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ORIGINAL RESEARCH

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ZNF263 promotes gastric cancer cell growth by regulating TXNDC9 transcription

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Abstract

Background: Zinc finger protein 263 (ZNF263) has been linked to the progression of several cancer types, although its significance in gastric cancer (GC) is poorly understood. This work sought to elucidate ZNF263's biological function and underlying mechanism in GC formation. **Methods**: The expression of ZNF263 in GC tissues and its relationship with patient survival were investigated using public databases. ZNF263 protein expression was analyzed after transfecting GC cell lines with both the ZNF263 knockdown and overexpression plasmids. Cell viability and proliferation were assessed using Cell Counting Kit-8 (CCK8) and 5-Ethynyl-2′-deoxyuridine (EdU) assays, respectively. Flow cytometry was used to assess apoptosis rates, as well as the expression of associated proteins (cleaved-caspase3 and cleaved-Poly(ADP-ribose) polymerase) was analyzed by Western blot. Chromatin immunoprecipitation followed by quantitative Polymerase Chain Reaction (ChIP-qPCR) and Dual luciferase assay were used to validate the transcriptional regulation of Thioredoxin Domain Containing 9 (TXNDC9) by ZNF263. TXNDC9 mRNA levels were measured by quantitative Reverse Transcription (qRT)-PCR. **Results**: ZNF263 was highly elevated in GC tissues, and high expression was related with a poor patient prognosis. ZNF263 overexpression promoted the malignant development of GC cells, while ZNF263 knockdown suppressed cell growth and induced apoptosis. These effects were accompanied by increased levels of cleaved-caspase3 and cleaved-PARP. Mechanistically, ZNF263 directly bound to the TXNDC9 promoter region, enhanced TXNDC9 transcription, and increased TXNDC9 mRNA expression. ZNF263 also elevated luciferase activity driven by the wild type, but not mutant, TXNDC9 promoter. **Conclusions**: ZNF263 promotes GC cell growth and inhibits apoptosis, which may be related to ZNF263 promoting TXNDC9 transcription. ZNF263–TXNDC9 axis may serve as a potential therapeutic target in GC.

Keywords

Apoptosis; Gastric cancer; Proliferation; TXNDC9; ZNF263

1. Introduction

Gastric cancer (GC) is a common malignancy of the digestive tract, originating in the stomach. It is a highly aggressive disease with one of the highest incidence and fatality rates globally [1, 2]. The incidence of GC is influenced by various factors, including genetics, environmental exposure, and dietary habits. Early detection and good treatment are critical for increasing the survival rates and treatment results [3, 4]. However, GC frequently presents with few or no symptoms in its early stages, many individuals are recognized only after the disease has progressed to an advanced level [5]. Therefore, identifying suitable molecular targets for early diagnosis and targeted therapy is of great significance in the fight against GC.

Zinc finger protein (ZNF) 263, a member of the zinc finger family of transcriptional factors [6], has been implicated in the regulation of gene expression and the development of various cancers. Previous studies have demonstrated that ZNF263 is overexpressed in hepatocellular carcinoma, where its knockdown reduces tumor cell proliferation resistance [7]. Moreover, ZNF263 promotes the growth, migration, invasion, and glycolysis of renal cell carcinoma by regulating downstream signalling pathways [8].

Thioredoxin domain-containing protein 9 (TXNDC9), also called phagocytic-like protein 3 (PHLP3), is a TRX family member that regulates Adenosine Triphosphate (ATP)ase function [9]. TXNDC9 knockdown has been shown to inhibit cell invasion, microtubule formation, and epithelial-mesenchymal transition (EMT) in GC cells *in vitro* [10]. Additionally, TXNDC9 overexpression has been associated with increased viability, invasion, and migration of GC cells, supporting its oncogenic role in GC [11].

Despite these findings, the role of ZNF263 in GC is unknown. In this study, we demonstrate for the first time that

ZNF263 stimulates gastric cancer cell proliferation while inhibiting apoptosis, suggesting its involvement in the progression of GC. Mechanistically, ZNF263 stimulates the transcription of TXNDC9. These results highlight ZNF263 as a possible target for the development of gene-targeted therapies in GC.

2. Materials and methods

2.1 Bioinformatics analysis

Survival data for GC patients were obtained from the Kaplan Meier Plotter database, provided the survival curve, ZNF263 expression levels in GC tissues were analyzed using the University of Alabama at Birmingham Cancer Data Analysis Portal (UALCAN) database.

2.2 Cell culture and transfection

Human gastric cancer cell lines MNK-45 and AGS were obtained from the Chinese Academy of Sciences Type Culture Collection. Cell lines were grown in Dulbecco's Modified Eagle Medium (DMEM) with 10% fetal bovine serum (FBS), 100 U/mL penicillin, and 100 mg/mL streptomycin in a 95% humidified environment at 37 °C and 5% CO_2.

For gene knockdown and overexpression studies, si-ZNF263 (GTGCAACATTTGCGGAAAA), negative control siRNA (si-NC), ZNF263, and TXNDC9 overexpression plasmids were transfected using Lipofectamine 2000.

2.3 Cell viability

Cells were planted in 96-well plates with 1×10^3 cells per well. After transfection, add 10 μL of CCK8 reagent to each well and incubate for 2 hours at 37 °C. Every 24 hours, the optical density (OD) was measured at 450 nm with an enzyme microplate reader.

2.4 Cell proliferation

Cells in logarithmic growth phase were seeded into 96-well plates and treated with EdU medium. The medium was discarded, and cells were rinsed with Phosphate Buffered Saline (PBS). The cells were then treated with 4% paraformaldehyde before being removed. Glycine was added, and the cells were shaken for 5 minutes to decolorize them. The glycine solution was removed, and the cells were washed again with PBS. Permeabilization was performed using TritonX-100 in PBS, and cells were incubated for 10 minutes. The Apollo staining reaction solution was added, and the cells were incubated in the dark at room temperature for 30 minutes. Hoechst 33342 was used to stain the nuclei, and the cells were incubated in the dark at room temperature for 10 minutes. The cells were studied, photographed, and quantified using a fluorescence microscope.

2.5 Apoptosis

GC cells were planted in 6-well plates and incubated for 48 hours. Cells were collected with trypsin without Ethylenediaminetetraacetic acid (EDTA), washed with PBS, and stained with Annexin V-Fluorescein isothiocyanate (FITC) and Propidium Iodide (PI) in the dark for 15 minutes at room temperature using the Annexin V-FITC/PI Apoptosis Detection Kit. Flow cytometry was used to assess cellular apoptosis.

2.6 Western blotting

The proteins were isolated from GC cells using Radioimmunoprecipitation Assay (RIPA) lysis buffer. The protein concentration was measured using the Bicinchoninic Acid Assay (BCA) Protein Assay Kit to establish the electrophoresis loading volume. Protein samples were combined with loading buffer before being denatured in a water bath at 95 °C. Proteins were separated using Sodium Dodecyl Sulfate-Polyacrylamide Gel Electrophoresis (SDS-PAGE), then transferred to a Polyvinylidene Fluoride (PVDF) membrane and blocked with 5% milk in Tris Buffered Saline with Tween-20 (TBST). After washing with TBST, primary antibodies were applied to the membrane, which was then incubated overnight at 4 °C. Secondary antibodies were added after washing with TBST and incubated for 2 hours at room temperature. Membranes were imaged with a gel imaging device, and densitometry was done with ImageJ software. Antibodies used: cleaved-caspase 3 (Abcam, Cambridge, UK, ab2302, 1:500); cleaved-PARP (Abcam, Cambridge, UK, ab32064, 1:1000); ZNF263 (Abcam, Cambridge, UK, ab129100, 1:1000).

2.7 Quantitative real-time PCR (qRT-PCR)

Total RNA was isolated from GC cells using TRIzol reagent, and its purity and concentration were determined using a Nanodrop 1000 spectrophotometer. cDNA was produced using a reverse transcription kit. The qRT-PCR was carried out using an Applied Biosystems 7300 Real-Time PCR System (Foster City, CA, USA). *TXNDC9* gene expression was standardized to Glyceraldehyde-3-phosphate dehydrogenase (*GAPDH*). Relative expression levels were obtained using the $2^{-\Delta\Delta Ct}$ technique. *TXNDC9* forward primer: 5′-CTGCTTCAGACTACCAAACTGG-3′, *TXNDC9* reverse primer 5′-CTCTGTAGAAATGGCAAACCACA-3′.

2.8 Chromatin immunoprecipitation (ChIP)

The Simple ChIP Enzymatic Chromatin IP kit was used as directed by the manufacturer to accomplish chromatin immunoprecipitation. HEK293T cells were cross-linked with 1% formaldehyde for 10 minutes, then quenched with glycine for 5 minutes at room temperature before being lysed. Lysates were treated with anti-ZNF263 antibody or normal rabbit Immunoglobulin G (IgG). DNA was purified after reverse DNA cross-linking using Sodium Chloride (NaCl) and protease. DNA samples were analyzed by qRT-PCR to assess TXNDC9 promoter binding.

2.9 Dual-luciferase reporter assay

TXNDC9 and ZNF263 binding sites (wild-type or mutant) were cloned into the pGL3 reporter vector. Using Lipofectamine® 3000, these reporter plasmids were co-transfected with ZNF263 and NC into HEK293T cells in 24-well plates. Luciferase activity was determined using the Dual Luciferase Reporter Gene Assay Kit.

2.10 Statistical analysis

Data from three independent experiments are presented as mean ± Standard Deviation (SD). The statistical analysis was carried out using SPSS 22.0 (IBM Corp, Armonk, NY, USA). An unpaired Student's *t*-test was used to compare the two groups. For comparisons across groups, one-way Analysis of Variance (ANOVA) was employed, followed by Tukey's *post hoc* test. Statistical significance was considered as $p < 0.05$.

3. Results

3.1 ZNF263 is highly expressed in GC

To assess ZNF263 expression in GC tissue and its impact on patient survival, we analyzed ZNF263 expression using the UALCAN database and investigated its association with patient survival. The results revealed that GC patients had significantly higher ZNF263 expression compared to normal tissues (Fig. 1A), and this overexpression was correlated with poorer survival outcomes (Fig. 1B). These findings suggest that elevated ZNF263 expression in GC is associated with a worse prognosis.

3.2 ZNF263 promotes GC cell growth

GC cells were transfected with either si-ZNF263 or a ZNF263 overexpression plasmid. Western blot analysis confirmed successful transfection, with increased ZNF263 expression in the overexpression group and reduced expression in the si-ZNF263 group si-ZNF263 group (Fig. 2A). The cell viability was evaluated using the CCK8 test (Fig. 2B), and cell proliferation was evaluated by EdU experiment (Fig. 2C,D). The results demonstrated that ZNF263 significantly increased the proportion of EdU-positive cells and enhanced cell viability, while si-ZNF263 dramatically decreased both parameters. Therefore, ZNF263 promotes GC cell growth.

3.3 ZNF263 inhibits apoptosis in GC cells

Flow cytometry was used to measure GC cell apoptosis rate. Our findings demonstrated that si-ZNF263 significantly increased both the cleaved-caspase3 and cleaved-PARP protein expressions, as well as the apoptosis rate. In contrast, ZNF263 group exhibited a significant reduction in both cell apoptosis rate (Fig. 3A) and the protein levels of cleaved-caspase3 and cleaved-PARP (Fig. 3B). These findings suggest that ZNF263 inhibits apoptosis in GC cells.

3.4 ZNF263 promotes TXNDC9 transcription

The hTFtarget database predicted that TXNDC9 is a downstream target of ZNF263. Sequence analysis of the TXNDC9 promoter region with ZNF263 using the JASPAR database (Fig. 4A) indicated that ZNF263 might function as a transcription factor for TXNDC9. The top three binding sites were selected for further study (Fig. 4B). ChIP-qPCR confirmed that ZNF263 was recruited to the E1 site of the TXNDC9 promoter region, but not the E2 and E3 sites (Fig. 4C). Dual luciferase reporter assays demonstrated that ZNF263 greatly increased the luciferase activity in the Wild Type (WT)-TXNDC9 area but had no effect on the Mutant (MUT)-TXNDC9 (Fig. 4D). qPCR analysis showed that si-ZNF263 significantly reduced the TXNDC9 mRNA expression in GC cells, whereas ZNF263 increased the TXNDC9 mRNA expression (Fig. 4E). These results confirm that ZNF263 acts as a transcription factor for TXNDC9. Furthermore, overexpression of TXNDC9 reversed the inhibitory effects of si-ZNF263 on GC cell survival and increased apoptosis (Fig. 4F–H).

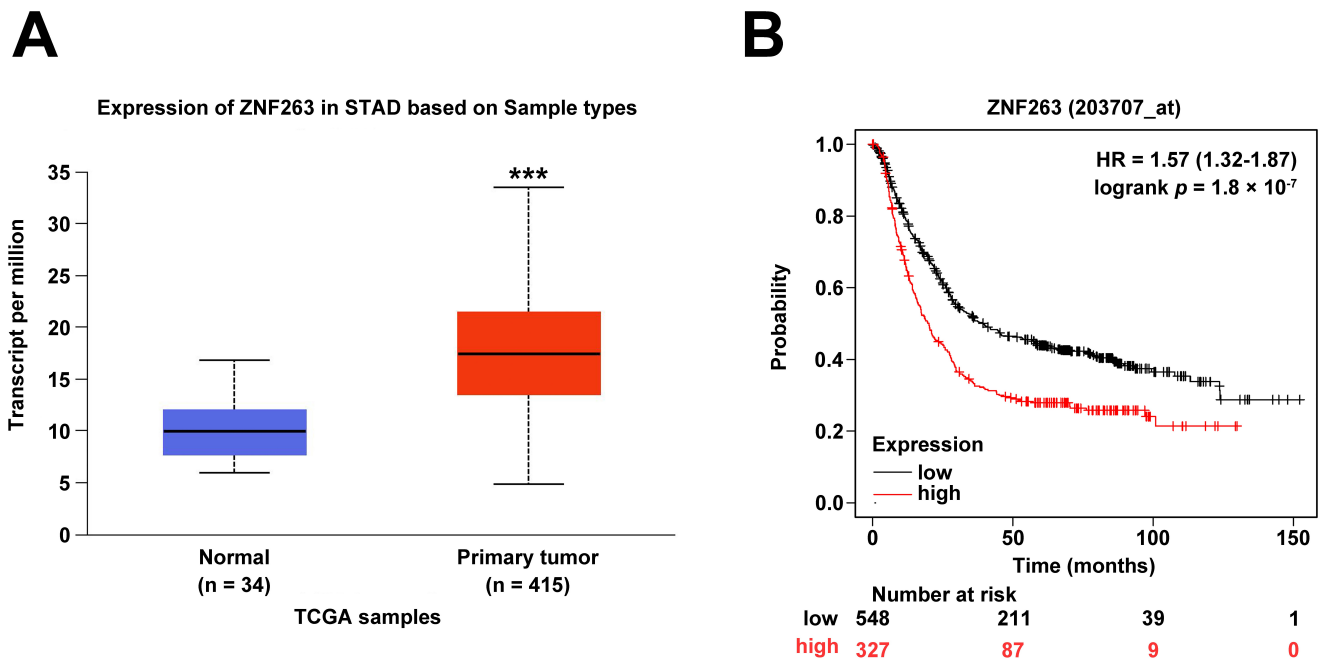

FIGURE 1. ZNF263 is highly expressed in GC. (A) UALCAN database query ZNF263 expression in GC patient tissues. (B) The influence of ZNF263 expression on GC patient survival rate as determined by a Kaplan Meier Plotter database query. ***$p < 0.001$. ZNF: Zinc finger protein; TCGA: The Cancer Genome Atlas; STAD: Stomach Adenocarcinoma; HR: Hazard Ratio.

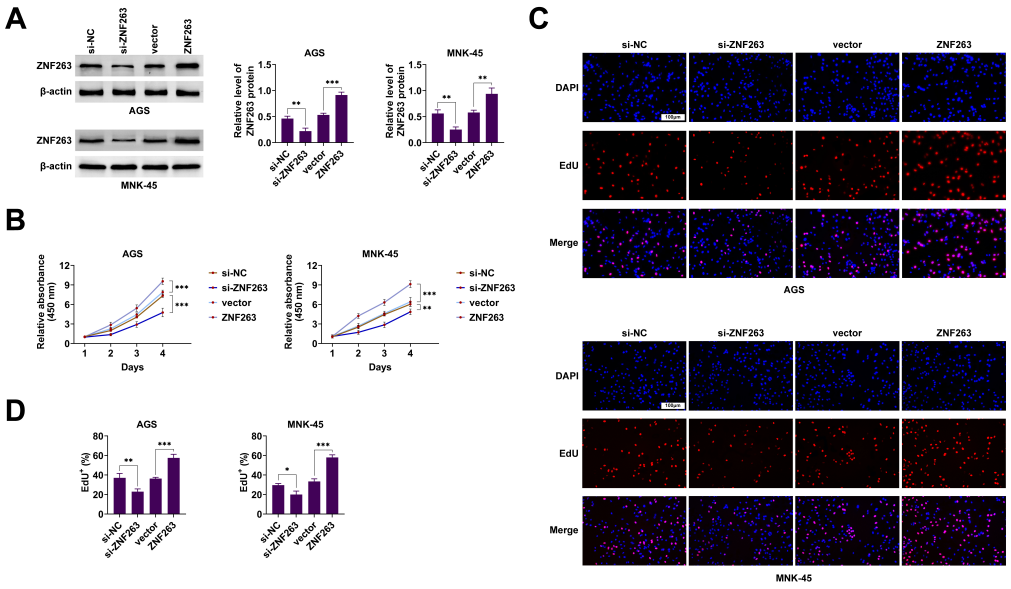

FIGURE 2. ZNF263 promotes GC cell growth. (A) Western blot analysis of ZNF263 protein expression in transfected cells. (B) CCK8 assay for cell viability. (C) EdU detection of cell proliferation. (D) Quantification of EdU-positive cells. $*p < 0.05$, $**p < 0.01$, $***p < 0.001$. ZNF: Zinc finger protein; si-NC: negative control siRNA; EdU: 5-Ethynyl-2′-deoxyuridine; DAPI: 4′,6-Diamidino-2-phenylindole.

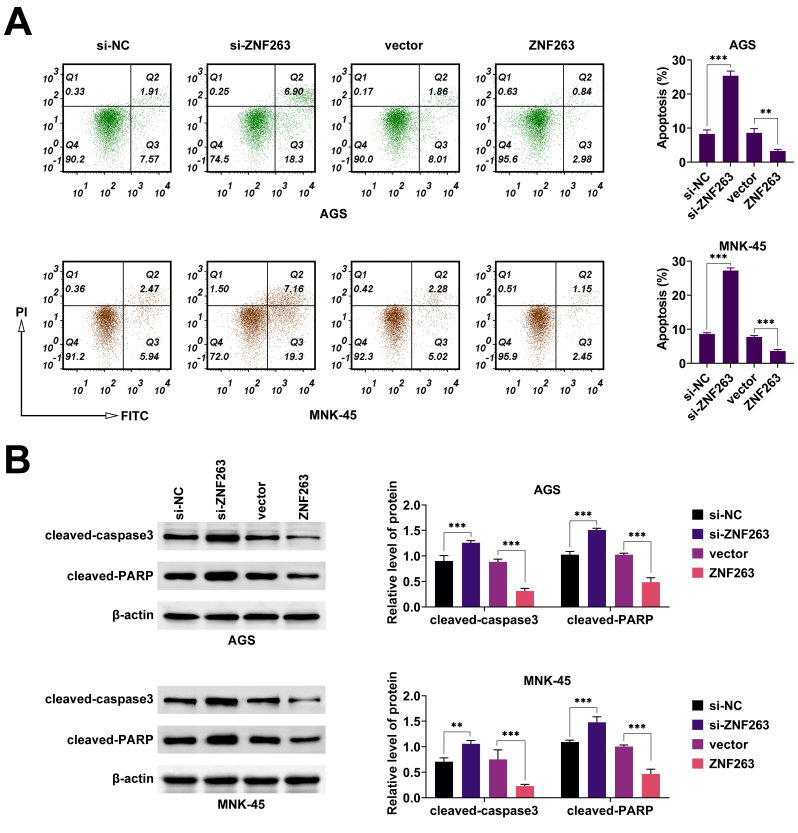

FIGURE 3. ZNF263 inhibits apoptosis in GC cells. (A) Flow cytometry detection of cell apoptosis rate. (B) Western blot analysis of cleaved-caspase3 and cleaved-PARP expressions in cells. $**p < 0.01$, $***p < 0.001$. ZNF: Zinc finger protein; si-NC: negative control siRNA; PI: Propidium Iodide; FITC: Fluorescein isothiocyanate; PARP: Poly(ADP-ribose) polymerase.

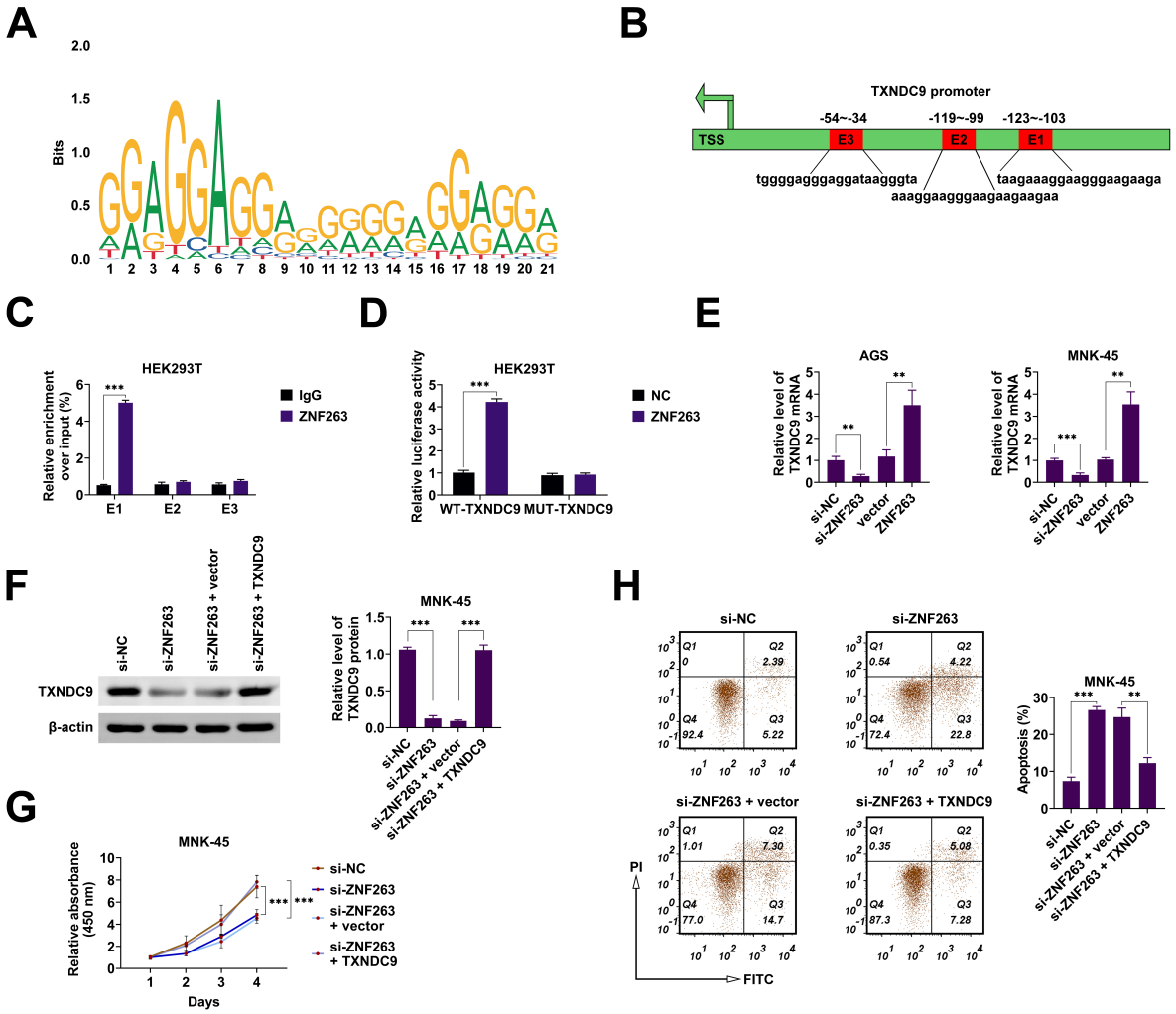

FIGURE 4. ZNF263 promotes TXNDC9 transcription. (A) ZNF263 binding motif found in the JASPAR database. (B) Three sites binding to the TXNDC9 promoter. (C) ChIP-qPCR confirmed the recruitment of ZNF263 to the TXNDC9 promoter sites. (D) Dual-luciferase reporter gene assay confirmed the interaction between ZNF263 and the TXNDC9 promoter. (E) PCR analysis of TXNDC9 mRNA expression following transfection with si-ZNF263 or ZNF263 in GC cells. (F) Western blot analysis of TXNDC9 protein expression in transfected cells. (G) CCK8 assay for cell viability. (H) Flow cytometry detection of cell apoptosis rate. $^{**}p < 0.01$, $^{***}p < 0.001$. TXNDC9: Thioredoxin domain-containing protein 9; ZNF: Zinc finger protein; TSS: Transcription Start Site; IgG: Immunoglobulin G; si-NC: negative control siRNA; PI: Propidium Iodide; FITC: Fluorescein isothiocyanate; WT: Wild Type; MUT: Mutant.

4. Discussion

The genesis and progression of gastric cancer (GC) is characterized by alterations in multiple causal genes, contributing to its genetic diversity. However, the specific molecular mechanisms underlying these processes remain unclear [12]. This work confirms for the first time that ZNF263 stimulates TXNDC9 transcription, facilitating GC growth and inhibiting GC cell apoptosis. These findings provide valuable insights into potential gene-targeted therapies for GC.

ZNF263 is dysregulated in various cancers, where it accelerates the development of malignant tumors. ZNF263 transcriptionally activates Ring Finger Protein 126 (RNF126), driving drug resistance, proliferation, and epithelial-mesenchymal transition (EMT) in pancreatic ductal cancer [13]. ZNF263 expression correlates positively with malignancy, and its levels in non-small cell lung cancer tissues and cell lines (NSCLC). ZNF263 suppresses autophagy and cell death while promoting the migration and proliferation of NSCLC cells [14]. Additionally, hypoxia inhibits transcription of dopamine receptor D5 pseudogene 2 (DRD5P2) through the Hypoxia-Inducible Factor 1-alpha (HIF-1α)/ZNF263 axis, leading to EMT and metastasis in GC [15]. This study shows that ZNF263 is substantially expressed in GC and that patients with elevated ZNF263 expression have a poor prognosis. Knocking down ZNF263 expression results in slower proliferation and increased apoptosis for GC cells, while overexpression of ZNF263 inhibits cell apoptosis and promotes tumor progression.

Thioredoxin (TRX) superfamily of proteins catalyzes the reversible oxidation of cysteine thiol to disulfide. These proteins play a crucial role in regulating oxidative stress, disulfide bond formation, transcription, and redox homeostasis [16–

18]. Recent studies have revealed the crucial role of TRX in carcinogenesis, with increased TRX expression was observed in aggressive tumors [16]. TXNDC9, a unique member of TRX family, can bind proteins with ATP and modulate the folding of actin and tubulin, as well as the ATPase activity of the chaperonin T-Complex Protein 1 (TCP1) complex, which is essential for proper protein folding [19]. TXNDC9 promotes the growth of prostate cancer cells and exhibits pro-oncogenic effects across various malignancies [20]. TXNDC9 knockdown induces apoptosis while inhibiting proliferation, migration, and invasion in lung cancer cells [21]. Elevated TXNDC9 expression has been associated with enhanced Nuclear Factor kappa-light-chain-enhancer of activated B cells (NF-κB) regulation, promoting GC metastatic and predicting poor prognosis [10]. Furthermore, MiR-643 inhibits cell proliferation and invasion by targeting TXNDC9, acting as a potential tumor suppressor gene in GC [11]. This study confirms that ZNF263 accelerates the malignant progression of GC by promoting TXNDC9 transcription, thus uncovering a novel molecular mechanism.

5. Conclusions

In conclusion, this study demonstrates that ZNF263, a transcription factor, stimulates TXNDC9, which, in turn, promotes the carcinogenic development of GC. However, several limitations exist. First, tumor tissue samples were not obtained for measuring ZNF263 and TXNDC9 expressions. Second, no rescue studies were conducted to directly verify that ZNF263 stimulates TXNDC9 transcription to facilitate GC progression. Lastly, *in vivo* studies were not performed to confirm the functional role and mechanisms of action. These aspects will be addressed in future investigations.

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

SLC and BP—designed the study and carried them out; prepare the manuscript for publication and reviewed the draft of the manuscript. SLC, YDW and WYY—supervised the data collection; analyzed the data. SLC, YDW, WYY and HMJ—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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