hasbal N, Isel E, Roggenbuck D, Korkmaz KS (2023) Androgen receptor contributes to repairing DNA damage induced by inflammation and oxidative stress in prostate cancer. Turk J Biol 47(5):325–335

Dinescu S, Ignat S, Lazar AD, Constantin C, Neagu M, Costache M (2019) Epitranscriptomic signatures in LncRNAs and their possible roles in Cancer. Genes (Basel) 10(1):52

Gao Q, Cheng X, Gao X (2024) Circ_0089761 accelerates colorectal cancer metastasis and immune escape via miR-27b-3p/PD-L1 axis. Physiol Rep 12(23):e70137

Ge Q, Li J, Yang F, Tian X, Zhang M, Hao Z et al (2023a) Molecular classifications of prostate cancer: basis for individualized risk stratification and precision therapy. Ann Med 55(2):2279235

Ge WJ, Huang H, Wang T, Zeng WH, Guo M, Ren CR et al (2023b) Long non-coding RNAs in hepatocellular carcinoma. Pathol Res Pract 248:154604

Gogola S, Rejzer M, Bahmad HF, Abou-Kheir W, Omarzai Y, Poppiti R (2023) Epithelial-to-Mesenchymal Transition-Related markers in prostate cancer: from bench to bedside. Cancers (Basel) 15(8):2309

Goncharov AP, Vashakidze N, Kharaishvili G, Epithelial-Mesenchymal Transition (2024) A fundamental cellular and microenvironmental process in benign and malignant prostate pathologies. Biomedicines 12(2):418

Haghighi R, Castillo-Acobo RY, A HA, Ehymayed HM, Alhili F, Mirzaei M et al (2023) A thorough Understanding of the role of LncRNA in prostate cancer pathogenesis; current knowledge and future research directions. Pathol Res Pract 248:154666

Hu Y, Ma Y, Liu J, Cai Y, Zhang M, Fang X (2019) LINC01128 expedites cervical cancer progression by regulating miR-383-5p/SFN axis. BMC Cancer 19(1):1157

Kanesvaran R, Castro E, Wong A, Fizazi K, Chua MLK, Zhu Y et al (2022) Pan-Asian adapted ESMO clinical practice guidelines for the diagnosis, treatment and follow-up of patients with prostate cancer. ESMO Open 7(4):100518

Kazimierczyk M, Wrzesinski J (2021) Long Non-Coding RNA epigenetics. Int J Mol Sci 22(11):6166

Le Guevelou J, Ploussard G, Roubaud G, Sargos P (2024) The testosterone replacement therapy for prostate cancer patients: Time to take the leap? Andrology 13(2):173–175

Liang D, Tian C, Zhang X (2022) LncRNA MNX1–AS1 promotes prostate cancer progression through regulating miR–2113/MDM2 axis. Mol Med Rep 26(1):231

Lin XH, Liu ZY, Zhang DY, Zhang S, Tang WQ, Li DP et al (2022) circRanGAP1/miR-27b-3p/NRAS Axis May promote the progression of hepatocellular carcinoma. Exp Hematol Oncol 11(1):92

Liu B, Jiang HY, Yuan T, Zhou WD, Xiang ZD, Jiang QQ et al (2021) Long Non-coding RNA AFAP1-AS1 facilitates prostate Cancer progression by regulating miR-15b/IGF1R Axis. Curr Pharm Des 27(41):4261–4269

Nelson WG, Brawley OW, Isaacs WB, Platz EA, Yegnasubramanian S, Sfanos KS et al (2022) Health inequity drives disease biology to create disparities in prostate cancer outcomes. J Clin Invest 132(3):e155031

Pan SY, Chen WC, Huang CP, Hsu CY, Chang YH (2023) The association of prostate Cancer and urinary tract infections: A new perspective of prostate Cancer pathogenesis. Med (Kaunas) 59(3):483

Park EG, Pyo SJ, Cui Y, Yoon SH, Nam JW (2022) Tumor immune microenvironment LncRNAs. Brief Bioinform 23(1):bbab504

Rafikova G, Gilyazova I, Enikeeva K, Pavlov V, Kzhyshkowska J (2023) Prostate cancer: genetics, epigenetics and the need for immunological biomarkers. Int J Mol Sci24(16):12797

Shree B, Das K, Sharma V (2023) Emerging role of transforming growth factor-beta-regulated long non-coding RNAs in prostate cancer pathogenesis. Cancer Pathog Ther 1(3):195–204

Taheri M, Badrlou E, Hussen BM, Kashi AH, Ghafouri-Fard S, Baniahmad A (2023) Importance of long non-coding RNAs in the pathogenesis, diagnosis, and treatment of prostate cancer. Front Oncol 13:1123101

Tomecka P, Kunachowicz D, Gorczynska J, Gebuza M, Kuznicki J, Skinderowicz K et al (2024) Factors determining Epithelial-Mesenchymal transition in Cancer progression. Int J Mol Sci 25(16):8972

Wang M, Yin C, Wu Z, Wang X, Lin Q, Jiang X et al (2023) The long transcript of LncRNA TMPO-AS1 promotes bone metastases of

prostate cancer by regulating the CSNK2A1/DDX3X complex in Wnt/beta-catenin signaling. Cell Death Discov 9(1):287

Wu C, Chen J, Wang D (2023) LncRNA VPS9D1-AS1 regulates miR-187-3p/fibroblast growth factor receptor-like 1 axis to promote proliferation, migration, and invasion of prostate cancer cells. Chin J Physiol 66(5):295–305

Yang G, Li T, Liu J, Quan Z, Liu M, Guo Y et al (2023) LncRNA MAGI2-AS3 suppresses castration-resistant prostate cancer proliferation and migration via the miR-106a-5p/RAB31 axis. Genomics 115(2):110599

Yang L, Ruan Y, Chen B, Zhu Y, Xu H (2024) Circ_0001671 regulates prostate cancer progression through miR-27b-3p/BLM axis. Sci Rep 14(1):12181

Ye Y, Li SL, Wang SY (2018) Construction and analysis of mRNA, MiRNA, LncRNA, and TF regulatory networks reveal the key genes associated with prostate cancer. PLoS ONE 13(8):e0198055

Zhang Q, Shao W, Li Y, Liu L, Chen W, Wang C et al (2022) Long non-coding RNA LINC01128 affects proliferation, migration, and invasion of glioma cells by regulating miR-27b-3p. Folia Neuropathol 60(3):338–345

Zhong M, Fang Z, Ruan B, Xiong J, Li J, Song Z (2022) LINC01128 facilitates the progression of pancreatic cancer through up-regulation of LDHA by targeting miR-561-5p. Cancer Cell Int 22(1):93

Zhou X, Li Y, Wu L, Tian C, Wu X (2024) Upregulated LncRNA LINC01128 in colorectal cancer accelerates cell growth and predicts malignant prognosis through sponging miR-363-3p. J Cancer Res Clin Oncol 150(5):276

Zhu C, Bi W, Li H, Wang W (2022) CircLONP2 accelerates esophageal squamous cell carcinoma progression via direct MiR-27b-3p-ZEB1 Axis. Front Oncol 12:822839

Zhu Y, Wang Z, Li H, Ren Z, Zi T, Qin X et al (2024) LncRNA HCG18 promotes prostate cancer progression by regulating the miR-512-3p/HK-2 axis. Asian J Urol 11(4):575–585