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LncRNA PSMA3-AS1 promotes colorectal cancer cell migration and invasion *via* regulating miR-4429

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Abstract. – OBJECTIVE: Many studies have revealed that long non-coding RNAs (lncRNAs) are related to various cancers, including colorectal cancer (CRC). This study aims to explore the biological function of lncRNA PSMA3-AS1 in CRC progression.

MATERIALS AND METHODS: The expression levels of PSMA3-AS1 and miR-4429 were assessed by RT-qPCR. CRC progression was explored by cell viability, migration, and invasion using CCK-8 and transwell assays. The interaction between PSMA3-AS1 and miR-4429 was verified by bioinformatics analysis, Dual-Luciferase assay, and RIP assay.

RESULTS: It was found that PSMA3-AS1 expression was increased and miR-4429 expression was decreased in CRC tissues and cells. In addition, PSMA3-AS1 interference markedly hindered the proliferation, migration, and invasion of CRC cells. MiR-4429 was a direct target of PSMA3-AS1, and the knockdown of PSMA3-AS1 significantly suppressed miR-4429 expression. The depletion of PSMA3-AS1 inhibited CRC progression, which was neutralized by miR-4429 inhibitor.

CONCLUSIONS: PSMA3-AS1 accelerated CRC progression by regulating miR-4429 expression, which could be used as a potential therapeutic target for CRC patients.

Key Words:
PSMA3-AS1, MiR-4429, Colorectal cancer.

Introduction

Colorectal cancer (CRC) is one of the most common malignancies[1-3]. In the past decade, significant advances have been achieved in the diagnosis and treatment of CRC[4]. However, the unlimited proliferation and high metastasis of tumors lead to poor prognosis in patients with CRC[5]. Therefore, a better understanding of the potential mechanisms correlated with proliferation and metastasis in CRC is urgent.

Long non-coding RNAs (lncRNAs) are a class of RNAs with more than 200 nucleotides (nts) in length[6,7]. LncRNAs have been shown to play vital roles in several biological processes, such as cell apoptosis, metastasis, proliferation, and invasion[8-10]. Chen et al[11] have demonstrated the regulatory roles of lncRNAs in multiple cancers, including CRC. For example, lncRNA CCAT1 contributed to CRC tumorigenesis through suppression of miR-181b-5p[12]. LncRNA FOXD2-AS1 facilitated the malignancy of CRC *via* the miR-25-3p/Sema4c axis[13]. LncRNA UCA1 regulated the miR-143/MYO6 axis to accelerate the development of CRC[14]. The lncRNA SLCO4A1-AS1/miR-508-3p axis regulated CRC development by targeting PARD3[14]. However, the biological function of PSMA3-AS1 in CRC remains obscure.

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MicroRNAs (miRNAs) have been reported to be vital regulators in CRC tumorigenesis[16-18]. The supplementation of miR-1258 suppressed cell proliferation by regulating E2F8 in CRC[19]. MiR-4319 inhibited CRC development by directly targeting ABTB1[20]. The upregulation of miR-141 remarkably restrained CRC growth by regulating TRAF5[21]. MiR-335-5p suppressed the proliferation, migration, and invasion of CRC cells through inhibiting LDHB[22]. However, the exact mechanism by which miR-4429 modulates the progression of CRC remains unclear.

In this study, lncRNA PSMA3-AS1 facilitated CRC development *via* regulating miR-4429. Our findings revealed that lncRNA PSMA3-AS1 might be a new diagnostic biomarker and therapeutic target for CRC treatment.

Materials and Methods

Patients and Specimens

30 pairs of tumor and adjacent healthy tissues were obtained from CRC patients who had surgical treatment at the Second Affiliated Hospital of Nanjing Medical University. Specimens were stored at -80°C immediately after surgical resection. This research was approved by the Ethics Committee of Nanjing Medical University (Nanjing, Jiangsu, China). Written informed consent was obtained from each participant.

Cell Culture

Normal colorectal mucosa epithelial cells (NCM460) and human CRC cell lines (HT29, SW837, and HCT8) were purchased from the American Type Culture Collection (ATCC). All cells were cultivated with RPMI-1640 medium (Haoranbio, Shanghai, China) with 10% FBS (Gibco; Thermo Fisher Scientific, Waltham, MA, USA). The cells were cultured at 37°C in a humidified incubator containing 5% CO_2.

Cell Transfection

Short hairpin RNA (shRNA) targeting PSMA3-AS1 (shPSMA3-AS1), negative control (shNC), miR-4429 mimics, control group (NC mimics), miR-4429 inhibitor, and negative control (NC inhibitor) were purchased from GenePharma (Shanghai, China). HT29 and SW837 cells were transfected with these oligonucleotides using Lipofectamine 2000 transfection reagent (Invitrogen, Carlsbad, CA, USA).

RT-qPCR

Total RNA was extracted from tissues or cells using TRIzol (TaKaRa Bio, Inc., Dalian, China). Subsequently, synthesis of cDNA was performed using PrimeScript RT Reagent kit (TaKaRa Bio, Inc., Dalian, China). RT-qPCR was conducted using the SYBR Green PCR kit (TaKaRa Bio, Inc., Dalian, China). U6 and GAPDH were adopted as the internal controls. The relative expression of genes was analyzed using the $2^{-\Delta\Delta Cq}$ method.

Cell Counting Kit-8 (CCK-8) Assay

HT29 and SW837 cells were incubated in 96-well plates ($1x10^4$ cells per well) and cultured for 0, 24, 48, or 72 h. Then, 10 μL CCK-8 solution was added to each well and appended for 2 h incubation. The optical density (OD) of each well was detected at a wavelength of 450 nm using a microplate reader.

Transwell Assay

For the migration assay, transfected cells (1×10^4) in serum-free culture medium were added to the upper chamber. Afterward, 10% FBS was added to the lower chamber. After 24 h, 0.1% crystal violet was performed stain cells, and cells were counted under a light microscope. For cell invasion, the chamber was precoated with Matrigel (Becton Dickinson, Franklin Lakes, NJ, USA). The other steps were consistent with the cell migration assay.

Dual-Luciferase Reporter Assay

The wild-type (WT) or mutant (Mut) reporter plasmids of PSMA3-AS1 (PSMA3-AS1-WT and PSMA3-AS1-Mut) were obtained from GenePharma (Shanghai, China). Then, HT29 and SW837 cells were co-transfected with PSMA3-AS1-WT or PSMA3-AS1-Mut and miR-4429 mimics or NC mimics using Lipofectamine 3000 transfection reagent. The Luciferase activities were determined by the Dual-Luciferase Reporter System.

RIP Assay

RIP assay was carried out using Magna RIP RNA-Binding Protein Immunoprecipitation Kit (Millipore, MA, USA). The transfected cells were dissolved in RIP lysis buffer, and then, cell lysate was incubated with magnetic beads bound with the Ago2 antibody. IgG was used as a control group. Then, immunoprecipitated RNA was detected RT-qPCR assay.

Statistical Analysis

The data were assessed using GraphPad Prism 7 (LaJolla, CA, USA) and expressed as the mean ± standard deviation (SD). Student's *t*-test or a one-way ANOVA was used to evaluate the differences between groups. Survival analysis was performed using Kaplan-Meier survival curves and log-rank tests. The correlation between PSMA3-AS1 and miR-4429 level was assessed by Pearson's correlation analysis. $p<0.05$ represented statistical significance.

Results

PSMA3-AS1 Was Highly Expressed in CRC

To investigate the function of PSMA3-AS1 in CRC, its expression was detected in CRC tissues by RT-qPCR. The results revealed that a distinct upregulation of PSMA3-AS1 level in CRC tissues compared to adjacent normal tissues (Figure 1A). Then, PSMA3-AS1 expression was measured in CRC cells. The data showed that PSMA3-AS1 expression was remarkably enhanced in CRC cells (HT29, SW837, and HCT8) compared with normal cells NCM460 (Figure 1B). Furthermore, as shown in Figure 1C, the high expression of PSMA3-AS1 in CRC patients displayed poorer prognosis. Taken together, PSMA3-AS1 was prominently upregulated in CRC and might be implicated in the development of CRC.

Interference of PSMA3-AS1 Suppressed CRC Progression

To explore the biological function of PSMA3-AS1 in CRC, HT29 and SW837 cells were transfected with three shRNAs against PSMA3-AS1 or shNC. The transfection efficiency in HT29 and SW837 cells was confirmed by RT-qPCR (Figure 2A). CCK-8 assay indicated that the depletion of PSMA3-AS1 impeded the viability of HT29 and SW837 cells (Figure 2B). Moreover, PSMA3-AS1 silencing resulted in a restriction of cell migration and invasion in HT29 and SW837 cells (Figure 2C and D). Overall, these results demonstrated that the knockdown of PSMA3-AS1 could restrain the proliferation, migration, and invasion of CRC cells.

PSMA3-AS1 Acted as a Sponge for MiR-4429

It has been declared that lncRNAs are implicated in tumorigenesis by competitively binding with miRNAs. StarBase was performed to predict the binding sites of PSMA3-AS1 and miR-4429 (Figure 3A). To further affirm this prediction, Luciferase reporter assay was utilized by establishing luciferase reporter vectors PSMA3-AS1-WT and PSMA3-AS1-Mut. Results indicated that transfection of miR-4429 mimics reduced the Luciferase activity of PSMA3-AS1-WT reporter in HT29 and SW837 cells, but had no significant effect on PSMA3-AS1-Mut activity (Figure 3B). RIP assay in-

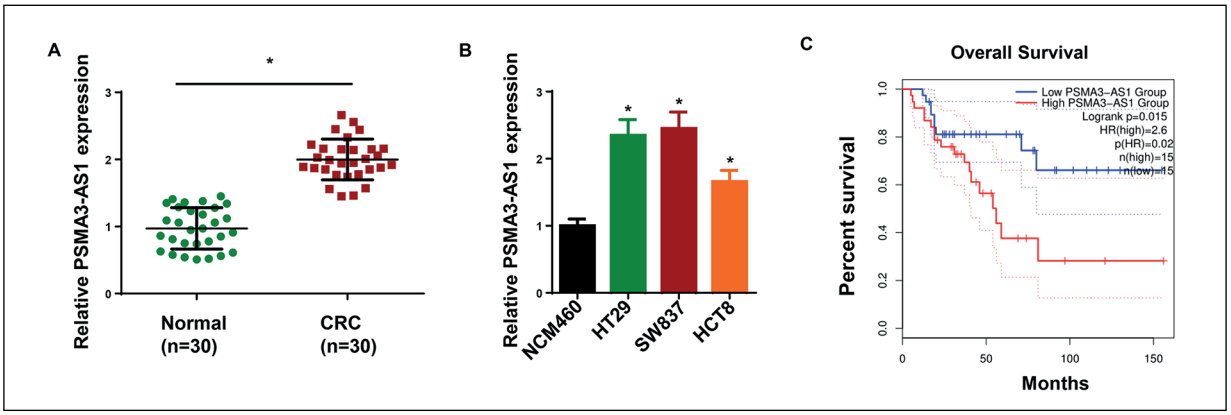

Figure 1. PSMA3-AS1 was highly expressed in CRC. **A,** The expression levels of PSMA3-AS1 in CRC tissues (n=30) and adjacent normal tissues (n=30) were measured by RT-qPCR. **B,** The expression levels of PSMA3-AS1 in normal colorectal mucosa epithelial cells (NCM460) and human CRC cell lines (HT29, SW837, and HCT8) were detected by RT-qPCR. **C,** Kaplan-Meier analysis indicated the association between PSMA3-AS1 expression and the overall survival of CRC patients. The data were presented as mean ± SD (*$p < 0.05$).

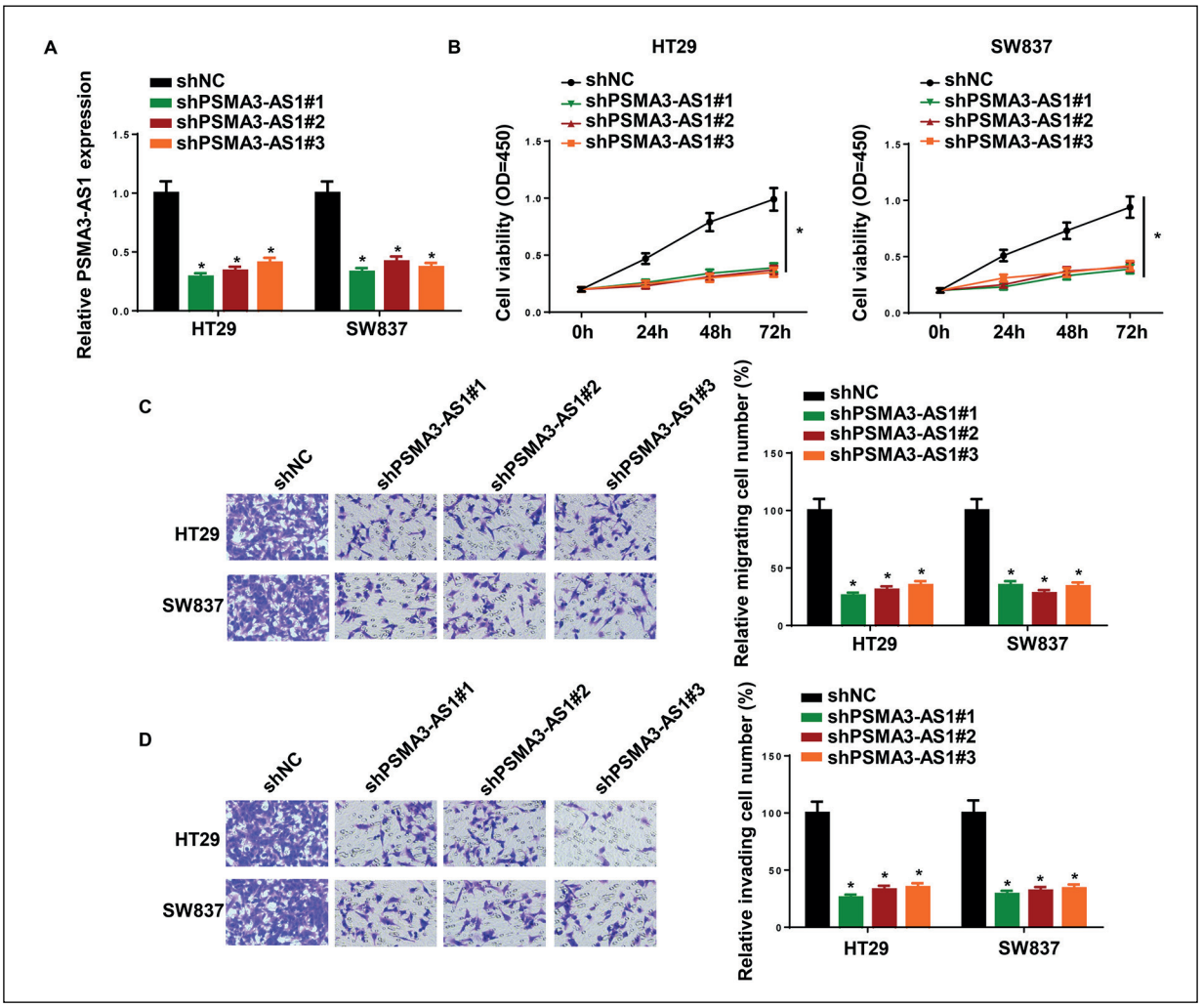

Figure 2. Interference of PSMA3-AS1 suppressed CRC progression. **A,** PSMA3-AS1 expression levels in HT29 and SW837 cells transfected with shNC or shPSMA3-AS1 lines (shPSMA3-AS1#1, shPSMA3-AS1#2, and shPSMA3-AS1#3) were detected by RT-qPCR. **B,** CCK-8 assay was used to detect viability of HT29 and SW837 cells transfected with shNC or shPSMA3-AS1 (shPSMA3-AS1#1, shPSMA3-AS1#2, and shPSMA3-AS1#3). **C,** and **D,** Transwell assay was used to measure migration and invasion of HT29 and SW837 cells transfected with shNC or shPSMA3-AS1 (shPSMA3-AS1#1, shPSMA3-AS1#2, and shPSMA3-AS1#3) (magnification ×40). The data were presented as mean ± SD (*$p < 0.05$).

dicated that PSMA3-AS1 and miR-4429 were highly enriched by Ago2, but the enrichment effect of IgG was not evident (Figure 3C). Subsequently, RT-qPCR assay displayed that miR-4429 was decreased in CRC tissues and cells (Figure 3D and E). Moreover, there was an inverse correlation between PSMA3-AS1 and miR-4429 in CRC tissues (Figure 3F). In addition, miR-4429 expression was increased in HT29 and SW837 cells by depleting PSMA3-AS1 (Figure 3G). Collectively, these results indicated that PSMA3-AS1 directly targeted miR-4429 in CRC cells.

PSMA3-AS1 Regulated Cell Viability, Migration, and Invasion in CRC Cells by Sponging MiR-4429

To further investigate whether PSMA3-AS1 could regulate the progression of CRC by sponging miR-4429, shNC, shPSMA3-AS1#1, shPSMA3-AS1#1+NC inhibitor, and shPSMA3-AS1#1+miR-4429 inhibitor was transfected into HT29 and SW837 cells. The inhibition of miR-4429 abolished the suppressive effects of PSMA3-AS1 silencing on PSMA3-AS1 level in HT29 and SW837 cells (Figure 4A). The depletion of PSMA3-AS1 remarkably suppressed the

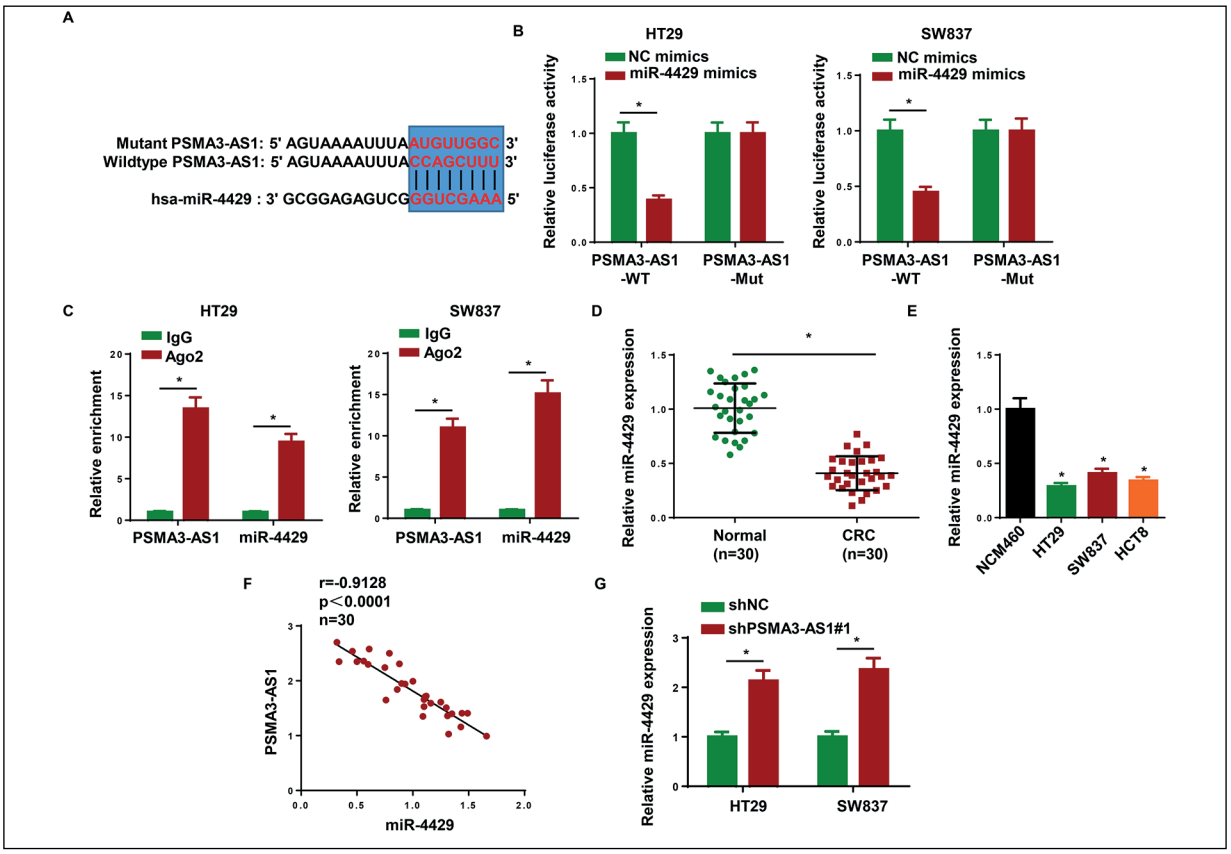

Figure 3. PSMA3-AS1 acted as a sponge for miR-4429. **A,** The predicted binding sites of PSMA3-AS1 and miR-4429. **B,** The Luciferase activity of HT29 and SW837 cells co-transfected with the miR-4429 mimics and PSMA3-AS1-WT or PSMA3-AS1-Mut was detected Dual-Luciferase reporter assay. **C,** Correlations between PSMA3-AS1 and miR-4429 detected by RIP assay. **D,** The expression of miR-4429 in CRC tissues (n=30) and adjacent normal tissues (n=30) were detected by RT-qPCR. **E,** The expression of miR-4429 in normal colorectal mucosa epithelial cells (NCM460) and human CRC cell lines (HT29, SW837, and HCT8) was measured by RT-qPCR. **F,** The relationship between PSMA3-AS1 and miR-4429 expressions in CRC tissues. **G,** The expression of miR-4429 in HT29 and SW837 cells transfected with shNC or shPSMA3-AS1#1 was analyed by RT-qPCR. The data were presented as mean ± SD (*$p < 0.05$).

viability, migration, and invasion of HT29 and SW837 cells, whereas the effects could be counteracted by miR-4429 inhibitor (Figure 4B-D). In summary, our results revealed that PSMA3-AS1 accelerated the progression of CRC by regulating miR-4429.

Discussion

It has been demonstrated that aberrant expression of lncRNAs contributes to the regulation of multiple cancers, including CRC. Here, we observed that the depletion of PSMA3-AS1 restrained the development of CRC cells *via* sponging miR-4429. The present study demonstrated for the first time that PSMA3-AS1 acted as an oncogenic role in CRC progression.

Although various lncRNAs have been reported to play crucial biological functions in multiple malignant tumors, the molecular mechanisms of PSMA3-AS1 in modulating CRC development remain largely unknown. PSMA3-AS1 has been confirmed as an oncogene in various cancers. For example, the overexpression of PSMA3-AS1 accelerated the development of glioma *via* miR-302a-3p/RAB22A axis[23]. PSMA3-AS1 facilitated lung cancer cell invasion and growth *via* regulating miR-4504[24]. LncRNA PSMA3-AS1 accelerated esophageal cancer growth by regulating miR-101/EZH2 axis[25]. In this study, PSMA3-AS1 was decreased in CRC tissues and cells, and the high expression of PSMA3-AS1 was associated with poor prognosis in CRC. Functionally, the knockdown of PSMA3-AS1 significantly inhibited the proliferation, migra-

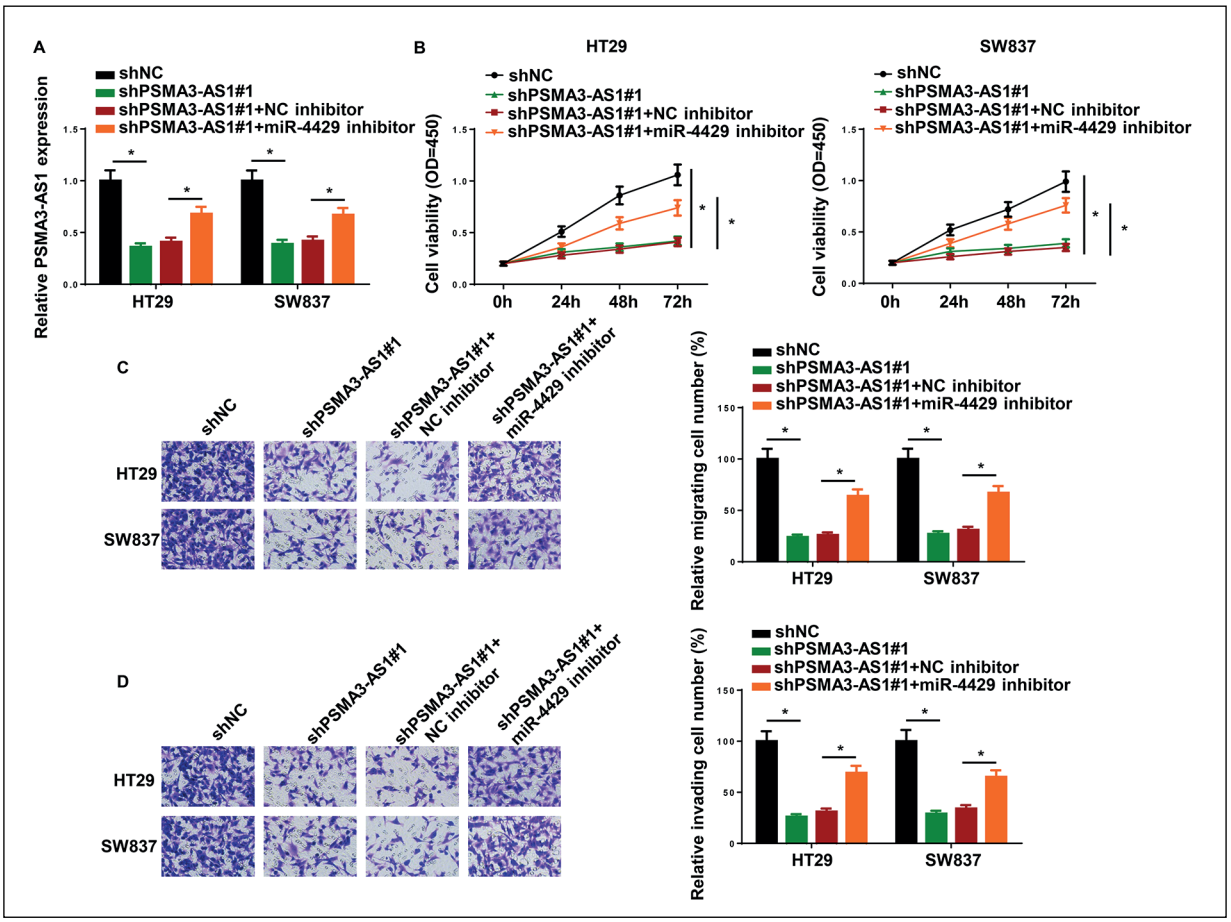

Figure 4. PSMA3-AS1 regulated cell viability, migration and invasion in CRC cells by sponging miR-4429. **A,** The expression of PSMA3-AS1 in HT29 and SW837 cells transfected with shNC, shPSMA3-AS1#1, shPSMA3-AS1#1+NC inhibitor or shPSMA3-AS1#1+miR-4429 inhibitor was detected by RT-qPCR. **B,** HT29 and SW837 cells were transfected with shNC, shPSMA3-AS1#1, shPSMA3-AS1#1+NC inhibitor or shPSMA3-AS1#1+miR-4429 inhibitor, followed by evaluation of cell viability. **C,** and **D,** HT29 and SW837 cells were transfected with shNC, shPSMA3-AS1#1, shPSMA3-AS1#1+NC inhibitor or shPSMA3-AS1#1+miR-4429 inhibitor, followed by detection of cell migration and invasion (magnification ×40). The data were presented as mean ± SD (*$p < 0.05$).

tion, and invasion of CRC cells. Taken together, these results indicated that PSMA3-AS1 promoted the development and progression of CRC.

Anastasiadou et al[26] have indicated that lncRNAs can act as competing endogenous RNAs (ceRNAs) to modulate miRNAs levels in human cancers. Of note, SNHG8 facilitated gastric cancer development by regulating miR-491[27]. TP73-AS1 targeted miR-329-3p to accelerate cell viability and migration in cervical cancer[28]. MYOSLID exerted ceRNA roles in gastric cancer *via* regulating miR-29c-3p[29]. In this study, we predicted the possible PSMA3-AS1-related ceRNA pathway through StarBase and identified that PSMA3-AS1 acted as a sponge for miR-4429 *via* Luciferase reporter and RIP assays. MiR-4429 has been reported to act as a tumor suppressor in cervical cancer[30], gastric cancer[31], and clear cell renal cell carcinoma[32]. In addition, previous studies indicated that several lncRNA, such as LINC00313[33], SNHG12[34], and NR2F2-AS1[35] regulated the development and progression of human cancers by interacting with miR-4429. In this study, it was found that miR-4429 expression was downregulated in CRC tissues and cells, and the depletion of PSMA3-AS1 increased miR-4429 expression through direct interaction. Moreover, the knockdown of PSMA3-AS1 inhibited the progression of CRC, which was counteracted following miR-4429 inhibitor transfection.

Conclusions

We revealed that the depletion of PSMA3-AS1 suppressed cell viability, migration, and invasion in CRC by regulating miR-4429. This study suggested that PSMA3-AS1 might be a potential therapeutic target for CRC intervention.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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References

1) Siegel RL, Miller KD, Jemal A. Cancer statistics, 2019. CA Cancer J Clin 2019; 69: 7-34.
2) Li M, Wang Q, Xue F, Wu Y. LncRNA-CYTOR works as an oncogene through the CYTOR/miR-3679-5p/MACC1 axis in colorectal cancer. DNA Cell Biol 2019; 38: 572-582.
3) Walther A, Johnstone E, Swanton C, Midgley R, Tomlinson I, Kerr D. Genetic prognostic and predictive markers in colorectal cancer. Nat Rev Cancer 2009; 9: 489-99.
4) Rousseau B, Chibaudel B, Bachet JB, Larsen AK, Tournigand C, Louvet C, Andre T, de Gramont A, Gercor. Stage II and stage III colon cancer: treatment advances and future directions. Cancer J 2010; 16: 202-9.
5) Cui M, Chen M, Shen Z, Wang R, Fang X, Song B. LncRNA-UCA1 modulates progression of colon cancer through regulating the miR-28-5p/HOXB3 axis. J Cell Biochem 2019. doi: 10.1002/jcb.27630. Online ahead of print.
6) Tsai MC, Spitale RC, Chang HY. Long intergenic noncoding RNAs: new links in cancer progression. Cancer Res 2011; 71: 3-7.
7) Sana J, Faltejskova P, Svoboda M, Slaby O. Novel classes of non-coding RNAs and cancer. J Transl Med 2012; 10: 103.
8) Khandelwal A, Bacolla A, Vasquez KM, Jain A. Long non-coding RNA: a new paradigm for lung cancer. Mol Carcinog 2015; 54: 1235-51.
9) Tang Y, He Y, Zhang P, Wang J, Fan C, Yang L, Xiong F, Zhang S, Gong Z, Nie S, Liao Q, Li X, Li X, Li Y, Li G, Zeng Z, Xiong W, Guo C. LncRNAs regulate the cytoskeleton and related Rho/ROCK signaling in cancer metastasis. Mol Cancer 2018; 17: 77.
10) Xue X, Yang YA, Zhang A, Fong KW, Kim J, Song B, Li S, Zhao JC, Yu J. LncRNA HOTAIR enhances ER signaling and confers tamoxifen resistance in breast cancer. Oncogene 2016; 35: 2746-55.
11) Chen G, Wang Z, Wang D, Qiu C, Liu M, Chen X, Zhang Q, Yan G, Cui Q. LncRNADisease: a database for long-non-coding RNA-associated diseases. Nucleic Acids Res 2013; 41: D983-6.
12) Ding D, Li C, Zhao T, Li D, Yang L, Zhang B. LncRNA H19/miR-29b-3p/PGRN axis promoted epithelial-mesenchymal transition of colorectal cancer cells by acting on Wnt signaling. Mol Cells 2018; 41: 423-435.
13) Zhang M, Jiang X, Jiang S, Guo Z, Zhou Q, He J. LncRNA FOXD2-AS1 regulates miR-25-3p/Sema4c axis to promote the invasion and migration of colorectal cancer cells. Cancer Manag Res 2019; 11: 10633-10639.
14) Luan Y, Li X, Luan Y, Zhao R, Li Y, Liu L, Hao Y, Oleg Vladimir B, Jia L. Circulating lncRNA UCA1 promotes malignancy of colorectal cancer via the miR-143/MYO6 axis. Mol Ther Nucleic Acids 2020; 19: 790-803.
15) Wang Z, Jin J. LncRNA SLCO4A1-AS1 promotes colorectal cancer cell proliferation by enhancing autophagy via miR-508-3p/PARD3 axis. Aging (Albany NY) 2019; 11: 4876-4889.
16) Ghasabi M, Mansoori B, Mohammadi A, Duijf PH, Shomali N, Shirafkan N, Mokhtarzadeh A, Baradaran B. MicroRNAs in cancer drug resistance: basic evidence and clinical applications. J Cell Physiol 2019; 234: 2152-2168.
17) Kim SW. [The Role of microRNAs in colorectal cancer]. Korean J Gastroenterol 2017; 69: 206-211.
18) Afgar A, Fard-Esfahani P, Mehrtash A, Azadmanesh K, Khodarahmi F, Ghadir M, Teimoori-Toolabi L. MiR-339 and especially miR-766 reactivate the expression of tumor suppressor genes in colorectal cancer cell lines through DNA methyltransferase 3B gene inhibition. Cancer Biol Ther 2016; 17: 1126-1138.
19) Zhang Z, Li J, Huang Y, Peng W, Qian W, Gu J, Wang Q, Hu T, Ji D, Ji B, Zhang Y, Wang S, Sun Y. Upregulated miR-1258 regulates cell cycle and inhibits cell proliferation by directly targeting E2F8 in CRC. Cell Prolif 2018; 51: e12505.
20) Huang L, Zhang Y, Li Z, Zhao X, Xi Z, Chen H, Shi H, Xin T, Shen R, Wang T. MiR-4319 suppresses colorectal cancer progression by targeting ABTB1. United European Gastroenterol J 2019; 7: 517-528.
21) Liang Z, Li X, Liu S, Li C, Wang X, Xing J. MiR-141-3p inhibits cell proliferation, migration and invasion by targeting TRAF5 in colorectal cancer. Biochem Biophys Res Commun 2019; 514: 699-705.
22) Zhang D, Yang N. MiR-335-5p inhibits cell proliferation, migration and invasion in colorectal cancer through downregulating LDHB. J BUON 2019; 24: 1128-1136.
23) Zhou LL, Zhang M, Zhang YZ, Sun MF. Long non-coding RNA PSMA3-AS1 enhances cell proliferation, migration and invasion by regulating miR-302a-3p/RAB22A in glioma. Biosci Rep 2020; 40: BSR20191571.

24) Li F, Yu L, Zhu J. LncRNA PSMA3-AS1 promotes lung cancer growth and invasion via sponging miR-4504. Cancer Manag Res 2020; 12: 5277-5283.

25) Qiu BQ, Lin XH, Ye XD, Huang W, Pei X, Xiong D, Long X, Zhu SQ, Lu F, Lin K, Zhang XQ, Xu JJ, Sheng LL, Zhang XM, Zhang PF, Wu YB. Long non-coding RNA PSMA3-AS1 promotes malignant phenotypes of esophageal cancer by modulating the miR-101/EZH2 axis as a ceRNA. Aging (Albany NY) 2020; 12: 1843-1856.

26) Anastasiadou E, Jacob LS, Slack FJ. Non-coding RNA networks in cancer. Nat Rev Cancer 2018; 18: 5-18.

27) Zhang P, Li S, Chen Z, Lu Y, Zhang H. LncRNA SNHG8 promotes proliferation and invasion of gastric cancer cells by targeting the miR-491/PDGFRA axis. Hum Cell 2020; 33: 123-130.

28) Xu J, Zhang J. LncRNA TP73-AS1 is a novel regulator in cervical cancer via miR-329-3p/ARF1 axis. J Cell Biochem 2020; 121: 344-352.

29) Han Y, Wu N, Jiang M, Chu Y, Wang Z, Liu H, Cao J, Liu H, Xu B, Xie X. Long non-coding RNA MYOSLID functions as a competing endogenous RNA to regulate MCL-1 expression by sponging miR-29c-3p in gastric cancer. Cell Prolif 2019; 52: e12678.

30) Liang L, Zheng YW, Wang YL. MiR-4429 regulates the proliferation, migration, invasion, and epithelial-mesenchymal transition of cervical cancer by targeting FOXM1. Cancer Manag Res 2020; 12: 5301-5312.

31) He H, Wu W, Sun Z, Chai L. MiR-4429 prevented gastric cancer progression through targeting METTL3 to inhibit m(6)A-caused stabilization of SEC62. Biochem Biophys Res Commun 2019; 517: 581-587.

32) Pan H, Hong Y, Yu B, Li L, Zhang X. MiR-4429 inhibits tumor progression and epithelial-mesenchymal transition via targeting CDK6 in clear cell renal cell carcinoma. Cancer Biother Radiopharm 2019; 34: 334-341.

33) Wu WJ, Yin H, Hu JJ, Wei XZ. Long noncoding RNA LINC00313 modulates papillary thyroid cancer tumorigenesis via sponging miR-4429. Neoplasma 2018; 65: 933-942.

34) Cai P, Wu M, Zhang B, Wu S, Wei H, Wei L. Long noncoding RNA SNHG12 regulates cell proliferation, invasion and migration in endometrial cancer by targeting miR4429. Mol Med Rep 2020.

35) Liu D, Huang K, Wang T, Zhang X, Liu W, Yue X, Wu J. NR2F2-AS1 accelerates cell proliferation through regulating miR-4429/MBD1 axis in cervical cancer. Biosci Rep 2020; 40: BSR20194282.