Long noncoding RNA LINC00460 promotes carcinogenesis via sponging miR-613 in papillary thyroid carcinoma

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Abstract
Long intergenic noncoding RNA 460 (LINC00460) has been identified as a critical regulator for multiple types of cancers. However, the biological role and underlying mechanism in human papillary thyroid carcinoma (PTC) still remain unclear and need to be uncovered. This study was aimed to ascertain the biological role and molecular mechanism of LINC00460 in PTC progression. Our findings revealed that the level of LINC00460 was significantly upregulated in PTC tissues and cell lines, which was positively correlated with advanced tumor–node–metastasis (TNM) stage and lymph node metastasis. Cellular experiments exhibited that knockdown of LINC00460 decreased proliferative, migratory, and invasive abilities of PTC cells. Mechanism assays noted that knockdown of LINC00460 suppressed cell proliferation, migration, and invasion, and inhibited expression of sphingosine kinase 2 (SphK2, a target of miR-613) in PTC cells, at least in part, by regulating miR-613. These findings suggested that LINC00460 could function as a competing endogenous RNA to regulate SphK2 expression by sponging miR-613 in PTC. Targeting LINC00460 could be a promising therapeutic strategy for patients with PTC.

KEYWORDS
lncRNA LINC00460, miR-613, papillary thyroid carcinoma, SphK2

1 INTRODUCTION

Papillary thyroid carcinoma (PTC) is the most common type of thyroid cancer, accounting for about 90% of all cases (Takano, 2017). The incidence of PTC has steadily increased in the last three decades (Siegel, Miller, & Jemal, 2017). Most PTCs are effectively treated by surgical removal, followed by adjuvant radioactive iodine therapy, and the 5-year survival rate is about 90%. However, some patients have a high degree of invasion with the development trend of dedifferentiation, which lead to poor prognosis (Iftikhar, Ikram, Muhammad, & Nathani, 2018). Therefore, it is urgently need to understand the molecular mechanism involved in PTC tumorigenesis and metastasis for further improving the cure rate and survival rate of patients.

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LINC00460, a lncRNA from chromosome 13, has been shown to be upregulated and function as an oncogene in several types of cancers, including non-small-cell lung cancer (Li et al., 2018), esophageal squamous cell carcinoma (Liang et al., 2017), nasopharyngeal carcinoma (Kong et al., 2018), head and neck squamous cell carcinoma (Cao et al., 2017). It has been shown that LINC00460 expression levels were upregulated in PTC tissues, and correlated with the pathologic stage of PTC (Zhao et al., 2018). However, the specific biological role of LINC00460 in PTC has yet to be elucidated.

The aim of this study was to explore the effect of LINC00460 on the progression of PTC. Our data suggested that the expression of LINC00460 was increased in PTC tissues and cell lines. Knockdown of LINC00460 inhibited PTC cell proliferation, migration, and invasion. Mechanistically, we showed that LINC00460 inhibited cell proliferation, migration, and invasion in PTC by regulating miR-613/sphingosine kinase 2 (SphK2) axis. These results revealed functions of LINC00460/miR-613/SphK2 axis in PTC, which might provide a new insight for the treatment of PTC.

2 | MATERIALS AND METHODS

2.1 | Patient and tissue specimens

Forty-eight pairs of PTC tissues and adjacent noncancerous thyroid tissues were obtained from patients with PTC who underwent radical surgical resections at China–Japan Union Hospital of Jilin University (Changchun, China) at between March 2016 and March 2017. All the samples were diagnosed by pathological examination. None of the patients received any anticancer therapy before surgery. All the clinicopathological features of the patients are summarized in Table 1. All the patients were staged using the eighth edition, TNM classification of American Joint Committee on Cancer. All tissue samples were immediately frozen in liquid nitrogen after resection and sorted at −80°C until use. This study was conducted in strictly accordance with the guidelines and principles of the Declaration of Helsinki, and approved by the Ethical Committee of China–Japan Union Hospital of Jilin University. Written informed consent was obtained from all patients.

2.2 | Cell culture

The human PTC cell lines TPC-1, BCPAP, and IHH-4 and the normal thyroid epithelial cell line (Nthy-ori 3-1) were obtained from the American Type Culture Collection (ATCC, Manassas, VA). All cell lines were maintained in Dulbecco’s modified Eagle’s medium (DMEM; Invitrogen, Carlsbad, CA) supplemented with 10% fetal calf serum (FBS; Sigma-Aldrich, St. Louis, MO), 100 U/ml penicillin (Sigma-Aldrich), and 100 μg/ml streptomycin (Sigma-Aldrich) at 37°C in a humidified atmosphere of 5% CO2.

2.3 | RNA interference and transfection

Small interfering RNAs of LINC00460 (si-LINC00460) and the negative control small interfering RNA (siRNA; si-NC) were synthesized by GenePharma (Shanghai, China). MiR-613 mimics as well as the negative control mimics (miR-NC), miR-613 inhibitor (anti-miR-613) and corresponding negative control (anti-miR-NC) were purchased from RiboBio (Guangzhou, China). PTC cells were transfected with abovementioned molecular production using Lipofectamine 2000 (Invitrogen) according to the manufacturer’s protocol.

2.4 | RNA extraction and quantitative reverse-transcription polymerase chain reaction (qRT-PCR)

Total RNAs from cultured cells or tissues were isolated using TRIzol reagent (Invitrogen) according to the manufacturer’s protocol. Complementary DNA was synthesized from total RNA by the PrimeScript RT Master Mix or the SYBR PrimeScript miRNA RT-PCR Kit (Takara, Dalian, China). Real-time PCR was performed using the SYBR Premix Ex Taq II or the SYBR PrimeScript miRNA RT-PCR Kit (Takara) on the 7900 Real-Time PCR System (Applied Biosystems, Carlsbad, CA). All the primers used in this study are listed Table 2. The expressions of messenger RNA (mRNA) and microRNA (miRNA) were quantified by glyceraldehyde 3-phosphate dehydrogenase (GAPDH) mRNA expression, and U6 as endogenous control, respectively. The relative levels were calculated using the method.

2.5 | Cell proliferation assay

Cell proliferation was detected by Cell Counting Kit-8 (CCK-8; Dojindo, Tokyo, Japan). Briefly, transfected cells were grown in...
96-well plate with 5 x 10^3 cells/well and incubated in 37°C with 5% CO₂ for 24–72 hr. At indicated time, the CCK-8 assay solution (10 μl) was added to each well. The absorbance at 450 nm was measured with an enzyme immunoassay analyzer (Thermo Fisher Scientific, Shanghai, China).

### 2.6 | Wound healing assay

The cell migration ability was detected wound healing assay. Briefly, transfected cells were seeded onto six-well plates, and cultured in DMEM medium containing 10% FBS until 100% confluence. A linear wound was created using a sterile pipette tip. After washing three times with PBS, cells were cultured in free-serum DMEM medium for 24 hr. The migration of cells was observed at 0 and 24 hr after wounding and then photographed under an inverted Phase-contrast Microscope (Olympus, Tokyo, Japan). Migration distance (units) was analyzed as a reduction in the wound’s gap with the NIH ImageJ software (National Institutes of Health, Bethesda, MD).

### 2.7 | Transwell invasion assay

Cell invasion ability were determined by Transwell insert chambers (Corning Costar, Lowell, MA) covered with Matrigel (BD Bioscience, Franklin Lakes, NJ). Transfected cells (2 x 10^5) were suspended in serum-free medium and seeded into the upper chamber, whereas DMEM medium supplemented with 10% FBS was added into the bottom chambers as chemoattractant. After incubation at 37°C for 48 hr, the invaded cells were fixed in 20% methanol and stained with 0.1% crystal violet for 15 min. The cells were counted in at least three randomly selected microscopic fields (×100) per filter under an inverted Phase-contrast Microscope (Olympus).

### 2.8 | Dual luciferase reporter assay

LINC00460 was predicted to be a directly regulated target of miR-613 by miRcode bioinformatics tools (http://www.mircode.org/). The 3′-untranslated region (3′-UTR) UTR fragment of LINC00460 was amplified and cloned into psiCHECK-2 vectors (Promega Corp., Madison, WI), which was named as WT-LINC00460. The LINC00460-3′-UTR-mutant construct was performed using GeneTailor Site-Directed Mutagenesis System (Invitrogen, Guangzhou, China), which named as Mut-LINC00460. For reporter assays, 200 ng of reporter plasmid and 100 nM of either miR-613 mimics or miR-613 inhibitor were cotransfected into TPC-1 and BCPAP cells for 48 hr. The luciferase activity assays were determined using a Dual Luciferase Reporter Gene Assay Kit (Beiyotime Institute of Biotechnology, Shanghai, China) according to the manufacturer’s protocol. The relative luciferase activity was normalized to Renilla luciferase activity.

### 2.9 Western blot analysis

Total protein was lysed in radioimmunoprecipitation assay lysis buffer (Beiyotime Institute of Biotechnology) supplemented with protease inhibitor (Beiyotime Institute of Biotechnology) on ice for 30 min. The concentrations of protein were determined using the Bradford assay (Bio-Rad Laboratories, Inc., Hercules, CA). Proteins were isolated with 10% sodium dodecyl sulfate-polyacrylamide gel electrophoresis and transferred to polyvinylidene fluoride membranes (EMD Millipore, Billerica, MA) at 80 V for 2 hr at 4°C. Following blocking with 5% skim milk in Tris-buffered saline tween (TBST), the membranes were probed with the primary antibodies against SphK2 (1:2,000; Santa Cruz Biotechnology, Inc., Dallas, TX) and GAPDH (1:4,000; Santa Cruz Biotechnology, Inc.) overnight at 4°C. Subsequently, the membranes were incubated with the horse-radish peroxidase-conjugated goat anti-mouse IgG (1:5,000 dilution; Santa Cruz Biotechnology, Inc.) for 2 hr at room temperature. Protein bands were visualized using enhanced chemiluminescence (Thermo Fisher Scientific) by a Molecular Imager ChemiDoc XRS System (Bio-Rad Laboratories, Inc.).

### 2.10 Statistical analysis

All data were presented as the mean ± standard deviation from at least three independent experiments with similar results. The significant differences between different groups were analyzed by the Student t test (comparison for two groups) or one-way analysis of variance (comparison for >2 groups) using SPSS version 19.0 (IBM Corp., Armonk, NY). The correlations were analyzed using Spearman’s rank test. Differences were considered statistically significant when the p < 0.05.

### 3 RESULTS

#### 3.1 LINC00460 was upregulated in PTC tissues and cell lines

To investigate the LINC00460 expression profiles in PTC tissues, qRT-PCR analysis was performed in 48 PTC tissues and adjacent normal

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**Table 2** Real-time PCR primers used for mRNA expression analysis

| Target genes | Prime(5′–3′) |
|--------------|-------------|
| U6           | F-TCCGATCGTGAAGCGTTTC | R-GTGCAAGGGTCCAGAGGT |
| Mir-613      | F-GCGACACCATCGTTTGT   | R-AGAGATTCCGGTCGATGCT |
| LINC00460    | F-GTGGATGAGAAGCAGAGGTACG | R-CCTTCCCCAGCTGCTTTTT |
| SphK2        | F-TTCTATTGTCGTAATCCCCCTTGG | R-AGCCCCTTCACGACCTCA |
| GAPDH        | F-AAGTGGAGGTCGGAGTCAA  | R-AAATGAGGGGTATTGAGGG |

Note. F: forward; mRNA: messenger RNA; PCR: polymerase chain reaction; R: reverse.
The results showed that the expression of LINC00460 was significantly upregulated in PTC tissues (Figure 1a). Furthermore, we measured the LINC00460 expression with different clinical pathological status of patients with PTC and the result revealed that the patients with higher expression of LINC00460 had advanced tumor–node–metastasis (TNM) stage and lymph nodes metastasis (Table 1 and Figure 1b,c). However, no correlation was observed between LINC00460 expression and patient’s age, gender, and tumor size (Table 1). We also examined the LINC00460 expression in PTC cell lines by qRT-PCR. The data revealed that LINC00460 was remarkably upregulated in three PTC cell lines (TPC-1, BCPAP, and IHH-4) compared with the normal thyroid epithelial cell line Nthy-ori 3-1 (Figure 1d).

3.2 | LINC00460 knockdown suppressed cell proliferation, migration, and invasion in PTC cells

To investigate the potential role of LINC00460 in regulating PTC cell growth and metastasis, LINC00460 was knocked down in TPC-1 and BCPAP cells by transfection with an siRNA against LINC00460 (si-LINC00460). The result of qRT-PCR demonstrated that the transfection of si-LINC00460 significantly downregulated the level of LINC00460 expression in the two cell lines compared with cells transfected with si-NC (p < 0.05; Figure 2a). Subsequently, cell proliferation, migration, and invasion were determined in PTC cells transfected with si-LINC00460. We found that knockdown of LINC00460 significantly inhibited cell proliferation (p < 0.05; Figure 2b, c), migration (p < 0.01; Figure 2d) and invasion (p < 0.05; Figure 2e) in both cell lines compared with cells transfected with miR-NC. These results implied that LINC00460 might play crucial roles in the regulation of PTC progression.

3.3 | MiR-613 was a direct target of LINC00460 in PTC cells

It has been shown that lncRNA could serve as competing endogenous RNAs (ceRNA) to exert its regulatory functions (Bayoumi et al., 2016). To further determine the underlying mechanism by which LINC00460 exerted oncogene role in PTC progression, we adopted a prediction software miRcode to predict miRNAs that interacts with LINC00460. We found that miR-613 was a potential target of LINC00460 based on a potential binding site in LINC00460 with miR-613 (Figure 3a). To validate it, dual luciferase reporter assays were conducted. Results revealed that transfection with miR-613 mimic significantly inhibited the luciferase activity of Wt-LINC00460 in both TPC-1 and BCPAP cells, whereas miR-613 inhibitor increased the activity (both p < 0.05; Figure 3b,c). When the binding site was mutated, miR-613 mimics or inhibitors cannot regulate the luciferase activity, which implied that miR-613 directly bond to...
Furthermore, qRT-PCR showed that miR-613 overexpression significantly decreased the expression of LINC00460 in TPC-1 and BCPAP cells, whereas miR-613 inhibitor increased the expression of LINC00460 in two PTC cells (both p < 0.05; Figure 3d,e). Besides, qRT-PCR analysis showed that knockdown of LINC00460 significantly increased the expression of LINC00460 in TPC-1 and BCPAP cells (p < 0.01; Figure 3f). These results implied that miR-613 is a direct target of LINC00460 in PTC cells.

3.4 | MiR-613 mediated the tumor-suppressive effects of LINC00460 knockdown on PTC cells

To further determine whether LINC00460 exerted biological function in PTC relies on negatively regulating miR-613, we knocked down LINC00460 and inhibited miR-613 in the same time in TPC-1 and BCPAP cells, then cell proliferation, migration, and invasion were determined. The result of qRT-PCR demonstrated that knockdown of LINC00460 significantly increased miR-613
expression, whereas miR-613 inhibition in the meantime restore miR-613 expression in TPC-1 cells and BCPAP cells (p < 0.05; Figure 4a). CCK-8, wound healing and transwell invasion assays showed that LINC00460 knockdown significantly inhibited cell proliferation migration and invasion in TPC-1 cells and BCPAP cells, whereas miR-613 inhibitor in the meantime abrogated inhibition effects by LINC00460 knockdown (Figure 4b-e). These results suggested that that LINC00460 regulated PTC cell progression through mediating miR-613.

### 3.5 | LINC00460 modulated SphK2 expression by regulating miR-613 in PTC cell lines

SphK2, a known oncogene that promotes cancer progression in multiple types of cancers, was identified as a downstream effector of miR-613 in PTC (Qiu, Yang, Fan, & Zheng, 2016). Thus, we wonder whether LINC00460 regulated SphK2 via regulating miR-613. si-NC, si-LINC00460, si-LINC00460 + anti-miR-NC, and si-LINC00460 + miR-613 inhibitor were separately transfected into TPC-1 or BCPAP cells, then SphK2 expression was determined. qRT-PCR and western blot analyses revealed that knockdown of LINC00460 led to a significant decrease of SphK2 mRNA (p < 0.05; Figure 5a,b) and protein expression (p < 0.05, Figure 5c,d) in both TPC-1 and BCPAP cells, whereas miR-613 inhibitor reversed the trends. Moreover, we also found that LINC00460 expression was negatively correlated with miR-613 expression (Figure 5e) in PTC tissues, and positively correlated with SphK2 expression in PTC tissues (Figure 5f). These results indicated that LINC00460 modulated SphK2 by regulating miRNA-613 in PTC.

### 4 | DISCUSSION

Dysregulation of IncRNAs has been proven to be involved in tumorigenesis and progression of PTC, suggesting the potential of IncRNAs to serve as novel target for PTC diagnosis and therapy (Jing et al., 2018; Murugan et al., 2018). In the present study, we demonstrated that LINC00460 was upregulated in the PTC tissues and cell lines, and increased LINC00460 expression was closely associated with TNM stage and lymph node metastasis. Knockdown of LINC00460 in TPC-1 and BCPAP cells significantly inhibited cell proliferation, invasion, and migration. Further mechanistic studies...
highlighted LINC00460 knockdown inhibits cell proliferation, invasion, and migration at least partly via regulating miR-613/SphK2 axis in PTC cells. These results imply that LINC00460 acts as a promoter of PTC progression.

LINC00460, located at chromosome 13q33.2, has been reported to be closely associated with progression in several types of cancers. For instance, LINC00460 promoted nasopharyngeal carcinoma tumorigenesis through regulating miR-149-5p/IL6 signal pathway (Kong et al., 2018). LINC00460 promoted cell migration and invasion, and induces epithelial–mesenchymal transition in non-small-cell lung cancer by regulating hnRNPK (Peng et al., 2018). LINC00460 depletion suppressed esophageal squamous cell carcinoma cell growth through regulating cell proliferation and cell cycle and apoptosis (Liang et al., 2017). Although LINC00460 expression was reported to be upregulated and positively correlated with the pathologic stage of PTC (Zhao et al., 2018), the detail function and underlying mechanism of LINC00460 remain unclear. Here, we first examined the expression level of LINC00460 in PTC tissues and cell lines through qRT-PCR analysis. The data showed that LINC00460 was upregulated in the PTC tissues and cell lines, and increased LINC00460 expression was closely associated with TNM stage and lymph node metastasis, which was consistent with previous result (Zhao et al., 2018). And then we identified the function of LINC00460 by applying reduced expression approaches. It was found that knockdown of LINC00460 significantly inhibited proliferation, migration and invasion in TPC-1 cells and BCPAP cells. These results suggested that LINC00460 played oncogene role in PTC progression.

Many lncRNAs has been regarded as ceRNAs to sponge miRNAs via competitively binding common microRNAs (Thomson & Dinger, 2016). We hypothesized that LINC00460 might also serve as a ceRNA exerting its biological function in PTC. To explore the correlation between LINC00460 and miRNA in PTC pathogenesis, we made a predication by bioinformatics analysis and found that the miR-613 had a higher score binding to LINC00460. MiR-613 was proven to function as a tumor suppressor in multiple types of cancers (Li et al., 2016; Sang, Liu, & Sun, 2018). Importantly, it has been showed that miR-613 overexpression significantly inhibited PTC cell proliferation, migration and invasion (Qiu et al., 2016). By luciferase reporter assays, we validated that LINC00460 directly bond to miR-613. Meanwhile, knockdown of LINC00460 increased miR-613 expression in PTC cells. These results implied that LINC00460 was a target of miR-613 in PTC. To further determine whether LINC00460 exerted oncogene roles in PTC by inhibition of miR-613, we performed...
CCK-8, wound healing and transwell invasion assays. Results demonstrated that inhibitory effects on cell proliferation, migration, and invasion induced by LINC00460 knockdown were partially reversed by inhibition of miR-613 in the meantime. These findings indicated that the oncogene LINC00460 promoted PTC progression by negatively regulating miR-613.

It has been shown the binding of miRNAs to IncRNAs could decrease miRNA levels, which results in increasing in the expression of miRNA target genes (Awan, Shah, Rashid, & Shan, 2017; L. J. Li, Leng, Fan, Pan, & Ye, 2017). SphK2 has been proven to be a target of miR-613 in PTC (Qiu et al., 2016). To investigate whether LINC00460 regulated SphK2 via regulating miR-613, qRT-PCR and western blot

**FIGURE 5** LINC00460 modulated SphK2 expression by regulating miR-613 in PTC cell lines. (a, b) The relative mRNA expression level of SphK2 was detected in TPC-1 (a) and BCPAP (b) cells transfected with si-NC, si-LINC00460, si-LINC00460 + anti-miR-NC, si-LINC00460 + anti-miR-613. (c, d) The SphK2 protein expression was determined by western blot in TPC-1 (c) and BCPAP (d) cells transfected with si-NC, si-LINC00460, si-LINC00460 + anti-miR-NC, and si-LINC00460 + anti-miR-613. (e) Spearman’s correlation analysis was used to determine the correlations between LINC00460 expression level and miR-613 in PTC tissues. (f) Spearman’s correlation analysis was used to determine the correlations between LINC00460 expression level and SphK2 mRNA expression levels in PTC tissues. *p < 0.05, **p < 0.01. LINC00460: long intergenic noncoding RNA 460; miR: microRNA; mRNA: messenger RNA; PTC: papillary thyroid carcinoma; SphK2: sphingosine kinase 2
assay were performed. The results showed that knockdown of LINC00460 led to a significant decrease of SphK2 mRNA and protein expression in both TPC-1 and BCPAP cells, whereas miR-613 inhibitor reversed the trends. Moreover, LINC00460 expression was positively correlated with SphK2 expression in PTC tissues. These results suggested that LINC00460 modulated the expression of SphK2, at least in part, by competing with miR-613 in PTC.

Taken together, the present study first demonstrated that the expression of LINC00460 was upregulated in PTC tissues and cell lines. And the elevation of LINC00460 was correlated with advanced TNM stage and lymph node metastasis. Our results also revealed that the oncogene LINC00460 promoted PTC progression via partly regulating miR-613/SphK2 axis. These findings suggested that the LINC00460/miR-613/SphK2 might act as a novel therapeutic target for the treatment of PTC.

CONFLICTS OF INTEREST

All authors declare that there are no conflicts of interest.

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How to cite this article: Feng L, Yang B, Tang X-D. Long noncoding RNA LINC00460 promotes carcinogenesis via sponging miR-613 in papillary thyroid carcinoma. J Cell Physiol. 2019;234:11431–11439. https://doi.org/10.1002/jcp.27799