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ORIGINAL RESEARCH

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Inhibition of *PRPF19* impairs oncogenesis, radioresistance and DNA damage repair in cervical cancer

Qianqian Wang[1,*,†], Jinwei Zhang[1,†], Yue Zhang[1,*]

[1]Department of Gynecology, The Affiliated Wuxi People's Hospital of Nanjing Medical University, 214023 Wuxi, Jiangsu, China

***Correspondence**
Wangqianqian_6688@163.com
(Qianqian Wang);
yoyosonic@foxmail.com
(Yue Zhang)

[†] These authors contributed equally.

Abstract

Background: Cervical cancer (CC) is one prevalent and lethal gynecological malignancy. Pre-mRNA-processing factor 19 (*PRPF19*) has been implicated in the progression of multiple cancers and shown to play a role in modulating the DNA damage response. However, the specific regulatory effects of *PRPF19* and its associated pathways in the development of CC remain poorly understood. **Methods**: The protein expressions were inspected through western blot. The survival fraction and the number of colonies were examined through colony formation assay. The fluorescence intensity of gamma-histone H2A family member X (γH2AX) was verified through Immunofluorescence (IF) assay. The cell invasion and migration were tested through Transwell assay. **Results**: In this study, data from the Gene Expression Profiling Interactive Analysis (GEPIA) and User-friendly Analysis Tool for Cancer Gene Expression Data (UALCAN) online databases were analyzed, and the findings revealed significant overexpression of *PRPF19* in cervical squamous cell carcinoma (CESC) tissues. Additionally, we confirmed elevated *PRPF19* expression in CC, with the inhibition of *PRPF19* increasing the sensitivity of CC cells to X-ray treatment. Furthermore, *PRPF19* knockdown enhanced DNA damage following X-ray exposure, as evidenced by increased γH2AX fluorescence intensity and reduced levels of p-DNA-protein kinase (PK) and Rad51 recombinase (Rad51). *PRPF19* suppression also inhibited cell migration and invasion. Mechanistically, *PRPF19* promoted activation of the Sarcoma (Src)-Yes-associated protein 1 (YAP1) pathway by downregulating p-Src/Src and YAP1 levels. **Conclusions**: *PRPF19* inhibition impairs oncogenesis, reduces radioresistance and disrupts DNA damage repair in CC, partly through modulation of the Src-YAP1 pathway, thereby supporting *PRPF19* as one prospective bio-target for CC treatment.

Keywords

PRPF19; Radioresistance; DNA damage; Cervical cancer; Src-YAP1 pathway

1. Introduction

Cervical cancer (CC) is one prevalent gynecological malignancy and results into highly deaths among women worldwide [1]. Despite various treatment options, approximately two-thirds of CC patients at the advanced stage leads to hard treatment and high mortality [2]. Current treatment modalities for CC include surgery, radiotherapy, immunotherapy and chemotherapy [3]. For locally CC patients, concurrent chemoradiotherapy has shown significant survival benefits [4, 5]. However, the radioresistance of tumor cells is a major factor contributing to treatment failure [6]. Thus, understanding the mechanisms underlying radioresistance and seeking novel therapeutic targets is essential for improving survival outcomes in CC patients.

Pre-mRNA-processing factor 19 (*PRPF19*) is a highly conserved splicing factor across species [7]. *PRPF19* is a multifunctional protein involved in both the DNA damage response and pre-mRNA processing [8]. It has been proved that *PRPF19* can promote tumorigenesis and resistance to chemoradiotherapy in tongue cancer [9]. In prostate cancer, *PRPF19* has been uncovered to accelerate cell proliferation and migration by refraining solute carrier family 40 (iron-regulated transporter), member 1 (SLC40A1) while also suppressing autophagy [10]. In bladder cancer, *PRPF19* modulates DNA damage repair and enhances gemcitabine sensitivity by interacting with Damaged DNA Binding Protein 1 (DDB1) [11]. Additionally, *PRPF19* regulates fatty acid metabolism to facilitate the development of esophageal squamous cell carcinoma [12] and promotes liver metastasis in colorectal cancer through the ubiquitination of Myosin Light Chain 9 (MYL9) [13]. However, until now, the role of *PRPF19*

and its associated pathways in CC progression remain largely dimness.

In this study, findings testified that inhibition of *PRPF19* impairs oncogenesis, radioresistance, and DNA damage repair in CC by suppressing the Src-YAP1 pathway. This project may provide novel insights into the potential therapeutic impacts of targeting *PRPF19* for CC.

2. Materials and methods

2.1 Cell lines and culture

The human cervical epithelial immortalized cell line (H8) and CC cell lines (HeLa and CaSki) were obtained from the American Tissue Culture Collection (ATCC, USA). The culturing of cells was made in Dulbecco's Modified Eagle Medium (DMEM, 12800017, Invitrogen, Carlsbad, CA, USA) with 10% fetal bovine serum (FBS, 10099-141, Gibco Laboratories, Grand Island, NY, USA) in one humidified atmosphere at 37 °C with 5% CO_2.

HeLa cells were subjected to X-ray irradiation at doses of 0, 2, 4, 6 and 8 Gy using the RS2000 X-ray Biological Research Irradiator (3 mm copper filter, 160 kV, 25 mA; Rad Source Technologies, Buford, GA, USA).

2.2 Cell transfection

Short hairpin RNAs (shRNAs) targeting *PRPF19* (shPRPF19-1# or 2#) and the corresponding negative control (shNC) were purchased from GenePharma (Shanghai, China). Transfections were performed using Lipofectamine 2000 (11668019, Invitrogen, Carlsbad, CA, USA) following the manufacturer's instructions.

2.3 Western blot

CC cells were lysed using radioimmunoprecipitation assay (RIPA) lysis buffer (P0013B, Beyotime, Shanghai, China) to extract proteins. Proteins were then separated via sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE, 10%) and transferred to polyvinylidene fluoride (PVDF) membranes (Beyotime, Shanghai, China). Post blocking, the membranes were incubated with primary antibodies at 4 °C for 12 hours, followed by incubation with secondary antibodies (1:1000; ab7090) for 2 hours. Protein bands were visualized using a chemiluminescence detection kit (20148, Thermo Fisher Scientific, Inc., Waltham, MA, USA) and quantified using Image-Pro Plus 6.0 software (Media Cybernetics, Rockville, MD, USA).

The primary antibodies used included PRPF19 (1:1000; ab126776; Abcam, Shanghai, China), p-DNA-PK (1:5000; ab124918), Rad51 (1:10,000; ab133534), p-Src (1:5000; ab32078), Src (1:10,000; ab109381), YAP1 (1:5000; ab52771), and β-actin (1 μg/mL; ab8226).

2.4 Colony formation assay

HeLa and CaSki cells (1000 cells/well) were placed into 6-well plates and allowed to attach for 24 hours. After attachment, the cells were irradiated with X-rays at doses of 0, 2, 4, 6 or 8 Gy. After two weeks, the colonies formed were fixed and stained with 0.1% crystal violet, and the number of colonies was counted. The survival fraction (%) was calculated using the following formula: Survival fraction (%) = (Number of valid clones)/(Number of inoculated cells $\times$ 0 Gy colony formation rate) $\times$ 100%.

2.5 Immunofluorescence (IF) assay

HeLa and CaSki cells (1×10^5) were fixed with 4% paraformaldehyde, blocked with 5% bovine serum albumin (BSA), and permeabilized with 0.2% Triton X-100. The cells were then incubated with a primary antibody against H2AX (1:1000; ab124781, Abcam, Shanghai, China) followed by a secondary antibody (1:1000; ab7149). Nuclear staining was performed using 4′,6-diamidino-2-phenylindole (DAPI). Fluorescence images were captured using an Olympus BX53 microscope (Olympus Optical Co. Ltd., Tokyo, Japan).

2.6 Transwell assay

The Matrigel (356234, BD Biosciences, Franklin Lakes, NJ, USA) was pre-coated onto the upper chambers (pore size, 8 μm; Corning, NY, USA). HeLa or CaSki cells in serum-free medium (200 μL) were placed into the upper chambers, and DMEM containing 20% FBS (600 μL) was added to the lower chambers. After 48 hours, the invaded cells were fixed with 4% paraformaldehyde and dyed with 0.1% crystal violet. For the migration assay, the same steps were followed without Matrigel coating. The transferred cells were visualized and counted under a microscope (CX41, Olympus Optical Co., Ltd., Tokyo, Japan).

2.7 Statistical analysis

All data are presented as the mean $\pm$ standard deviation (SD). Statistical analysis was performed using GraphPad Prism Software version 9 (GraphPad Software, San Diego, CA, USA). Group comparisons were conducted using Student's *t*-test or one-way analysis of variance (ANOVA). A *p*-value of less than 0.05 was set statistically significant.

3. Results

3.1 Overexpression of *PRPF19* in CC

Analysis of the GEPIA and UALCAN online databases uncovered that *PRPF19* expression was markedly elevated in CESC tissues (Fig. 1A,B). Additionally, *PRPF19* protein expression was demonstrated to be upregulated in CC cell lines (Fig. 1C). Overall, *PRPF19* was shown to be overexpressed in CC.

3.2 Inhibition of *PRPF19* increased sensitivity to X-ray treatment

The knockdown efficiency of *PRPF19* in HeLa and CaSki cells was confirmed (Fig. 2A), showing significantly reduced *PRPF19* protein levels after *PRPF19* inhibition. Next, the survival fraction of these cells was found to be significantly decreased after *PRPF19* suppression (Fig. 2B). Further experiments showed that the number of colonies formed after X-ray irradiation (6 Gy) was reduced, and this effect was further enhanced by *PRPF19* silencing (Fig. 2C). Collectively,

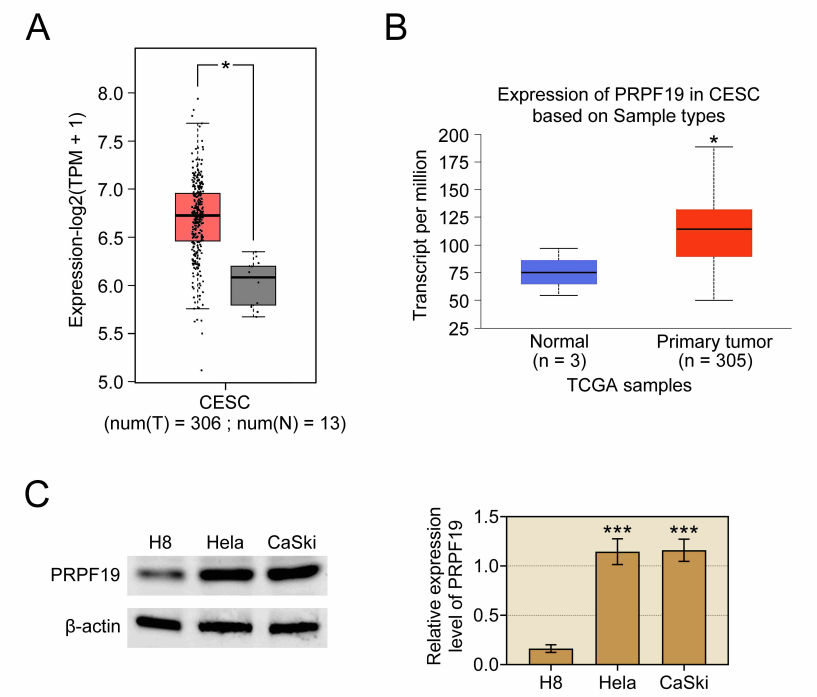

FIGURE 1. *PRPF19* is overexpressed in cervical cancer. (A) *PRPF19* expression was analyzed in normal tissues and cervical squamous cell carcinoma (CESC) tissues using the GEPIA online database. (B) *PRPF19* expression was further validated in normal and CESC tissues using the UALCAN online database. (C) PRPF19 protein levels were assessed in the human cervical epithelial immortalized cell line (H8) and cervical cancer cell lines (HeLa and CaSki) by western blot analysis. *$p < 0.05$, ***$p < 0.001$. *PRPF19*: Pre-mRNA-processing factor 19; TPM: Transcripts Per Million; TCGA: The Cancer Genome Atlas.

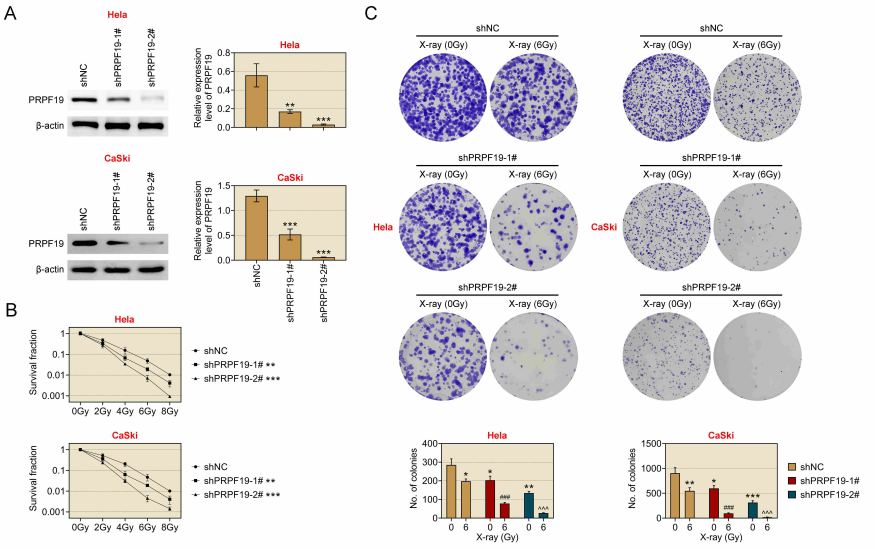

FIGURE 2. Inhibition of *PRPF19* increases sensitivity to X-ray treatment. (A) PRPF19 protein expression was measured in HeLa and CaSki cells via western blot, which were categorized into the following groups: shNC, shPRPF19-1# and shPRPF19-2#. **$p < 0.01$, ***$p < 0.001$. (B) Cell survival fractions were evaluated in HeLa and CaSki cells using colony formation assay after exposure to 0, 2, 4, 6 and 8 Gy of X-ray irradiation. The groups included shNC, shPRPF19-1#, and shPRPF19-2#. **$p < 0.01$, ***$p < 0.001$. (C) Colony numbers were quantified in HeLa and CaSki cells after treatment with 0 or 6 Gy X-ray irradiation. The groups included shNC + X-ray (0 Gy), shNC + X-ray (6 Gy), shPRPF19-1# + X-ray (0 Gy), shPRPF19-1# + X-ray (6 Gy), shPRPF19-2# + X-ray (0 Gy), and shPRPF19-2# + X-ray (6 Gy). *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, *vs.* the shNC + X-ray (0 Gy) group; ###$p < 0.001$ *vs.* the shPRPF19-1# + X-ray (0 Gy) group; ^^^$p < 0.001$ *vs.* the shPRPF19-2# + X-ray (0 Gy) group. PRPF19: Pre-mRNA-processing factor 19; shNC: Short hairpin negative control.

these data manifest that suppression of *PRPF19* increases the sensitivity of CC cells to X-ray treatment.

3.3 Knockdown of *PRPF19* accelerated DNA damage after X-ray treatment

After X-ray irradiation (6 Gy), γH2AX fluorescence intensity, an indicator of DNA damage, was found to be significantly increased following *PRPF19* inhibition (Fig. 3A). Furthermore, the protein expression levels of p-DNA-PK and Rad51, both of which are involved in DNA repair, were downregulated following *PRPF19* suppression (Fig. 3B). Overall, these results suggest that the knockdown of *PRPF19* enhances DNA damage in response to X-ray treatment.

3.4 Suppression of PRPF19 suppressed cell migration and invasion

PRPF19 knockdown resulted into the reduction in cell migration (Fig. 4A), and a similar effect was observed for cell invasion (Fig. 4B). Taken together, these results demonstrate that suppression of *PRPF19* retards both migration and invasion of CC cells.

3.5 *PRPF19* activated the Src-YAP1 pathway

In HeLa and CaSki cells, *PRPF19* inhibition resulted in decreased protein levels of p-Src/Src and YAP1 (Fig. 5), indicating that *PRPF19* activates the Src-YAP1 signaling pathway.

4. Discussion

PRPF19 has been identified as a pivotal player in the development of diversiform cancers, functioning as a facilitator

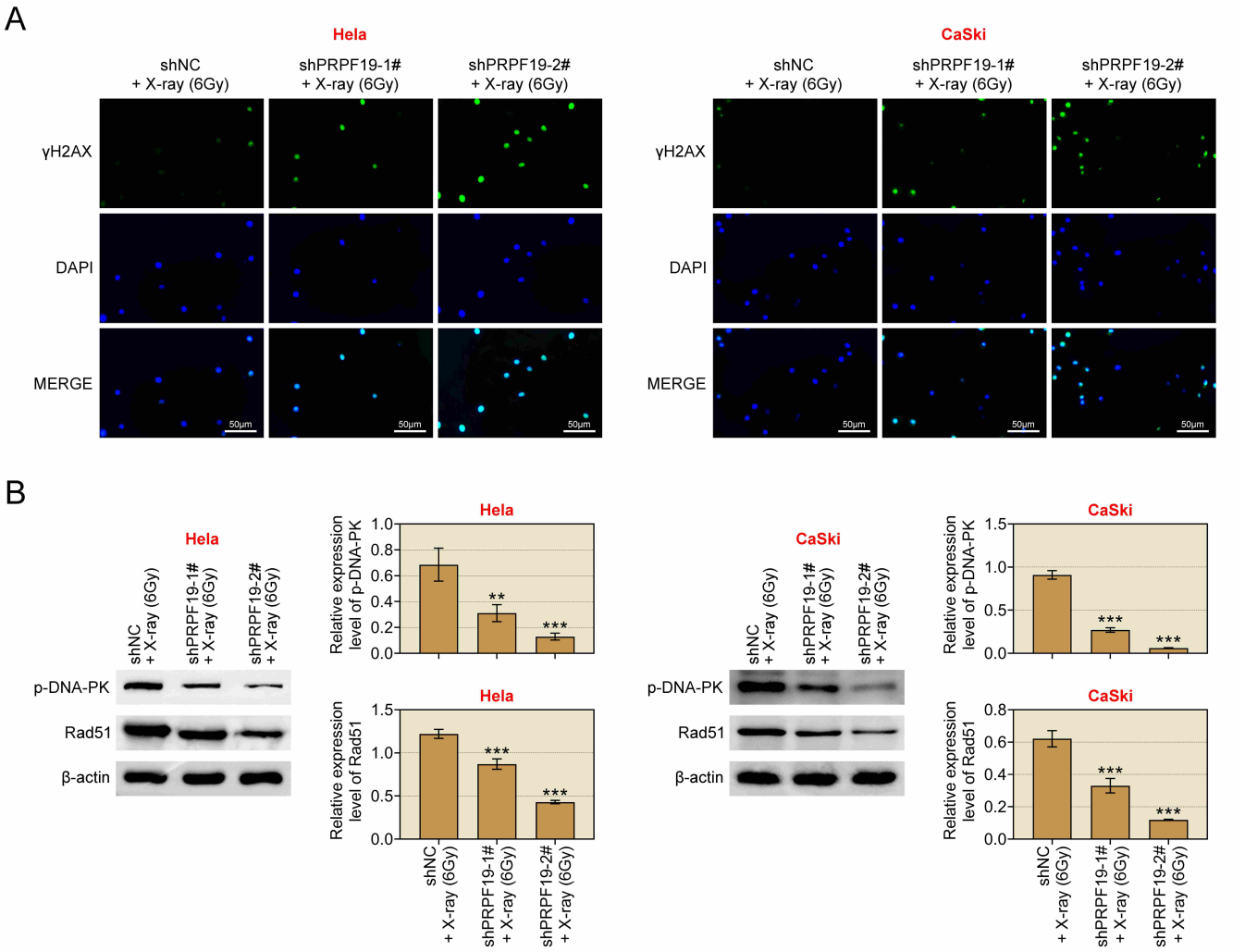

FIGURE 3. Knockdown of *PRPF19* enhances DNA damage after X-ray treatment. The groups assessed included shNC + X-ray (6 Gy), shPRPF19-1# + X-ray (6 Gy), and shPRPF19-2# + X-ray (6 Gy). (A) γH2AX fluorescence intensity was evaluated in HeLa and CaSki cells using immunofluorescence assay. (B) The protein expression levels of p-DNA-PK and Rad51 were measured in HeLa and CaSki cells via western blot. $^{**}p < 0.01$, $^{***}p < 0.001$. *PRPF19*: Pre-mRNA-processing factor 19; shNC: Short hairpin negative control; γH2AX: gamma-histone H2A family member X; DAPI: 4′,6-diamidino-2-phenylindole; Rad51: Rad51 recombinase; p-DNA-PK: p-DNA-protein kinase.

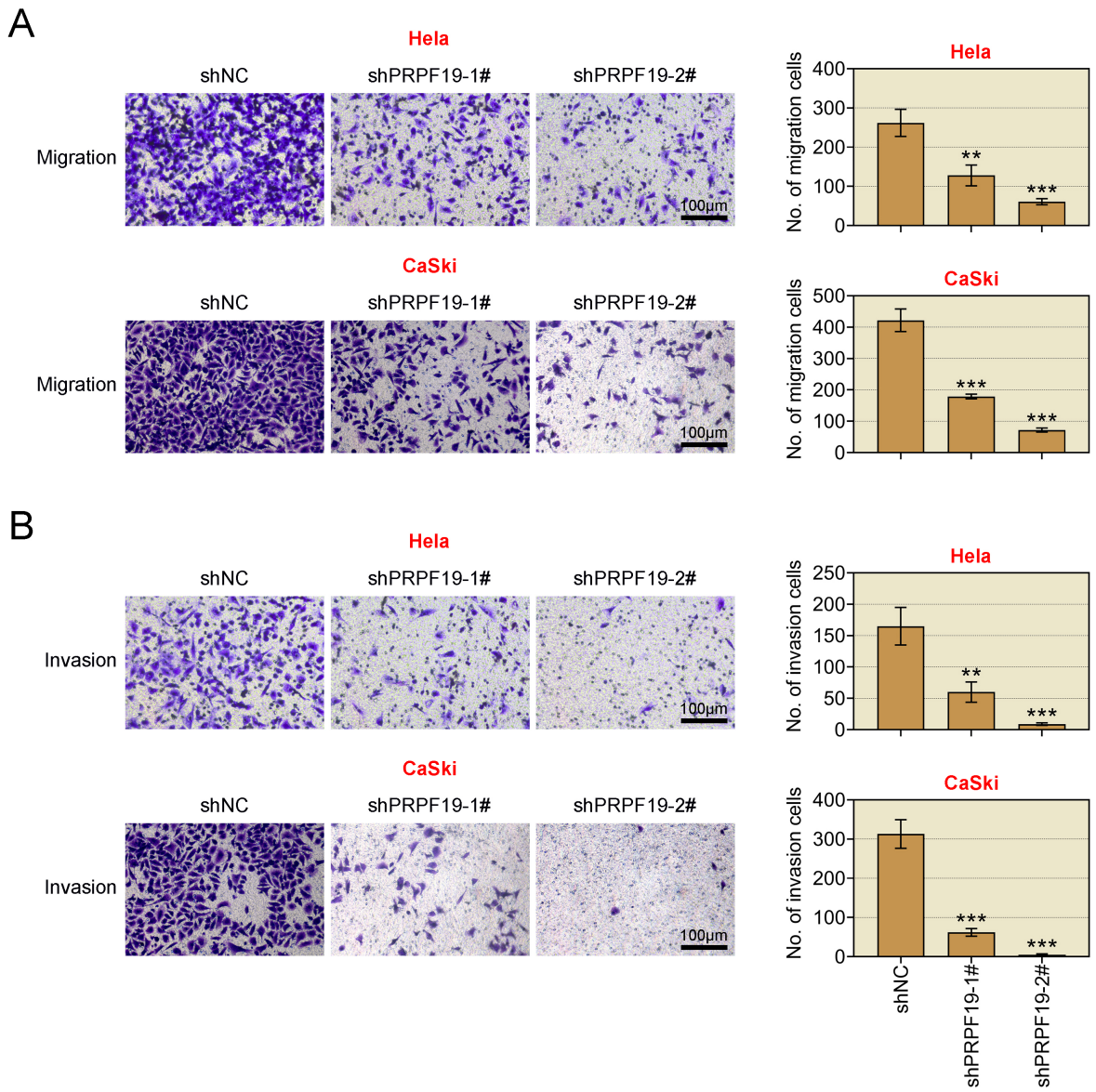

FIGURE 4. PRPF19 suppression inhibits cell migration and invasion. The groups assessed included shNC, shPRPF19-1#, and shPRPF19-2#. (A) Cell migration was assessed in HeLa and CaSki cells using a Transwell assay. (B) Cell invasion was evaluated in HeLa and CaSki cells using a Transwell assay. $^{**}p < 0.01$, $^{***}p < 0.001$. *PRPF19*: Pre-mRNA-processing factor 19; shNC: Short hairpin negative control.

of tumorigenesis and therapy resistance [9–13]. However, its regulatory role and associated pathways in CC remain unclear. In this study, assessed the GEPIA and UALCAN online databases and observed that *PRPF19* is overexpressed in CESC tissues, which was then confirmed in CC cell lines.

X-ray treatment is widely used in the management of CC and has received increasing attention due to its therapeutic potential. Several studies have highlighted the molecular mechanisms underlying radioresistance in CC. For instance, Heat Shock Proteins 90 (HSP90) affects Cluster of Differentiation (CD)147 polyubiquitination to enhance radioresistance [14], Ras-associated binding 12 (Rab12) promotes late phase of DNA synthesis (G2)/mitotic phase (M) arrest to support radioresistance [15], and Sp1 enhances Cyclin Dependent Kinase 1 (CDK1) expression to increase radioresistance in CC [16]. Data showed that inhibition of *PRPF19* increased the sensitivity of CC cells to X-ray treatment.

The mechanisms of radioresistance are complex and multifactorial, often involving enhanced DNA repair capabilities, cell survival and alterations in the tumor microenvironment [17, 18]. Radiation therapy can evoke DNA double-strand breaks, and efficient DNA repair can diminish radiosensitivity [19]. Several studies have investigated the relationship between DNA damage regulation and radioresistance in CC. For instance, Aldolase A was found to modulate glycolysis and DNA repair to enhance radioresistance [20], bromodomain-containing protein 4 (BRD4) knockdown impairs DNA repair, sensitizing CC cells to radiotherapy [21], and proteasome Activator Subunit 3 (PSME3) modulates poly (ADP-ribose) polymerase 1 (PARP1) activity to promote radioresistance

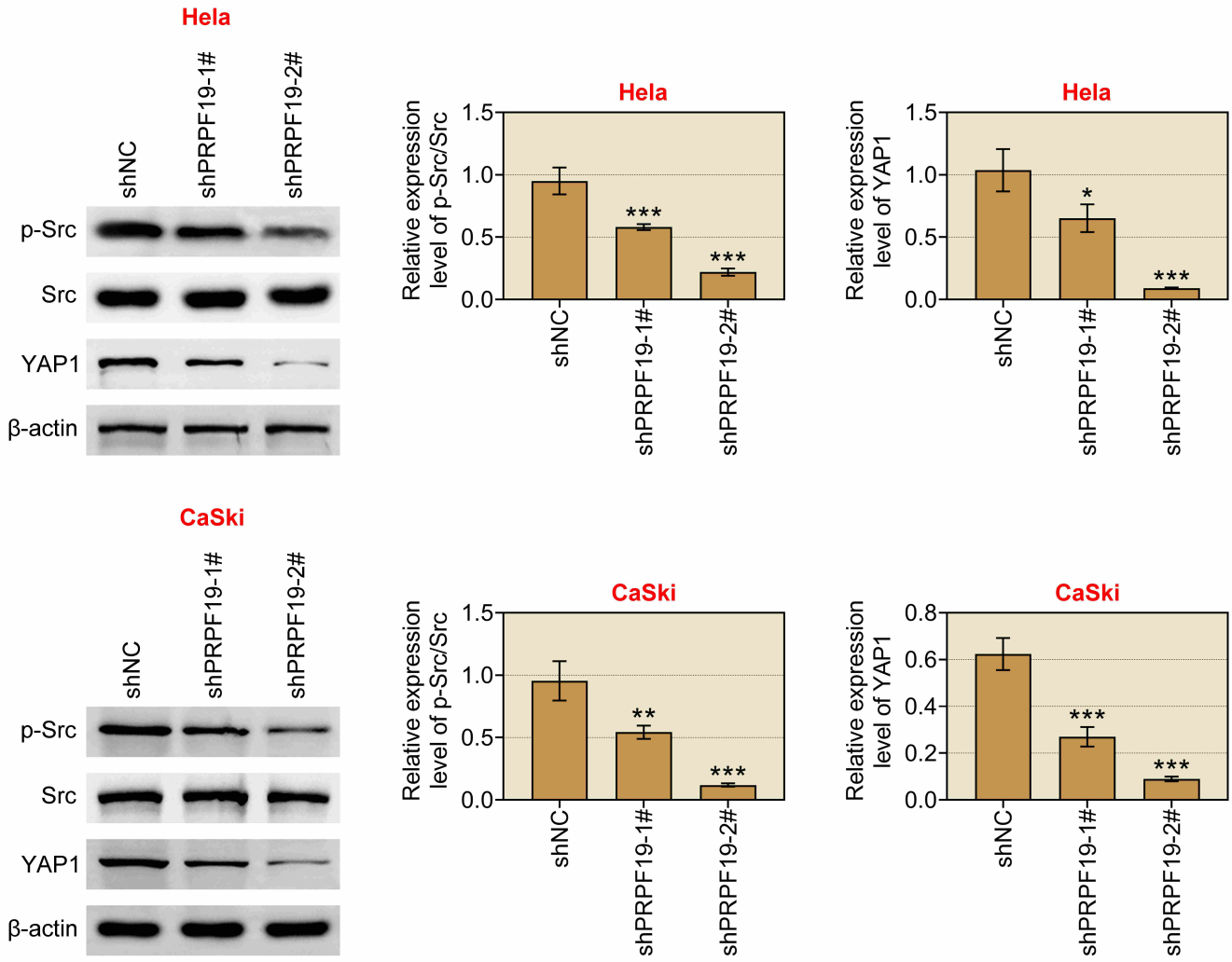

FIGURE 5. *PRPF19* activates the Src-YAP1 pathway. HeLa and CaSki cells were divided into the shNC, shPRPF19-1#, and shPRPF19-2# groups and the protein expression levels of p-Src, Src and YAP1 were detected via western blot. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$. *PRPF19*: Pre-mRNA-processing factor 19; shNC: Short hairpin negative control; Src: Sarcoma; YAP1: Yes-associated protein 1.

and aerobic glycolysis [22]. Additionally, sterile alpha motif domain-containing protein 1 (SND1) affects the DNA damage response, contributing to increased radioresistance in CC [23]. Similarly, our findings showed that *PRPF19* knockdown enhances DNA damage following X-ray treatment by increasing γH2AX fluorescence intensity and reducing p-DNA-PK and Rad51 levels. Furthermore, we found that *PRPF19* inhibition suppressed both cell migration and invasion, further underscoring its role in CC progression.

The Src-YAP1 pathway has been testified to join in the development of diversiform cancers. For instance, the integrin-Src-YAP1 pathway confers resistance to targeted therapies in melanoma [24], αE-catenin suppresses the Src-YAP1 pathway in skin squamous cell carcinoma [25], and Disabled homolog 2 (DAB2) promotes gastric tumorigenesis via activation of the Src-YAP1 pathway [26]. Importantly, previous research has shown that *PRPF19* stimulates the Src-YAP1 pathway in colorectal cancer [13]. However, the role of *PRPF19* in modulating the Src-YAP1 pathway in CC keep vague. In this project, we clarified that *PRPF19* triggers the Src-YAP1 pathway by downregulating p-Src/Src and YAP1 levels in CC cells.

5. Conclusions

This study is the first to demonstrate that inhibition of *PRPF19* impairs oncogenesis, radioresistance and DNA damage repair in CC while also inhibiting the Src-YAP1 pathway. However, there were several limitations that should be considered, such as the lack of clinical and animal model investigations, as well as assessments of other cell phenotypes and molecular mechanisms. Future studies will aim to address these limitations by conducting further experiments to investigate the impacts of *PRPF19* in CC development.

AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

AUTHOR CONTRIBUTIONS

QQW, JWZ—designed the study and carried them out; prepared the manuscript for publication and reviewed the draft of the manuscript. QQW, JWZ, YZ—supervised the data collection; analyzed the data; interpreted the data. All authors have read and approved the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This article does not contain any studies with human participants or animals performed by any of the authors.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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