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ORIGINAL RESEARCH

Downregulated LINC02381 Predicts Tumor Progression and Correlates with Poor Survival in Gastric Cancer

Dong Wang[1,*], Jiahui Zhou[1,*], Tangdan Ding[1], Jingcui Yu[1,2]

[1]Scientific Research Centre, The Second Affiliated Hospital of Harbin Medical University, Harbin, Heilongjiang, People's Republic of China; [2]Key Laboratory of Preservation of Human Genetic Resources and Disease Control in China (Harbin Medical University), Ministry of Education, Harbin, Heilongjiang, People's Republic of China

*These authors contributed equally to this work

Correspondence: Jingcui Yu, Scientific Research Centre, The Second Affiliated Hospital of Harbin Medical University, Harbin, Heilongjiang, People's Republic of China, Tel +86 451 86605908, Email yujingcui@ems.hrbmu.edu.cn

Purpose: The elevated incidence and mortality rates of gastric cancer (GC) highlight the urgent need to identify novel prognostic biomarkers. In this study, we aimed to evaluate the expression of LINC02381 in GC tissues and its correlation with clinicopathological features, and to assess its prognostic significance.

Methods: The expression of LINC02381 was assessed in 49 cases of GC tissues and noncancerous gastric tissues using real-time PCR. The correlation between the expression of LINC02381 and the clinicopathological parameters of GC patients was analyzed by in situ hybridization histochemistry (ISHH) in 78 GC tissues. Subsequently, univariate and multivariable Cox regression analysis, and Kaplan-Meier survival analysis were performed to correlate with clinicopathological variables.

Results: We found the expression of LINC02381 was decreased in GC tissues ($P<0.05$) and was correlated with poorly differentiated GC (Grade III/IV) ($P<0.05$). Kaplan-Meier survival analysis showed that the low expression of LINC02381 was significantly correlated with the overall survival rate of patients for GC, GC patients with low expression of LINC02381 had a poor prognosis ($P<0.05$). Univariate Cox regression analysis showed the expression of LINC02381, tumor size, grade, T stage, and N stage were associated with the prognosis of GC. Multivariate Cox regression analysis revealed that N stage remained an independent prognostic factor.

Conclusion: LINC02381 is downregulated in GC tissues, and its low expression is associated with malignant progression and shorter survival. Although not an independent prognostic factor, it may serve as a potential adjunct prognostic indicator for GC.

Keywords: gastric cancer, LINC02381, prognosis, ISHH

Introduction

Globally, gastric cancer (GC) ranks as the fifth most prevalent malignant tumor and stands as the fourth leading cause of cancer-related deaths.[1] Unfortunately, despite its prevalent occurrence, most patients of GC are diagnosed at advanced stages with poor prognosis, primarily due to the absence of discernible clinical indications.[2] Even after receiving radical resection and adjuvant therapy, postoperative recurrence and mortality rates remain persistently high, with the 5-year overall survival rate for patients with stage IV GC being merely 29.0% (± 3.9%).[3] This underscores the major challenges in achieving long-term survival for GC patients. Therefore, there is an urgent need to identify novel molecular prognostic markers for GC and explore more effective therapeutic strategies.

Genomics studies have revealed that less than 2% of mammalian genomes are transcribed into proteins, while over 98% are transcribed into non-coding RNAs (ncRNAs).[4] Based on their length, ncRNAs are classified into small non-coding RNAs (sncRNAs) and long non-coding RNAs (lncRNAs), with lncRNAs playing a crucial role as potential regulators in physiological processes, particularly at the levels of gene expression and protein synthesis.[5,6] Importantly, functional lncRNAs have been shown to regulate the progression of many types of tumors, acting as either oncogenes or tumor suppressors, this indicates that

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lncRNAs possess significant potential as novel biomarkers and therapeutic targets for cancer.[7,8] LncRNA LINC02381 (aliases: LOC400043, RP11-834C11.4) is a well-characterized competing endogenous RNA (ceRNA) that exhibits context-dependent dual roles in tumorigenesis. It exerts tumor-suppressive effects in colorectal cancer, where it partially regulates the PI3K/Akt pathway by upregulating PTEN and downregulating phosphorylated Akt,[9] and in breast cancer, where its downregulation correlates with aggressive features including lymph node metastasis and estrogen receptor positivity.[10] Conversely, LINC02381 acts as an oncogene in cervical cancer by sponging miR-133b to promote cell proliferation, migration, and invasion;[11] in osteosarcoma, its overexpression is associated with advanced disease and poor prognosis via the LINC02381/miR-503-5p/CDCA4 axis,[12] and in glioma, it interacts with transcription factor CEBPβ to synergistically activate CBX5 transcription.[13] In 2021, Pan et al constructed a prognostic model for GC patients using GEO datasets, in which LINC02381 was included as one of six risk genes involved in predicting clinical features and survival outcomes in GC patients.[14] This study provides important preliminary evidence for the prognostic potential of LINC02381 in GC. However, no independent clinical validation of this potential has been reported in the literature to date, which limits its clinical application as a prognostic biomarker for GC.

In this study, we first profiled LINC02381 expression in GC tissues and found it to be significantly downregulated. Subsequently, we further investigated the association of LINC02381 expression with clinicopathological parameters and the prognosis of GC patients, to assess its potential prognostic value in GC.

Materials and Methods

Tissues and Tissue Microarray

A total of 49 cases of GC and their paired corresponding noncancerous gastric (NG) tissues were collected from patients who underwent surgical resection between 2018 and 2019. The inclusion criteria were as follows: (1) patients had not received any preoperative radiotherapy, chemotherapy, or other anticancer treatments; (2) no history of concurrent or previous malignant tumors. This study was approved by the Ethics Committee of the Second Affiliated Hospital of Harbin Medical University (Ethics Number: KY2018-369).

The tissue microarray containing 90 cases of primary GC tissues (Serial number: HStmA180Su11) with corresponding clinicopathological information was purchased from Shanghai Outdo Biotech Co., Ltd. After excluding 3 cases of tissue detachment (C07, D07, G17), 1 case of nonspecific stromal staining (I03), and 8 cases without GC epithelium (A15, B01, B03, F03, F11, F13, H13, I15), the actual remaining valid information encompassed 78 cases of GC, with 1 case missing tumor size information. The follow-up period for all cases commenced in July 2015 and lasted for 7 to 7.4 years, during which 58 GC patients were confirmed deceased. Prior to the experiment, the tissue microarray was approved by the Ethics Committee of Shanghai Outdo Biotech Co. Ltd. (YB M-05-02).

Real-Time PCR

For each case, approximately 0.1g of tissue was taken and ground with liquid nitrogen, followed by transfer into a 1.5mL microcentrifuge tube. The total RNA from these tissues was extracted using TRIzol reagent (Invitrogen, 66003, USA) and subsequently reverse transcribed into cDNA using the All-in-one First Strand cDNA Synthesis Kit (SEVEN, SM131, China). All PCR reactions were carried out using the 2 × SYBR Green qPCR Master Mix (SEVEN, SM133, China). The expression differences of LINC02381 between different tissue samples were normalized against β-actin and presented as fold differences ($2^{-\Delta\Delta\ CT}$). The sequences of the PCR primers are provided as follows:

LINC02381-forward, 5′-GTGGAGGAACAGAAGTGAAATGA-3′

LINC02381-reverse, 5′-CAATGCTTGGCACAGAATAGGTA-3′

β-actin-forward, 5′-ACCGCGAGAAGATGACCCAG-3′

β-actin-reverse, 5′-TTAATGTCACGCACGATTTCCC-3′

Data Collection and Analysis

The RNA level of LINC02381 in 408 GC tissues and 211 normal gastric tissues (comprising 36 normal tissues and 175 stomach tissues) were performed via the Gene Expression Profiling Interactive Analysis (GEPIA) databases, available at https://gepia2.cancer-pku.cn.

In situ Hybridization Histochemistry (ISHH)

The tissue microarray was baked at 63°C, followed by dewaxing with xylene and alcohol. After digestion with proteinase K (SIGMA, 39450–01-6, Germany) and washing with PBS, dehydration was performed. Subsequently, 20μL of hybridization solution (Exiqon, America) was added to the tissue microarray, which was then sealed and hybridized at 50°C. After hybridization, gradient washing was conducted using the Roche DIG Wash and Block Buffer set (Roche, 11585762001, Switzerland), followed by blocking with Block Buffer. After blocking, the tissue microarray was incubated with a 1:800 dilution of anti-DIG-AP Fab fragments (Roche, 11093274910, Switzerland) at 4°C overnight. Then, following the instructions of the BCIP/NBT Substrate kit (VECTOR Laboratories, SK-5400, United States), the color development process was carried out under light-blocking conditions. After that, the tissue microarray was counterstained using Nuclear Fast Red Staining Solution (Beyotime, C0151, China). Subsequently, the tissue microarray was mounted with neutral resin and observed and photographed under a microscope (NIKON, YS100, Japan).

The results of ISHH were evaluated using the immunoreactivity score (IRS) system and were evaluated in a blinded manner by two independent, board-certified pathologists who were unaware of the patients' clinical and survival data. Inter-observer discrepancies in scoring were resolved via consensus discussion. If no consensus was reached, a third senior pathologist was consulted to render the final decision. Specifically, Staining intensity: 0 points for negative, 1 point for 1+, 2 points for 2+, and 3 points for 3+. Ratio of positive cells: 0 points for negative, 1 point for 1%~25%, 2 points for 26%~50%, 3 points for 51%~75%, and 4 points for 76%~100%. The IRS score was calculated by multiplying the staining intensity and the ratio of positive cells. The cutoff value for defining high versus low LINC02381 expression was set at the median IRS score of all samples (0.1). An IRS score of less than 0.1 is considered negative (low expression), while a score of 0.1 or above is considered positive (high expression).

Statistical Analysis

Statistical analysis and graphing were performed using GraphPad Prism 10.0 software or R software. Experimental data were expressed as mean ± SD. Normality of data distribution was assessed using the Shapiro–Wilk test. For normally distributed data, paired-sample t-tests were used for statistical analysis, while Wilcoxon rank-sum tests were employed for non-normally distributed data. Chi-square tests were applied to analyze the correlation between the LINC02381 expression in GC tissues and the clinicopathological parameters of GC patients. Kaplan-Meier method was used to evaluate the survival prognosis of GC patients, and Log rank tests were performed for statistical analysis. The risk factors affecting the survival of GC patients were assessed using univariate and multivariate Cox proportional hazards regression models. Variables with $P<0.05$ in univariate analysis were included in the multivariate Cox regression model. $P<0.05$ was considered statistically significant.

Results

The Expression of LINC02381 was Decreased in Gastric Cancers

To elucidate the expression pattern of LINC02381 in gastric cancer (GC), we conducted an analysis of RNA-sequencing data from the GEPIA database, encompassing 408 GC tissues and 211 gastric tissues and revealed a distinct down-regulation of LINC02381 RNA levels in GC tissues compared to those observed in gastric tissues (Figure 1A, $P<0.05$). Subsequently, real-time PCR analysis was conducted to determine the expression levels of LINC02381 in a cohort of 49 patients, specifically comparing collected GC tissues with non-cancerous gastric (NG) tissues. The findings, presented in Figure 1B, showed a statistically significant decrease in LINC02381 expression in GC tissues compared to NG tissues ($P<0.05$). Furthermore, we performed an individualized, paired comparative analysis among the 49 patients, assessing the expression levels of LINC02381 specifically in their matched GC and NG tissue samples. As depicted in Figure 1C, an majority of patients (87.76%, 43 out of 49) exhibited a notable downregulation of LINC02381 expression in GC tissues compared to their corresponding NG tissues. These findings consistently demonstrate significant downregulation of LINC02381 in GC.

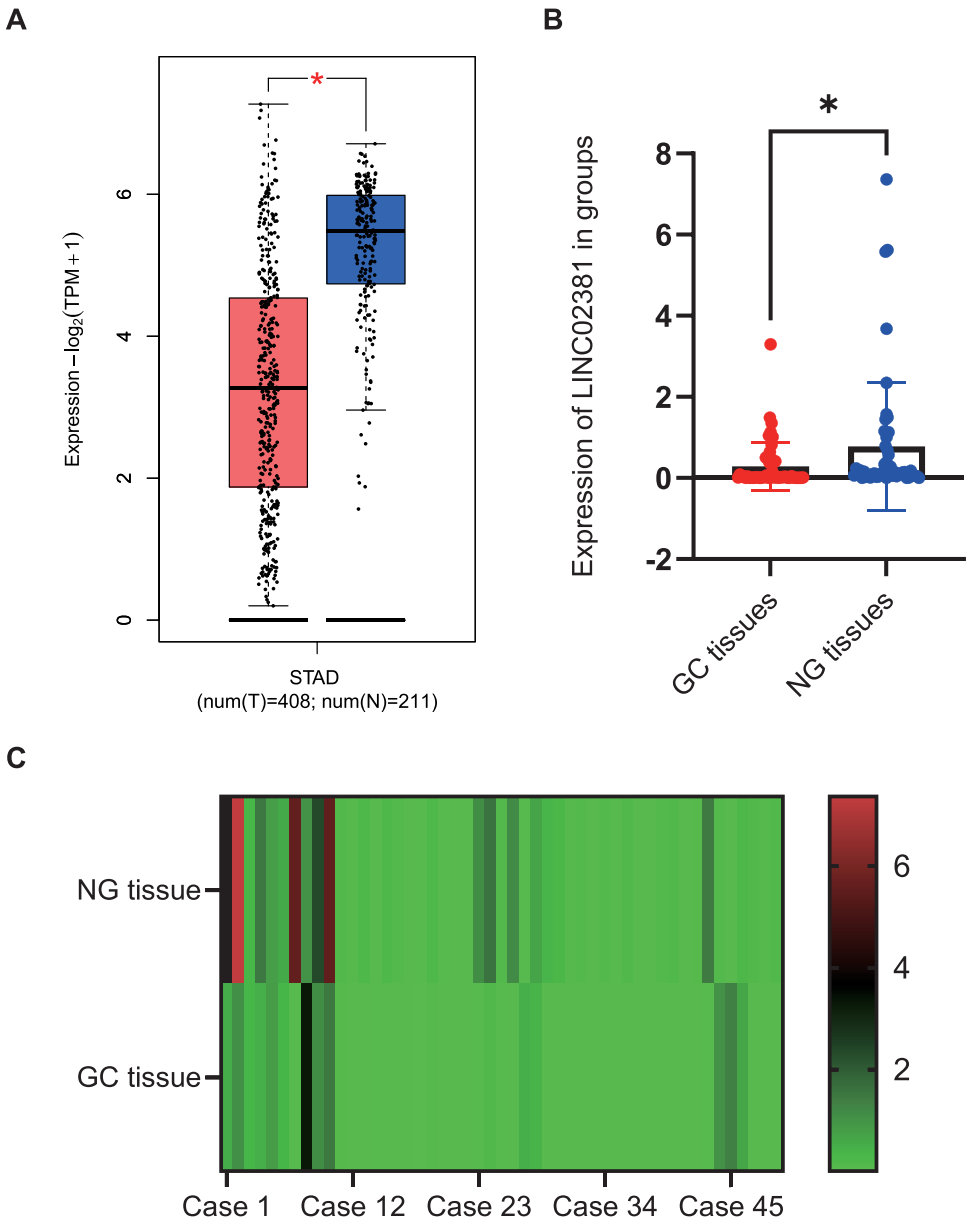

Figure 1 The expression analysis of LINC02381 in gastric cancer. (**A**) The RNA level of LINC02381 (ENSG00000250742) in 408 GC tissues and 211 gastric tissues using GEPIA database. (**B**) The expression of LINC02381 in 49 GC tissues and 49 NG tissues. (**C**) Heat maps showing the personalized comparative analysis of LINC02381 expression in GC patients. (*P< 0.05).

Decreased Expression of LINC02381 is Involved in the Process of Gastric Cancers

Further to evaluate the clinical significance of LINC02381 in GC, we utilized in situ hybridization histochemical (ISHH) to detect the expression of LINC02381 in 78 cases of GC tissues containing valid pathological information (Figure 2A). Based on the IRS score, LINC02381 was categorized into the groups for low expression (<0.1) and high expression (>0.1) (Figure 2B). Chi-square tests were then employed to analyze the correlation between the expression of LINC02381 in GC tissues and clinicopathological parameters of GC patients. The results, as shown in Table 1, indicate that a higher proportion (76.7%, 46/60) of poorly differentiated GCs (Grade III/IV) were observed in GC patients with low LINC02381 expression, whereas in patients with high LINC02381 expression, the proportion of well-to-moderately differentiated GCs (Grade I/II) (50%, 9/18) was significantly higher than the proportion of poorly differentiated cancers (23.3%, 14/60). The low expression of LINC02381 in GC tissues was significantly associated with poorly differentiated GC (Figure 2C, P<0.05). These results suggest the decreased expression of LINC02381 in GC tissues may be involved in the differentiation process of GC.

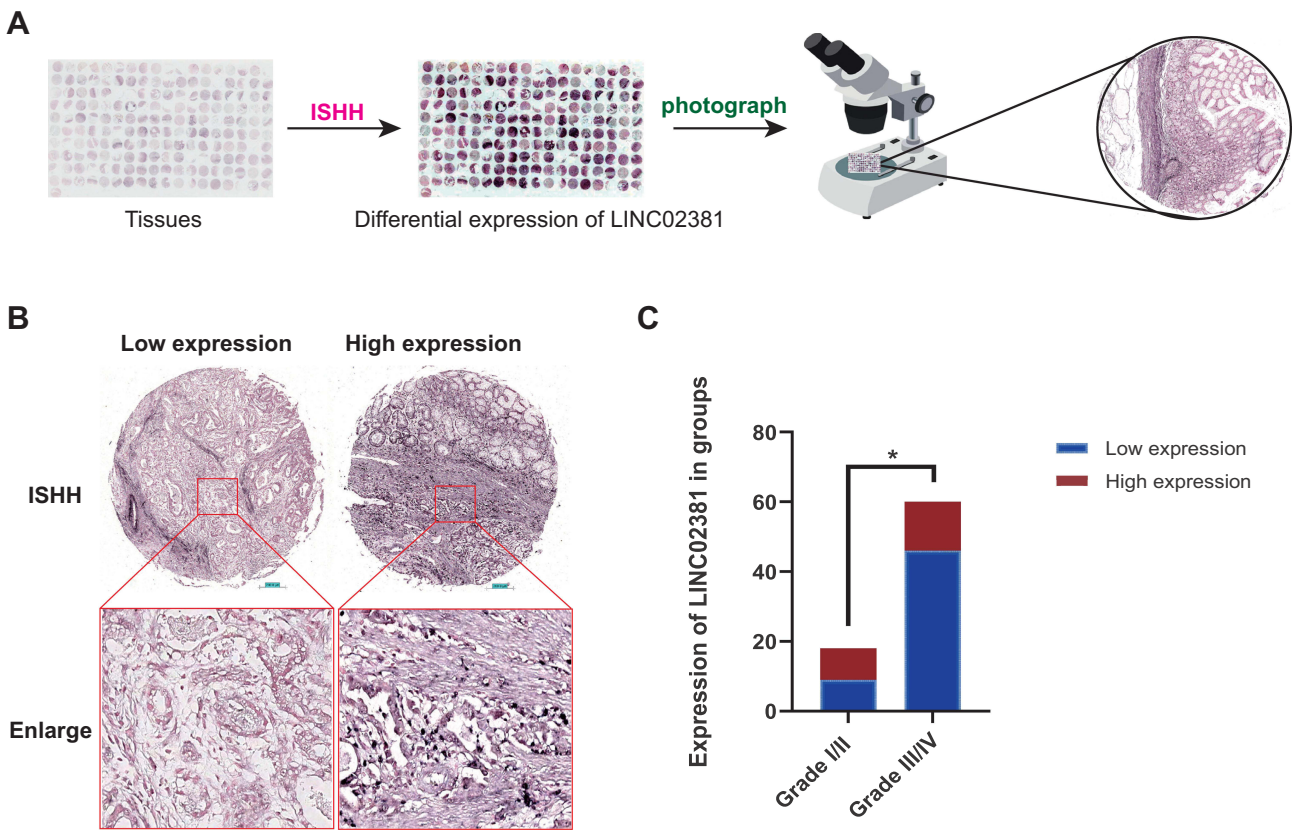

Figure 2 ISHH analysis of LINC02381 was performed in gastric cancers. (**A**) Detection and analysis flow chart of LINC02381 expression in GC tissue microarray. (**B**) Representative cases showing low and high expression of LINC02381 in GC tissues. (**C**) The relationship between the expression of LINC02381 in GC tissues and the grade stage of GC. (*$p<0.05$).

Decreased Expression of LINC02381 is Association with Poor Prognosis of Gastric Cancer Patients

In order to assess the relationship between LINC02381 expression in GC tissues and the prognosis of 78 GC patients, Kaplan-Meier survival curves were employed, followed by Log-rank statistical analysis. Of these, 55 patients exhibited low LINC02381 expression, while 23 displayed high expression. As shown in Figure 3, a statistically significant

Table 1 Correlation Between LINC02381 Expression and Clinicopathological Characteristics in GC Tissues

Variables		LINC02381 Expression		Total	χ^2	p value
		Low	High			
Age (year)	≤55	11	6	17	0.353	0.553
	>55	44	17	61		
Sex	Female	17	9	26	0.493	0.482
	Male	38	14	52		
Tumor size[†]	<5cm	13	10	23	2.899	0.089
	≥5cm	41	13	54		

(*Continued*)

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Table 1 (Continued).

Variables		LINC02381 Expression		Total	χ^2	p value
		Low	High			
Grade	I/II	9	9	18	4.735	**0.030***
	III/IV	46	14	60		
TNM stage	I/II	19	11	30	1.209	0.272
	III/IV	36	12	48		
T stage	T1/T2	3	3	6	1.315	0.251
	T3/T4	52	20	72		
N stage	N0	16	7	23	0.014	0.906
	N1/N2/N3	39	16	55		
M stage	M0	54	23	77	0.424	0.515
	M1	1	0	1		

Notes: *$p < 0.05$, †Tumor size data was missing in 1 case.

correlation (P=0.026) was observed between LINC02381 expression in GC tissues and the patients' overall survival rate. Notably, patients with low LINC02381 expression demonstrated a poorer prognosis.

To further clarify the independent prognostic variables for predicting the prognosis of GC patients, we performed both univariate Cox regression (UCR) and multivariate Cox regression (MCR) analyses (Table 2). The results of the UCR analysis indicated that LINC02381 expression (P=0.048), tumor size (P=0.026), grade (P=0.045), T stage (P=0.037), and N stage (P< 0.001) could be considered as risk factors for mortality in GC patients. However, after adjusting for confounding factors in the MCR analysis, only N stage (HR=6.704, 95% CI=1.861–24.153, P=0.004) remained a statistically significant independent prognostic factor.

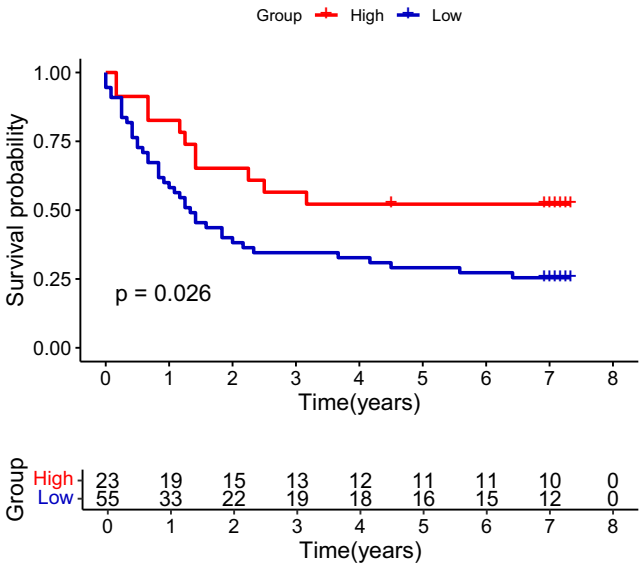

Figure 3 Relationship between LINC02381 expression in gastric cancer tissues and overall survival rate of gastric cancer patients.

Table 2 Univariate and Multivariate Analysis of Factors Associated with Overall Survival in Patients with Gastric Cancer

Variables	Univariate Analysis			Multivariate Analysis		
	HR	95% CI	P value	HR	95% CI	P value
Expression	0.522	0.274–0.995	**0.048***	0.621	0.310–1.242	0.178
Age	1.031	0.546–1.947	0.926			
Sex	0.850	0.494–1.461	0.556			
Tumor size	1.932	1.082–3.451	**0.026***	1.559	0.809–3.003	0.185
Grade	2.069	1.015–4.216	**0.045***	1.946	0.888–4.264	0.096
TNM stage	4.458	2.343–8.480	**<0.001*****	1.206	0.440–3.308	0.716
T stage	4.484	1.093–18.402	**0.037***	0.943	0.199–4.480	0.942
N stage	7.621	3.022–19.218	**<0.001*****	6.704	1.861–24.153	**0.004****
M stage	5.100	0.676–38.467	0.114			

Notes: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

Discussion

The high incidence and mortality of GC underscore the urgent need to identify novel prognostic biomarkers. Recent bioinformatics study have predicted that LINC02381 may be a key component of prognostic gene signatures for GC.[14] Therefore, clarifying the expression difference of LINC02381 in GC tissues and its association with the clinical prognosis of GC patients may provide additional evidence supporting its potential prognostic value in GC.

To characterize LINC02381 expression in GC, we first analyzed RNA sequencing data from 408 GC and 211 normal gastric tissues via the GEPIA database, which revealed reduced LINC02381 expression in GC. We further validated this finding using real-time PCR in 49 paired GC and NG tissues, with consistent results. The differential expression of LINC02381 in GC may indicates its potential prognostic value and suggests an association with tumor heterogeneity. In colon adenocarcinoma, the expression of LINC02381 was found to be up-regulated in tissues, and it's up-regulation suppressed the malignant phenotype of tumor cells.[9] Notably, a 2022 study identified LINC02381 as a pyroptosis-related lncRNA that is overexpressed in colon adenocarcinoma and associated with poor patient prognosis.[15] Similarly, LINC02381 is overexpressed in breast cancer tissues and plays a carcinogenic role.[16] Recent studies have shown that LINC02381 is significantly downregulated in breast cancer tissues and is associated with poor survival of patients.[17] Therefore, we performed paired analysis of LINC02381 expression in 49 pairs of GC and NG tissues. The results indicated that the expression of LINC02381 was decreased in 87.76% (43/49) of GC tissues, accounting for the majority of cases. This further clarified the decreased expression status of LINC02381 in GC patients.

Bioinformatic analysis revealed that LINC02381 RNA expression was positively correlated with patient age and negatively correlated with T stage in colon adenocarcinoma.[18] Furthermore, LINC02381 has been identified as a prognostic lncRNA associated with autophagy,[19] ferroptosis,[20] glycosyltransferase,[21] and pyroptosis,[15] and has emerged as an independent prognostic factor for colon adenocarcinoma patients.[22] To clarify the clinicopathological characterization of LINC02381 in GC, we analyzed the expression of LINC02381 in 78 GC tissues containing valid case information using ISHH, and further analyzed the correlation between the expression of LINC02381 and pathological parameters as well as the survival of GC patients. Our results showed that low LINC02381 expression was significantly associated with poor histological differentiation in GC, and patients with low LINC02381 expression had shorter overall survival. However, multivariate Cox regression analysis did not confirm LINC02381 as an independent prognostic factor in our cohort, which may be due to the relatively small sample size and retrospective study design.

While our study provides robust clinical evidence, several inherent limitations should be acknowledged. As an initial clinical investigation, we employed a single-center retrospective design with a moderate sample size, which may have reduced the statistical power of multivariate analysis and explains why LINC02381 exhibited a prognostic trend but failed to achieve independent statistical significance. Additionally, our findings were derived from a single Chinese cohort and lack validation in external independent populations. Furthermore, the present study focused exclusively on clinical correlation analysis, and functional experiments were not conducted to elucidate the precise molecular mechanisms by which LINC02381 regulates GC

progression. Nonetheless, these limitations do not diminish the clinical relevance of our findings, and future validation and mechanistic studies will help to confirm and extend our conclusions.

Conclusion

In conclusion, the present study provides consistent evidence that suggests LINC02381 is significantly downregulated in GC tissues, and its low expression correlates with poor histological differentiation and reduced overall survival. Although LINC02381 was not identified as an independent prognostic factor in this single-center cohort, it may have potential as an adjunct to conventional prognostic indicators for GC. Further large-scale multicenter prospective studies, functional mechanistic investigations, and exploration of integrating LINC02381 expression with existing clinical prognostic models are warranted to confirm these preliminary findings and improve survival stratification accuracy for GC patients.

Abbreviations

GC, Gastric cancer; ISHH, In situ hybridization histochemistry; ncRNAs, Non-coding RNAs; sncRNAs, Small non-coding RNAs; lncRNAs, Long non-coding RNAs; NG, Noncancerous gastric; GEPIA, Gene Expression Profiling Interactive Analysis; IRS, Immunoreactive score.

Data Sharing Statement

Data supporting the findings of this study are available from the corresponding author upon request.

Ethics Approval Statement

The study was approved by the Ethics Committee of the Second Affiliated Hospital of Harbin Medical University (Ethics Number: KY2018-369), and was conducted in accordance with the World Medical Association Declaration of Helsinki. All the study participants provided informed consent.

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Disclosure

The authors declare no conflicts of interest in this work.

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