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HSA Circ 0005273 Promotes Breast Cancer Tumorigenesis by Controlling the YAP1-Hippo Signalling Pathway

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Introduction

Although the long non-coding RNA (LncRNA) LINC00649 has been linked to acute myeloid leukaemia (AML), prostate cancer and colorectal cancer, its role in regulating other types of cancer, such as gastric cancer (GC), has not been investigated. This study examined the expression status of LINC00649 in GC tissues and cells using Real-Time qPCR and we discovered that LINC00649 was enriched in cancerous tissues and cells but not in their normal counterparts, which was supported by data from the TCGA dataset. Then, using gain- and loss-of-function experiments, we discovered that LINC00649 acted as an oncogene, accelerating GC cell proliferation, migration and epithelial-mesenchymal transition (EMT) in vitro and promoting tumorigenesis in vivo [1].

Description

The extensive transcription of RNA from non-protein-coding regions of the genome was one of the most unexpected discoveries in the genomics era of biology. In mammalian cells, tens of thousands of long noncoding RNAs (lncRNAs) have been identified, which are defined as transcripts longer than 200 nt with no or low protein-coding potential. Pioneering research on a small subset of lncRNAs revealed that lncRNAs are an essential component of the cellular control network, coexisting with proteins and playing important roles in cancer. Mechanistically, lncRNAs have two inherent functional properties: (1) sequence-mediated interaction with genomic DNA or other RNA and (2) secondary/tertiary structure-mediated interaction with RNA-binding proteins. All tissue samples (PTC tissues, n=92; corresponding normal tissues, n=92) used in this study were obtained from Jiangsu Normal University's Key Laboratory for Biotechnology on Medicinal Plants. Patients who were identified as having PTC through pathological examination were eligible for this study, as were those who had not received radiotherapy or chemotherapy prior to surgery. The Ethics Committee of Jiangsu Normal University approved the study. All participants had provided written informed consent. As soon as the samples were collected from patients, they were snap frozen in liquid nitrogen and kept at 80°C [2,3].

107 Hep-2 stable cells were injected subcutaneously into the right flanks of 4-week-old Balb/c nude mice to detect tumour growth. Every three days, tumour growth was monitored. 30 days after inoculation, tumour weights were calculated. Stable Hep-2 cells were intravenously injected (2106 cells per mouse) into the tail vein of mice to detect tumour metastasis. After 50 days, the mice were sacrificed and their lung tissues were extracted and fixed in Bolin's fluid. The tissues were then histologically examined for the presence of micrometastases using H&E staining. The Animal Experimentation Ethics Committee of The First Affiliated Hospital of Zhengzhou University Hospital approved all animal handling and experimental procedures [4].

Many lncRNAs have been shown to act as competing endogenous RNAs (ceRNAs) by competitively binding miRNAs. We investigated whether miRNAs play a role in the underlying mechanism of SNHG1 in LSCC progression. A bioinformatics analysis revealed that SNHG1 contains a miR-375 binding site. miR-375 is frequently downregulated in cancer and functions as a tumour suppressor. We used a RIP assay with MS2-binding protein (MS2bp), which specifically binds target RNA containing MS2-binding sequences, to validate the direct interaction between SNHG1 and miR-375 (MS2bs). We created a construct with SNHG1 transcripts and MS2bs elements and cotransfected it into LSCC cells with a GFP-MS2bp construct [5].

Conclusion

In conclusion, we discovered that hsa circ 0005273 is highly expressed and functions as an oncogene in BC. Furthermore, hsa circ 0005273 may regulate YAPl by acting as a sponge for miR-200a-3p, inactivating the Hippo signalling pathway. The hsa circ 0005273/miR-200a-3p/YAP1/Hippo pathway axis could be a new biomarker and therapeutic target for BC.

Acknowledgement

None.

Conflict of Interest

There are no conflicts of interest by author.

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