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A Systematic Review of the Expression Pattern of Long Non-Coding RNAs SNHG5, SNHG17, and SNHG26 in Human Solid Tumors

Elham Aminifar [1], Hassan Mehrad-Majd[2,3]*, Yalda Ravanshad[4], Amirhossein Sahebkar[5]

[1] *Student Research Committee, Islamic Azad University, Mashhad Branch, Mashhad, Iran*
[2] *Cancer Molecular Pathology Research Center, Mashhad University of Medical Sciences, Mashhad, Iran*
[3] *Clinical Research Development Unit, Ghaem Hospital, Mashhad University of Medical Sciences, Mashhad, Iran.*
[4] *Department of Community Medicine, Mashhad Medical Science Branch, Islamic Azad University, Mashhad, Iran*
[5] *Biotechnology Research Center, Pharmaceutical Technology Institute, Mashhad University of Medical Sciences, Mashhad, Iran*

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ABSTRACT

Introduction: Small nucleolar RNA host genes (SNHGs) are a class of long non-coding RNAs that have been increasingly associated with various cancers, and are emerging as key players in cancer progression. This systematic review aimed to analyze the expression patterns and potential roles of SNHG5, SNHG17, and SNHG26 in various solid tumors.

Methods: A comprehensive literature search was conducted across databases, including PubMed, Cochrane, and Google Scholar, to identify relevant studies. Initially, retrieved papers were screened based on titles and abstracts for relevance, with exclusion criteria applied to eliminate duplicate publications, case reports, conference abstracts, and irrelevant studies.

Results: A total of 21 papers met the criteria for this systematic review, focusing on SNHG5 (n=5), SNHG17 (n=7), and SNHG26 (n=9). These studies consistently highlighted the upregulation of SNHG5, SNHG17, and SNHG26 in various cancer types, emphasizing their potential as prognostic and therapeutic targets in solid tumors.

Conclusion: The findings suggest a consistent overexpression of SNHG5, SNHG17, and SNHG26 in cancerous tissues, supporting their potential as prognostic markers in solid tumors. Further investigations should consider the regulatory mechanisms of SNHGs and explore their therapeutic implications in cancer management. This review highlights the importance of understanding the roles of SNHGs in cancer progression and calls for continued research efforts.

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Introduction

One of the most prominent issues in recent years is cancer and its excessively negative effects on societies and their economies (1-3). Therefore, each new revelation of its molecular setting should be carefully assessed for its potential as biomarker and/or restorative target (1, 4). The carcinogenic effects of genes, after many examinations, have shown that they are primarily employed by transcription and protein encoding of genes (5-7). Nonetheless, most recent examinations have revealed that less than 2% of the human genome consists of coding genes, and over 90% comprises of noncoding genes that function as regulatory factors in most systems (8). Moreover, according to recent studies, the non-protein-coding portion of the genome is of pivotal functional significance for disease occurrence (9). The non-coding RNAs (ncRNAs) are characterized by three types: long ncRNAs, mid-size ncRNAs, and short ncRNAs (10). Long noncoding RNAs (lncRNAs), which are longer than 200 nucleotides, are among the key factors in the development of tumors (Figure 1) (11-13). LncRNAs can directly or indirectly associate with target genes at the transcriptional level (14). Aberrant expression of lncRNAs has been associated with cancers (3). The small nucleolar RNA host gene (SNHG) family is a subgroup of lncRNAs that are regarded as dysregulated in many human cancers (15). Furthermore, SNHGs is

***Corresponding author**: **Hassan Mehrad-Majd,** PhD in Molecular Medicine, Clinical Research development Unit, Ghaem Hospital, Faculty of Medicine, Mashhad University of Medical Sciences, P.O. Box: 9176699199, Mashhad, Iran.*
E-mail: Mehradmajd.h@gmail.com Mehradmajdh@mums.ac.ir
Tel: 098 51 38012694 Fax: 098 511 8828574
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improving cell proliferation, cell cycle progression, invasion and metastasis of urological cancer cells (16).

There are many instances such as, SNHG1, SNHG2/GAS5, SNHG3, SNHG4, SNHG5, SNHG6, SNHG7, SNHG8, SNHG9, SNHG10, SNHG11, SNHG12, SNHG13/DANCR, SNHG15, SNHG17, SNHG20 and SNHG28, SNHG26 that are newly recognized lncRNAs, which are considered oncogenes in several tumors (e.g., liver cancer, renal cancer, breast cancer, glioma, and gastrointestinal cancer) (17-19). Many studies have shown that lncRNA SNHGs impacts many signaling pathways to promote tumor cell growth, migration, and metastasis (18).

As few studies have explored the expression and clinical implications of SNHGs in solid tumors, we systemically determined the role of SNHG5, SNHG17, and SNHG26 expression in patients with solid tumors.

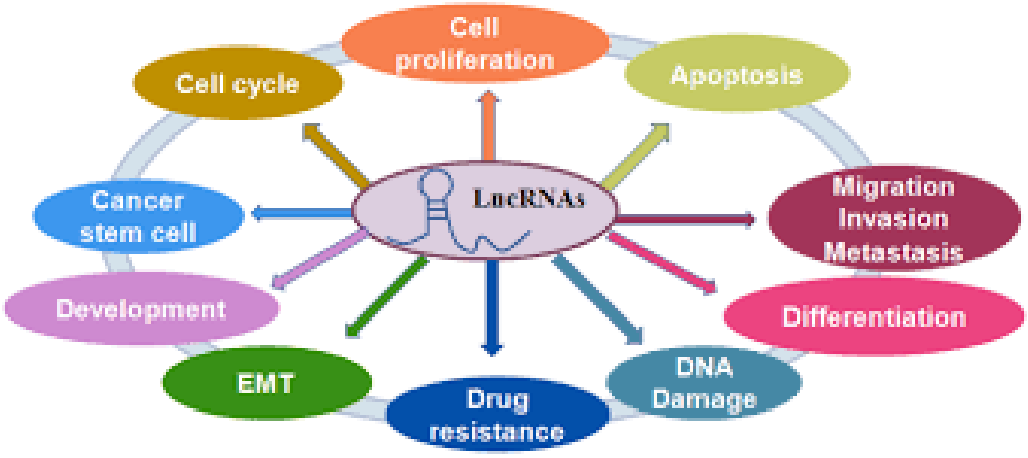

Figure 1. lncRNAs Function in controlling cellular activities (38)

Material and Methods

Search Strategy and Study Selection

A search of PubMed, Cochrane, and Google Scholar, was executed to pinpoint all related published studies about lncRNA SNHGs, as a prognostic factor for survival of patients with cancer, up to October 2024. The search terms included ("LncRNA-SNHG" OR "SNHG5" OR "SNHG17" OR "SNHG26" OR "lncRNA" OR "long noncoding RNA") AND ("cancer" OR "carcinoma" OR "neoplasm" OR "prognosis" OR "prognostic" OR "survival" or "recurrence" OR "tumor") with English language restrictions. Our search criteria were developed with an additional manual search of reference lists of all searched articles. The titles and abstracts of all citations were screened independently and separately by two authors (EA and HMM). To find more relevant publications, we did not consider abstracts or unpublished reports. In cases multiple studies were conducted by the same author using the same case series, we included only the most recent or complete study. The exclusion criteria for this search included single case reports. Studies that met the following criteria were considered eligible: (1) studies on any type of human tumor; (2) the tissue expression of lncRNA SNHGs was determined; (3) any study investigating the relationship between lncRNA SNHG expression and features of solid tumors or clinicopathological features of cancers. In addition, studies that failed to meet the inclusion criteria, duplicate publications, letters, case reports, editorials, and comments were excluded.

Extraction of Data and Quality of Studies Assessment

Two independent examiners, EA and HMM, assessed the quality of the studies using the Newcastle-Ottawa Scale (NOS). Any discrepancies in their evaluations were reconciled with the involvement of a third examiner, AS. The data extracted from each study included the last name of the first author, the year of publication, the title of the study, and the conclusions drawn from the research.

Results

In our initial search, 250 studies were identified. After a synchronous review of the titles and abstracts, 219 studies that were either unrelated or duplicates were excluded. Additionally, 10 relevant studies were excluded because they were published as case reports, or conference abstracts. Following a detailed examination of the remaining abstracts against our inclusion and exclusion criteria, 21 papers were deemed qualified for the systematic review.

The distribution of these articles across different SNHGs is as follows:

- **SNHG5:** 5 articles (Table 1)
- **SNHG17:** 7 articles (Table 2)
- **SNHG26:** 9 articles (Table 3)

Each table provides detailed insights into how these specific SNHGs are implicated in solid tumors, highlighting their potential roles and mechanisms.

Our results suggest that the overexpression of SNHG5, SNHG26, and SNHG17 correlates with proliferation and metastatic in liver cancer (hepatocellular carcinoma), colorectal cancer, lung cancer, breast cancer, bladder cancer, esophageal squamous cell carcinoma, prostate cancer, pancreatic cancer, non-small cell lung cancer, ovarian cancer, glioma, tongue squamous cell carcinoma, head and neck squamous cell carcinoma (HNSC), and gastric cancer.

Discussion

To the best of our knowledge, this research is the first review on SNHG5, SNHG26, and SNHG17 in solid tumors. In following paragraphs, the findings of mentioned SNHGs will be discussed in detail.

SNHG5

In the current study, the crucial role of SNHG5 in cancer cell properties was revealed (Table1).

Table 1. Studies investigating the role of SNHG5 in solid tumors

Author(s)	Year	SNHG Type	Article Title	Conclusion	NOS
Xiao, X., et al.(22)	2022	SNHG5	Tobacco Nicotine Promote TRAIL Resistance in Lung Cancer by SNHG5	SNHG5 overexpression promote TRAIL resistance in Lung Cancer.	6
Li, Y., et al(20).	2020	SNHG5	LncRNA SNHG5 promotes the proliferation and cancer stem cell-like properties of hepatocellular carcinoma by regulating UPF1 and Wnt signaling pathway	Overexpression of SNHG5 in GC tissues is related to poor overall survival and have a function in gastric cancer development.	6
Chi, J.-R., et al.(23)	2019	SNHG5	SNHG5 promotes breast cancer proliferation by sponging the miR-154-5p/PCNA axis	Upregulation of SNHG5 correlate with breast cancer proliferation.	6
Turgeon ,M.-O., et al.(24)	2018	SNHG5	DNA damage, repair, and cancer metabolism	Overexpression of SNHG5 increases proliferation of cancer cells.	6

A noteworthy impact on the proliferation and self-renewal capacity of liver cancer stem-cells (CSCs) was performed by SNHG5 alterations (knockdown or upregulation). Similarly, the expression of stem cell markers and stem factors plummeted after downregulation of SNHG5 (20). In short, Li et al. concluded that SNHG5 promoted HCC cell proliferation *in vitro* and *in vivo* and was responsible for the sphere formation of liver CSCs and their properties. The underlying function of SNHG5 in encouraging the proliferation and CSC-like properties of HCC was through regulating UPF1 and activation of the Wnt-signaling pathway. Our most recent data points out that SNHG5, along with its downstream mechanism and pathways, could give an updated understanding to a new potential therapeutic targets against liver CSCs (20). Zhang et al. (21) revealed that SNHG5 could promote proliferation, metastasis, and migration of colorectal cancer cells by modulating miR-132-3p/CREB5 signaling. In addition, revealed the SNHG5 expression was rapidly increasing in lung cancer tissues of the smokers than that of the nonsmokers (22). Although no significant connection was observed between the SNHG5 expression level and patients age, gender or the lung cancer tissue differentiation level, it had relations with tumor node metastasis stage (22). In SNHG5, overexpression enhances the ability of breast cancer cells to proliferate and go through cell cycle by releasing proliferating cell nuclear antigen (PCNA) from the inhibition of miR-154-5p (23). In bladder cancer, SNHG5 induces p27 silencing, followed by enhanced proliferation rate and cell cycle progression, which is associated with apoptosis inhibition (24).

SNHG17

Our results indicate that SNHG17 is involved in the progression of cancers (Table 2).

The results showed that SNHG17, a newly discovered lncRNA, is significantly upregulated in ESCC tissue compared to adjacent normal tissue (25). Correlation analysis of clinical characteristics and SNHG17 expression in ESCC illustrated that ESCC Patient Grade was associated with SNHG17 expression (25). Li et al. found that SNHG17 was upregulated in prostate cancer (PC) tumor specimens compared with benign PC tissues and adjacent normal PC tissues. Furthermore, the discovery of SNHG17 expression led to an understanding of its elevation in PC tumor tissues with advanced tumor stages and high Gleason's scores (17). More revelations regarding the results

indicated significant apoptosis in PC (pancreatic carcinoma) cells transfected with SNHG17 (26). Experiments performed by Zhao et al. revealed that lncRNA SNHG17 expression is higher in PC specimens than in normal healthy pancreatic tissue. They discovered that SNHG17 is crucial for maintaining the viability, invasion, and migration of PC cells and can prevent apoptosis by competitively interacting with miR-942, which subsequently upregulates the expression of downstream oncogenes (26). Moreover, what is essential for cancer cell proliferation and migration in NSCLC is [4] modulation of FOXA1 and BIK activities by SNHG17. Wu et al. showed that SNHG17 could be utilized to observe prostate cancer progression (27), while Pan et al. reported that SNHG17 may be an oncogene that arbitrate CDK6 expression in ovarian cancer (28).

In human glioma specimens and glioma cell lines, the expression level of SNHG17 is heightened. SNHG17 knockdown attenuates cellular proliferation, increases apoptosis, and represses tumor growth. The above-mentioned lncRNA is also upregulated by STAT3 in ovarian cancer, where a positive link between high SNHG17 expression and poor prognosis has been confirmed. Notably, functional analysis has shown that SNHG17 inhibits ovarian cancer growth (28).

Table 2. Studies investigating the role of SNHG17 in solid tumors

Author(s)	Year	SNHG Type	Article Title	Conclusion	NOS
Li, J., et al.(17)	2022	SNHG17	Integrative analysis and experimental validation indicated that SNHG17 is a prognostic marker in prostate cancer and a modulator of the tumor microenvironment via a competitive endogenous RNA regulatory network	SNHG17 can be prognostic factor related to progression, immunosuppression of prostate cancer.	6
Chen, W., et al.(25)	2021	SNHG17	LncRNA SNHG17 regulates cell proliferation and invasion by targeting miR-338-3p/SOX4 axis in esophageal squamous cell carcinoma	SNHG17 in esophageal squamous cell carcinoma can be a potential therapeutic target.	6
Zhao, L., et al.(26)	2021	SNHG17	lncRNA SNHG17 promotes pancreatic carcinoma progression via cross- talking with miR-942	SNHG17 can be a potential therapeutic target for pancreatic carcinoma.	6
Wu, G., et al.(27)	2020	SNHG17	LncRNA SNHG17 aggravated prostate cancer progression through regulating its homolog SNORA71B via a positive feedback loop	Upregulation of SNHG17 promote proliferation, invasion, and migration in prostate cancer.	6
Pan, X., et al.(28)	2020	SNHG17	STAT3-induced lncRNA SNHG17 exerts oncogenic effects on ovarian cancer through regulating CDK6	SNHG17 is a novel target for ovarian cancer	6
Wroblewska J.P., et al.(6)	2019	SNHG17	SF3B1, NRAS, KIT, and BRAF mutation; CD117 and cMYC expression; and tumoral pigmentation in sinonasal melanomas: an analysis with newly found molecular alterations and some population-based molecular differences	SNHG17 overexpression is related to growth of ovarian cancer cells.	6
Youssef, O., et al.(7)	2018	SNHG17	Expression of plasma miRNA-221 in colorectal carcinoma patients and its diagnostic significance in comparison with p53 expression	SNHG17 knockdown attenuates cellular proliferation, raises apoptosis, and represses tumor growth.	6

Table 3. Studies investigating the role of SNHG26 in solid tumors

Author(s)	Year	Type	Article Title	Conclusion	NOS
Wu et al.(19)	2024	SNHG26	LncRNA SNHG26 promotes gastric cancer progression and metastasis by inducing c-Myc protein translation and an energy metabolism positive feedback loop.	SNHG26 levels were increased in Gastric Cancer tissue.	6
Jiang et al. (33)	2022	SNHG26	lncRNA SNHG26 promoted the growth, metastasis, and cisplatin resistance of tongue squamous cell carcinoma through PGK1/Akt/mTOR signal pathway.	SNHG26 is relate to the growth and prognosis of tongue squamous cell carcinoma (TSCC).	6

Wang et al. (31)	2021	SNHG26	Identification and validation of a four-long non-coding RNA signature associated with immune infiltration and prognosis in colon cancer.	SNHG26 expression is significantly increased in colon cancer.	6
Samdal et al.(32)	2021	SNHG26	The G2-phase enriched lncRNA SNHG26 is necessary for proper cell cycle progression and proliferation.	SNHG26 is necessary for a normal G2/M progression.	6
Wu et al. (35)	2021	SNHG26	Ellagic acid resensitizes gemcitabine-resistant bladder cancer cells by inhibiting epithelial-mesenchymal transition and gemcitabine transporters.	SNHG26 increase cisplatin resistance.	6
Bao et al. (30)	2017	SNHG26	A potential prognostic lncRNA signature for predicting survival in patients with bladder urothelial carcinoma	SNHG26 upregulated in bladder urothelial carcinoma and associated with poor survival	6

SNHG26

SNHG26, which is identified as playing a role in carcinogenesis and clinical outcomes in several cancers, is one of the small nucleolar RNA host genes. Previous studies have shown that SNHG26 is dysregulated in various cancers, including bladder, lung, and colon cancer, and it affects the cell cycle and proliferation (29-32).

Long noncoding RNA SNHG26 is closely related to the growth and prognosis of tongue squamous cell carcinoma (TSCC), it facilitates tumor cell growth, migration, and resistance to cisplatin via the PGK1/Akt/mTOR signaling pathway (33). Herein, in several discoveries have showed that SNHG26 did not impact apoptosis and autophagy of TSCC cells. SNHG26 could indirectly increase the resistance of tongue cancer by enhancing its proliferative capacity. Recent studies indicate that the emergence of acquired drug resistance in tumor cells co-occurs with EMT-like changes (34-37).

Jiang et al. found high SNHG26 expression in HNSC and unveiled notably higher SNHG26 expression in cancer tissues compared with that of the adjacent normal tissues. These findings illustrate the significant diagnostic potential of SNHG26 for HNSC patients. Their analysis showed that the survival chances of patients in the high SNHG26 expression group were higher than those in the low SNHG26 expression group. Overall, they found that patients expressing high SNHG26 levels were strongly correlated with poor prognosis compared to those expressing low SNHG26 levels (33).

Wu et al. demonstrated that SNHG26 levels were dramatically increased in GC tissue compared to those in adjacent tissue and revealed that higher expression of SNHG26 led to shorter disease-free survival times and overall survival times. Moreover, gastric cancer tumors exhibiting SNHG26 overexpression were significantly larger than those in the control group, suggesting that SNHG26 may facilitate tumor growth. Collectively, these findings indicate that SNHG26 enhances the proliferation and metastasis of gastric cancer (19).

Limitations

It is noteworthy to state that our study faced some limitations. First and foremost, the sample size and the number of studies included in the review were relatively small, which could have influenced the results. Secondly, selection bias may be a factor in the exclusion of studies due to language restrictions. Thirdly, a bias may also exist in favor of published articles with positive results.

Conclusion

We believe that the general activity of SNHGs in invasive disease is related to the stimulation of the following malignant processes. Through our literature search, we found that SNHGs are increasingly becoming essential for molecular research in cancer due to their versatility and function in the majority of cancers. One clear observation in recent years is that the data on these non-coding RNAs are copious, and the validation of their role in the progression and severity of malignant diseases has been established. However, a common oversight regarding these lncRNAs is still exists, as well as a need for a better understanding of their influence at the molecular level. The majority of SNHGs (SNHG26, SNHG5, and SNHG17) function by suppressing solid tumor microRNAs, allowing oncogene transcripts to be expressed. One cause of epigenetic alterations in the genome is that these SNHGs can also initiate transcription through interaction with transcription factors. Additionally, These SNHGs activate signaling pathways.

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