



# LncRNA IGBP1-AS1 targets miR-150-5p to increase ZEB1 expression in nasopharyngeal carcinoma

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**Background:** Nasopharyngeal carcinoma (NPC), which is a form of cancer arising from the epithelium of the nasopharynx, remains highly prevalent, particularly in Southeast Asia and Southern China. Dysregulated long non-coding RNAs (lncRNAs) are involved in several malignancies, including the growth and aggression of tumors. LncRNA IGBP1-AS1 plays an important role in the advancement of breast cancer, but its role in NPC has not yet been explored.

**Methods:** LncRNA IGBP1-AS1 levels in human NPC samples compared with their matched adjacent normal tissues by quantitative reverse transcription polymerase chain reaction (qRT-PCR). The proliferation alteration of NPC cells was tested *in vitro*. Luciferase reporter assay and RNA immunoprecipitation (RIP) were carried out to reveal the interaction between lncRNA IGBP1-AS1, miR-150-5p, and zinc finger e-box binding homeobox 1 (ZEB1).

**Results:** In the study, we found that IGBP1-AS1 was found to be significantly upregulated in the NPC samples and cell lines. The knockdown of IGBP1-AS1 impeded *in-vitro* proliferation of the NPC cells. Bioinformatics and reporter assays revealed an association between IGBP1-AS1 and miR-150-5p. The expression of ZEB1 was increased by the microRNA (miRNA) sequestering in human NPC.

**Conclusions:** The progression of NPC tumors is facilitated by lncRNA IGBP1-AS1 via miR-150-5p and regulates ZEB1 expression.

**Keywords:** Zinc finger e-box binding homeobox 1 (ZEB1); miR-150-5p; metastasis; IGBP1-AS1; nasopharyngeal carcinoma (NPC)

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## Introduction

As the most frequently occurring malignancy in humans, nasopharyngeal carcinoma (NPC) is a major reason for cancer-related deaths around the world (1,2). NPC is aggressive and exhibits a high recurrence of tumors and metastasis (3). There has been no significant increase in the 5-year survival rate for NPC patients (3); thus, it is essential that new therapeutic strategies are developed that are more

effective at treating patients with NPC.

Long non-coding RNAs (lncRNAs; i.e., those 200 bases long) (4,5) cannot undergo translation, and their dysregulation has been observed in several cancers, such as NPC (6-8). For example, IGBP1-AS1 is a novel lncRNA that has been reported to play a key role in the advancement of malignant breast cancer (9). The mechanistic aspects of this molecule in NPC need to be elucidated. This study sought to assess the level, pattern,

and knockdown of IGBP1-AS1 in NPC samples and cell lines. The involvement of this molecule in aggressive NPC, its interaction with miR-150-5p, and the expression of zinc finger e-box binding homeobox 1 (ZEB1) were also evaluated. Finally, we attempted to elucidate the function of the IGBP1-AS1/miR-150-5p/ZEB1 regulatory network in NPC progression. We present the following article in accordance with the MDAR reporting checklist (available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-291/rc>).

## Methods

### *Sample and cell line details*

The cell lines SUNE-1, C666-1, CNE-2, and CNE-1, NP69 (human nasopharyngeal epithelial cells), and 293T were provided by Nanjing KeyGen Biotech Co., Ltd. (Nanjing, China). The cell lines for NPC and NP69 were maintained using 1640 media with 10% fetal bovine serum (Invitrogen, Carlsbad, CA, USA), with 100 µg/mL of streptomycin plus penicillin. Each cell line was maintained in a 37 °C incubator with air containing and 5% carbon dioxide (CO<sub>2</sub>). The Shijiazhuang People's Hospital provided 30 NPC and equivalent adjacent normal tissue samples. Written informed consent was obtained from the subjects prior to the sampling. No patient received any therapeutic intervention before the collection, and the samples were subjected to immediate freezing in liquid N<sub>2</sub>, and kept at -80 °C. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by committee board of Shijiazhuang People's Hospital.

### *Assay for quantitative reverse transcription PCR (qRT-PCR)*

RNA was isolated using TRIzol from Invitrogen. Total miRNAs were isolated using the miRNeasy FFPE kit from Qiagen (Redwood City, CA, USA). Preparation of the first-strand of the complementary DNA (cDNA) involved the reverse-transcription of RNA using the reverse transcriptase kit of TakaraBio (Tokyo, Japan). The qRT-PCR was performed using the SYBR Green PCR Master Mix from TakaraBio on the ABI Prism 7500 real-time PCR system of Applied Biosystems Life Technologies. The  $2^{-\Delta\Delta C_t}$  approach was used to assess the level of the genes. The

endogenous controls of GAPDH and U6 were employed to detect messenger RNA (mRNA) and microRNA (miRNA) expression, respectively.

### *Assays for cell proliferation*

Ninety-six-well plates received  $2 \times 10^3$  cells per well of the indicated NPC cells. After 12, 24, 36, or 48 h, 3-(4,5-dimethyl-2-thiazolyl)-2,5-diphenyl-2-H-tetrazolium bromide (20 µL) was added, after which the cells were incubated for 20 min with DMSO (200 µL) in 37 °C. The optical density (OD) values were estimated at 490 nm on an analyzer for enzyme immunoassays from Bio-Rad Laboratories (California, CA, USA).

### *Reporter assays for luciferase*

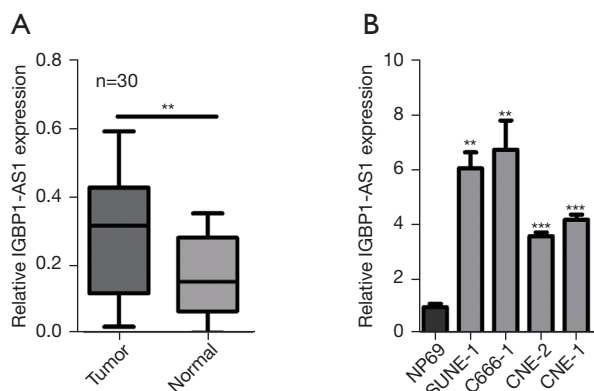
After culturing 293T in 24 well plates for 1 day, the cells were transfected with wild-type (WT) or mutant (MUT) IGBP1-AS1 (via the reporter vector pmirGLO; Promega) combined with miR-150-5p mimic (100 nM). The dual-luciferase reporter system (Promega) was used to assess the enzyme activity 48 h post-transfection.

### *Assays for RNA immunoprecipitation (RIP)*

The Magna RIP<sup>TM</sup> RNA-Binding Protein Immunoprecipitation Kit (Millipore, Billerica, MA, USA) was used in accordance with the prescribed protocols. Following the transfection of 293T cells with agomiR-150-5p or the NC (agomiR-NC), the assay was conducted 48h post-transfection by employing anti-AGO2 (Proteintech, 10686-1-AP, China). Next, qRT-PCR was used to analyze of the relative IGBP1-AS1 levels. The Ct value of the RIP fraction was normalized to that of the input RNA fraction.

### *Statistical analysis*

The data [displayed as the mean ± standard deviation (SD)] were analyzed using SPSS 20.0 from IBM Corp. (NY, USA). Paired *t*-tests were used to compare lncRNA IGBP1-AS1 and miR-150-5p expression between the tumor and adjacent non-tumor tissues. A one-way analysis of variance or Student's *t*-test (two-tailed) and Dunnett's post-hoc test was used. The miR-150-5p and IGBP1-AS1 interaction was analyzed on the basis of the Pearson correlation. A *P* value <0.05 was considered statistically significant.



**Figure 1** Upregulation of IGBP1-AS1 in NPC. (A) qRT-PCR to assess the level of IGBP1-AS1 in NPC *vs.* normal tissues (n=30). (B) qRT-PCR estimation of IGBP1-AS1 in NPC cell lines against NP69. To analyze the relative expression of IGBP1-AS1, the inverse log of  $\Delta\Delta Cq$  was obtained, after which the data were normalized with reference to GAPDH. \*\*,  $P < 0.01$ ; \*\*\*,  $P < 0.001$ . NPC, nasopharyngeal carcinoma; qRT-PCR, quantitative reverse transcription PCR.

## Results

### Upregulation of IGBP1-AS1 in human NPC

Thirty tissues of NPC and equivalent healthy tissues were sampled, and IGBP1-AS1 expression was examined via qRT-PCR. As *Figure 1A* shows, IGBP1-AS1 was more highly expressed in the malignant tissues than normal tissues. IGBP1-AS1 was more highly expressed in the cell lines mentioned in the “Methods” section (see above) than the control (NP69) cells (see *Figure 1B*). Thus, IGBP1-AS1 levels were elevated in NPC.

### The growth of NPC is promoted by IGBP1-AS1

To examine the role of IGBP1-AS1 in NPC progression, we used siRNA targeting IGBP1-AS1 (siIGBP1-AS1) and knocked down IGBP1-AS1 in SUNE-1 and C666-1 cells (see *Figure 2A*). We then used qRT-PCR to determine the IGBP1-AS1 levels. The viability of the SUNE-1 and C666-1 cells was impeded by the IGBP1-AS1 silencing (see *Figure 2B*).

### The binding of LncRNA IGBP1-AS1 with miR-150-5p in NPC

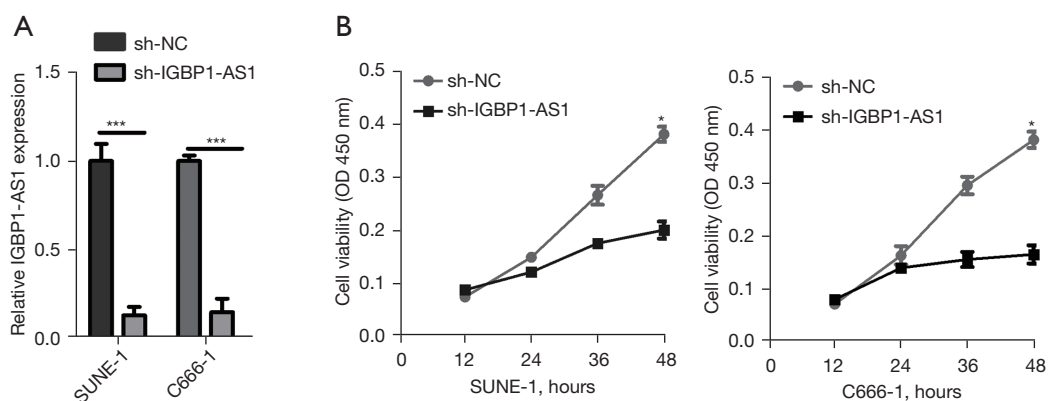
The starBase tool (<https://starbase.sysu.edu.cn/>) was used to

pinpoint potential miRNAs interacting with the lncRNA, and miR-150-5p was found to be targeted by IGBP1-AS1 (see *Figure 3A*). HEK-293T was co-transfected with IGBP1-AS1-WT (WT IGBP1-AS1) or IGBP1-AS1-MUT (MUT IGBP1-AS1) combined with miR-150-5p mimic using the pmirGLO reporter vector. The presence of miR-150-5p caused a dip in the luciferase activity of the cells transfected with IGBP1-AS1-WT that was not seen in the IGBP1-AS1-MUT-transfected cells (see *Figure 3B, 3C*). The RIP assay revealed an association between miR-150-5p and IGBP1-AS1 and the involvement of AGO2 (the molecule involved in the activity of miRNAs to control target gene expression) (see *Figure 3D*). The transfection of the miR-150-5p mimic resulted in a dip in IGBP1-AS1 levels, while a boost in IGBP1-AS1 levels was observed when miR-150-5p was used for silencing in SUNE-1 and C666-1 (see *Figure 3E-3H*).

### The aggressiveness of NPC cells is regulated by IGBP1-AS1 via miR-150-5p/ZEB1 axis modulation

The bioinformatics tool Targetscan (<http://www.targetscan.org/>) revealed that ZEB1 was a potential miR-150-5p target, and it was subject to subsequent analyses (see *Figure 4A*). After the miR-150-5p transfections, the activity of luciferase in NPC cells was significantly impeded following transfection with ZEB1 3'-untranslated region (3'-UTR) WT cells containing Renilla luciferase plasmids. However, no such inhibition was observed following transfection with ZEB1 3'-UTR MUT under the same conditions (see *Figure 4B*). An evident dip in ZEB1 levels due to miR-150-5p was revealed by qRT-PCR (see *Figure 4C*), which indicates the repressive effect of the miRNA on ZEB1 expression. Next, rescue assays were conducted to examine the function of the IGBP1-AS1/miR-150-5p axis in NPC. qRT-PCR was employed to examine ZEB1 expression following the transfection of SUNE-1 and C666-1 using siIGBP1-AS1 only, or siIGBP1-AS1 and the miR-150-5p-inhibitor (see *Figure 4D*), and the results showed that IGBP1-AS1 increased ZEB1 expression by miR-150-5p.

Finally, a lowered expression profile of miR-150-5p was detected in all the NPC lines used in this study compared to that of the NP69 control (see *Figure 4E*). The cancer tissues displayed an evidently lower miR-150-5p pattern compared to those of the surrounding normal samples (see *Figure 4F*). The Pearson correlation analysis revealed a negative correlation between IGBP1-AS1 and miR-150-5p in NPC tissues ( $P < 0.001$ ; see *Figure 4G*). Together, these results



**Figure 2** The growth and metastasis of NPC cells are promoted by IGBP1-AS1. (A) Knockdown efficiency assessment by qRT-PCR in the SUNE-1 and C666-1 cell lines after siIGBP1-AS1 transfection. GAPDH was used as the internal control. (B) MTT assay for cell viability after transfecting SUNE-1 and C666-1 cells with siIGBP1-AS1. \*,  $P < 0.05$ ; \*\*\*,  $P < 0.001$ . NC, negative control; OD, optical density; NPC, nasopharyngeal carcinoma; qRT-PCR, quantitative reverse transcription PCR.

suggest that IGBP1-AS1 facilitates the malignancy of NPC via the miR-150-5p/ZEB1 axis.

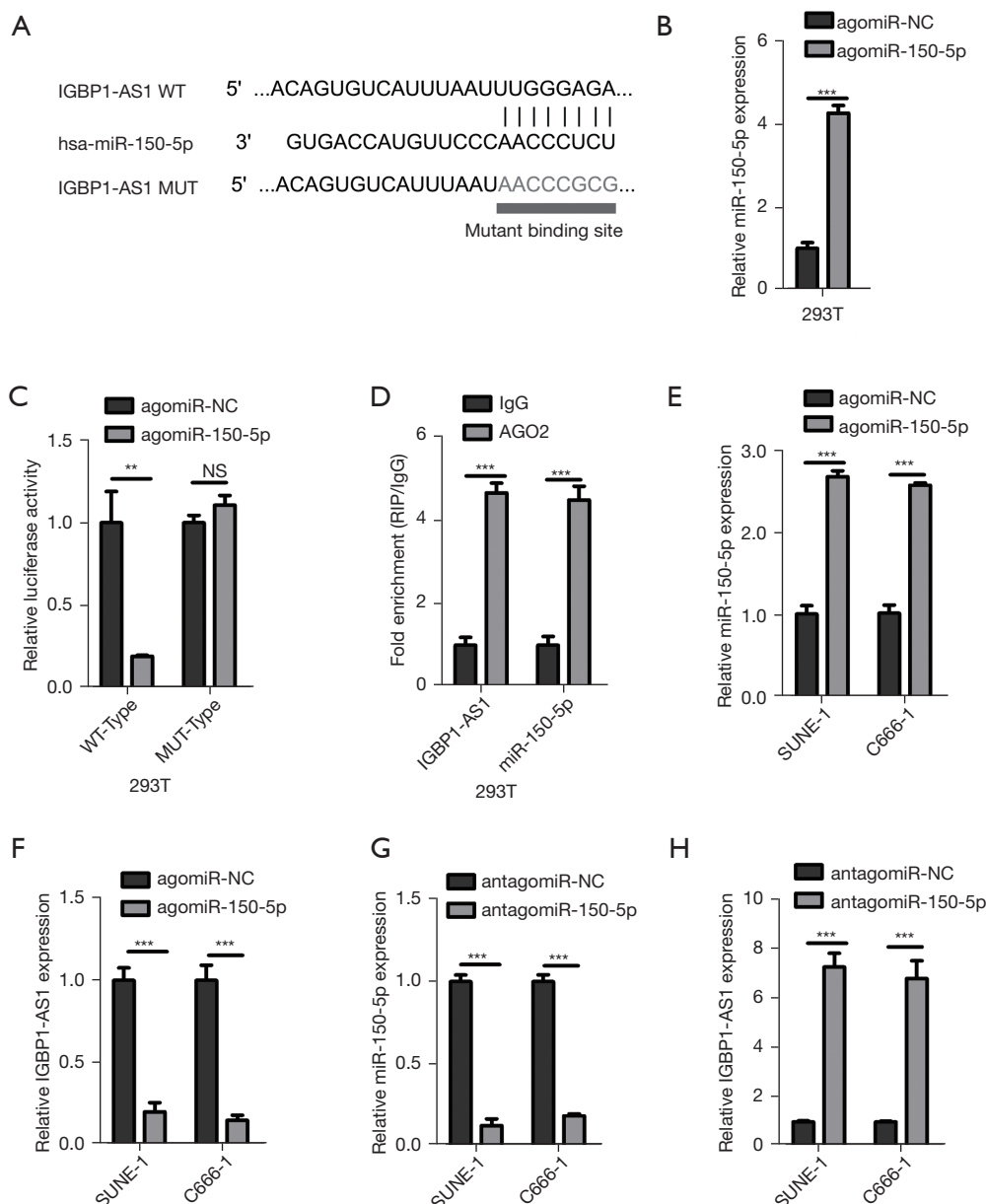
## Discussion

The dysregulation of several lncRNAs has been observed in NPC (10,11). These lncRNAs could also regulate NPC malignancy progression by acting as cancer oncogenes or suppressors (10,11). The close association between lncRNA IGBP1-AS1 and the malignancy of breast cancer has been highlighted (9). However, the involvement of IGBP1-AS1 in human NPC progression has yet to be proven. This study found that IGBP1-AS1 was more upregulated in NPC cell lines and clinical samples than relevant control samples. The *in-vitro* silencing of IGBP1-AS1 suppressed the ability of NPC cells to proliferate. Additionally, IGBP1-AS1 was found to bind to miR-150-5p and positively regulate the expression of ZEB1 (the functional miR-150-5p target). Finally, we showed that IGBP1-AS1 participated in NPC progression via miR-150-5p/ZEB1 regulation, and thus showed the oncogenic potential of IGBP1-AS1 in NPC.

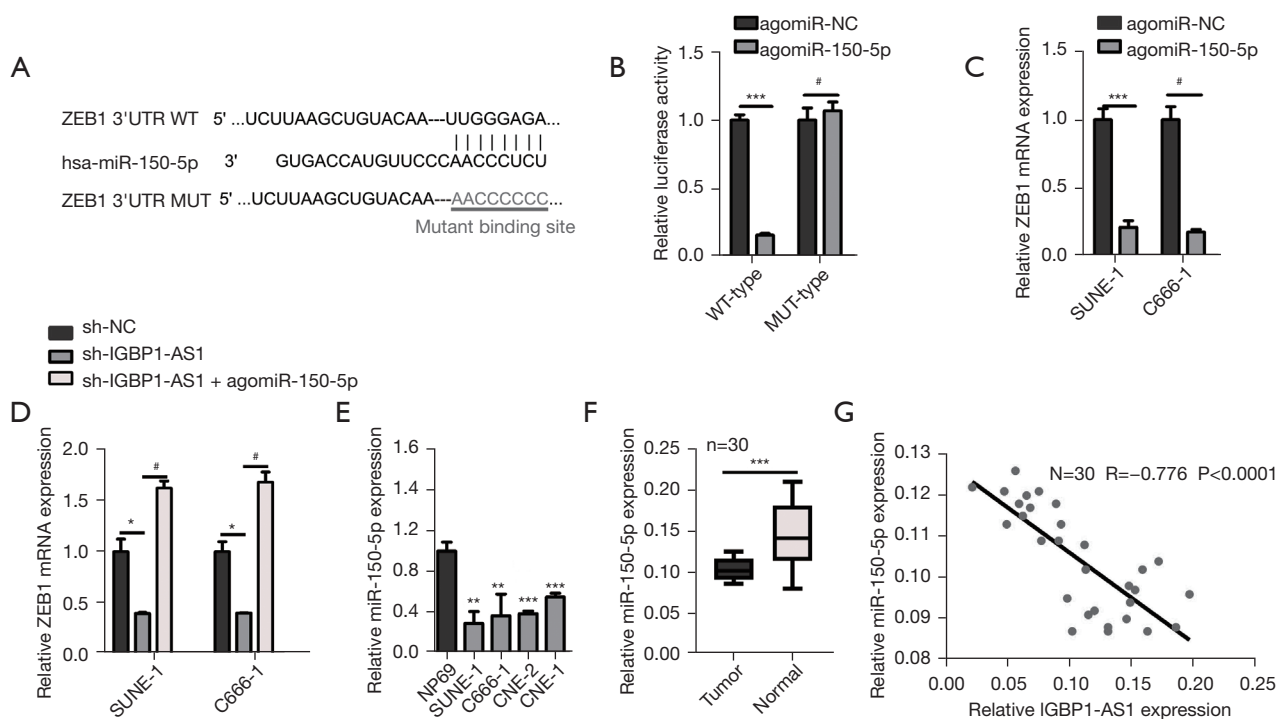
lncRNAs act as miRNA quenchers to control tumorigenesis (12-18). For example, SNHG7 facilitates angiogenesis and tumorigenicity by sequestering miR-216b in colorectal cancer (CRC) (19). lncRNA OIP5-AS1 sequesters miR-369-3p to influence the radio-resistance of CRC cell lines (20). We also examined the binding of IGBP1-AS1 to miR-150-5p, and found an inverse relationship between the 2 in NPC tissues. Additionally,

while the transfection of miR-150-5p mimic (agomiR-150-5p) caused a decline in IGBP1-AS1 levels, that of antagomiR-150-5p (an inhibitor) boosted the expression of IGBP1-AS1. The reporter assay for luciferase activity further revealed that IGBP1-AS1 may interact with miR-150-5p. miR-150-5p has been found to be aberrantly expressed in several tumors (21,22). Zhang *et al.* (23) reported that the targeting of PDCD4 by miR-150, the levels of which are elevated in cervical cancer, augmented the ability of these cells to divide, invade, and migrate. Non-small cell lung cancer cells display an upregulation of miR-150 that boost cancer cell survival and tumorigenesis (24). Recently, targeting of VEGFA by miR-150-5p in CRC has also been shown to hinder tumorigenesis (25).

This study identified ZEB1 as a novel miR-150-5p targeting NPC cell lines. ZEB1 has been found to be involved in epithelial-mesenchymal transition (EMT), cancer growth, and invasion, and have an elevated expression in many tumors (26). Recently, the control of the miRNA/ZEB1 axis by lncRNAs has been revealed in human cancers. For example, ZEB1 was found to be augmented in hepatocellular carcinoma (HCC) by the lncRNA ZFAS1, which abrogated the effects of miR-150 (27). The miR-101-3p/ZEB1 axis is targeted by the lncRNA PTAR, which increases the malignancy and EMT of serous ovarian cancer cells (28). We showed that the *in-vitro* sequestering of miR-150-5p by IGBP1-AS1 elevated the levels of ZEB1 in NPC. Further, our rescue experiments indicated that IGBP1-AS1 appears to be involved in the malignancy of NPC. The



**Figure 3** LncRNA IGBP1-AS1 binds with miR-150-5p in NPC. (A) The binding sites between miR-150-5p and WT-IGBP1-AS1 (the IGBP1-AS1 WT) or MUT-IGBP1-AS1 (the IGBP1-AS1 MUT) are shown. (B) The miR150-5p expression was determined by qRT-PCR in 293T. (C) Dual-luciferase reporter assays in 293T cells transfected with miR-150-5p plus WT-IGBP1-AS1, or MUT-IGBP1-AS1. (D) Precipitation of ZEB1, and the miR-150-5p complex in AGO2. qRT-PCR for miR-150-5p and ZEB1. (E-H) qRT-PCR for miR-150-5p and LNCRNA IGBP1-AS1 after transfection of SUNE-1 and C666-1 with miR-150-5p mimic or the miR-150-5p inhibitor. U6 and GAPDH were the controls. agomiR-NC: mimic NC; agomiR-150-5p: miR-150-5p mimic; antagomiR-NC: inhibitor NC; antagomiR-150-5p: miR-150-5p inhibitor. \*\*,  $P < 0.01$ ; \*\*\*,  $P < 0.001$ ; NS  $P > 0.05$ . WT, wild-type; MUT, mutant; NC, negative control; lncRNA, long non-coding RNA; NPC, nasopharyngeal carcinoma; ZEB1, zinc finger e-box binding homeobox 1; qRT-PCR, quantitative reverse transcription PCR.



**Figure 4** NPC cell aggressiveness is regulated by IGBP1-AS1 via the miR-150-5p/ZEB1 axis. (A) The binding sites between miR-150-5p and WT-ZEB1 (the ZEB1 WT) or MUT-ZEB1 (the ZEB1 MUT) are shown. (B) Luciferase reporter assay of 293T cells co-transfected with ZEB1 3'-UTR WT or ZEB1 3'-UTR MUT and miR-150-5p mimics. (C) qRT-PCR to determine the ZEB1 levels in miR-150-5p mimics or NC mimics-transfected SUNE-1 and C666-1 cells. (D) qRT-PCR for ZEB1 levels in SUNE-1 and C666-1. (E) qRT-PCR estimation of miR-150-5p levels in NPC cell lines and NP69 cells. (F) qRT-PCR assay for miR-150-5p in clinical NPC and equivalent normal tissues (n=30). (G) Pearson correlation analysis of IGBP1-AS1 and miR-150-5p in NPC samples. (D) \*: sh-NC vs. sh-IGBP1-AS1; # to sh-IGBP1-AS1 vs. sh-IGBP1-AS1 + antagonir groups. \*, P<0.05; \*\*, P<0.01; \*\*\*, P<0.001; #, P<0.05. WT, wild-type; MUT, mutant; NC, negative control; NPC, nasopharyngeal carcinoma; ZEB1, zinc finger e-box binding homeobox 1; 3'-UTR, 3'-untranslated region; qRT-PCR, quantitative reverse transcription PCR.

study still has some shortcomings, such as further studies will focus on applying more multiple vitro methods and vivo experiments for vivifying IGBT1-AS1 on NPC cell proliferation; meanwhile we will focus on whether lncRNA IGBT1-AS1 alter stem cell and radiosensitivity in NPC; and the signaling pathway involving IGBT1-AS1 in NPC cell.

This study showed that IGBP1-AS1 (a lncRNA) is overexpressed in NPC. The molecule sequesters miR-150-5p and boosts the level of ZEB1 to facilitate NPC tumorigenesis. These findings open up new avenues for research in NPC oncogenesis, especially with regards to IGBP1-AS1.

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## Footnote

*Reporting Checklist:* The authors have completed the MDAR reporting checklist. Available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-291/rc>

*Data Sharing Statement:* Available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-291/dss>

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <https://tcr.amegroups.com/article/view/10.21037/tcr-22-291/coif>). The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related

to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study involving human participants were in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by committee board of Shijiazhuang People's Hospital and informed consent was taken from all the patients.

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