

The role of long noncoding RNA SNHG7 in human cancers (Review)

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Abstract. Long non-coding RNAs (lncRNAs) have been demonstrated to serve important roles in a variety of human tumor types. The lncRNA small nucleolar RNA host gene 7 (SNHG7) is associated with a variety of cancer types, such as esophageal cancer, breast cancer and gastric neoplasia. Based on previous studies that examined SNHG7 expression in tumors, it has become clear that SNHG7 modulates tumorigenesis and cancer progression by acting as a competing endogenous RNA. SNHG7 can sponge tumor-suppressive microRNAs and regulate downstream signaling pathways. In addition, overexpression of SNHG7 is associated with the clinical characteristics of patients with cancer by regulating cellular proliferation, invasion and metastasis and by inhibiting apoptosis via a variety of mechanisms of action. The function of SNHG7 in tumorigenesis and cancer progression indicates that it can potentially act as a novel therapeutic target or a diagnostic biomarker for cancer therapy or detection, respectively.

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1. Introduction

Recently, several reports have been published regarding current global epidemiology of several cancers. It has been suggested that cancer is a major health and economical problem, which requires effective measures. Various studies have focused on the potential molecular mechanisms of human tumors in the past few years. Accumulating evidence indicates that non-coding RNAs (ncRNAs) lack the ability, or possess limited ability to encode proteins. However, they play important roles in the molecular regulation of cancers by affecting specific biological processes, including translation, transcription and epigenetic regulation (1-5). According to their length, ncRNAs can be divided into different types, such as microRNAs (miRNAs), circular RNAs, nucleolar RNAs and long non-coding RNAs (lncRNAs) (6-9).

lncRNAs are transcribed by RNA polymerase II and are longer than 200 nucleotides in length (10,11). Accumulating evidence indicates that lncRNAs regulate the molecular biological processes of cancer at the level of translation and transcription. At the epigenetic level, they also regulate cancer progression by transcriptional interference, chromosome reconstruction, gene imprinting, histone modification, DNA methylation and cell cycle modulation. Moreover, the expression of certain lncRNAs is specific in cells and tissues. Therefore, it is very important to analyze the function of lncRNAs and their potential role in cancer diagnosis, treatment and prognosis (12-18). Despite several recent studies on the contribution of lncRNAs in tumor progression, their exact functions and molecular mechanisms remain unclear.

The lncRNA small nucleolar RNA host gene 7 (SNHG7) is a novel oncogenic lncRNA, which is abnormally expressed in various cancer types and is associated with the expression of several oncogenic genes. The current review presents a comprehensive summary of data reported from previous studies regarding the expression, function, mechanism and clinical features of SNHG7 with regard to tumorigenesis and cancer progression.

2. Characterization of SNHG7

SNHG7 is a novel oncogenic lncRNA located on chromosome 9q34.3, which is 2,176 bp long (19). Recently, several reports

have demonstrated that the expression of SNHG7 is significantly increased in tumors and cancer cells, including those of colorectal, breast, prostate and gastric origin. Accumulating evidence has shown that SNHG7 can promote proliferation, invasion, and migration of various cancer types, as well as inhibit the induction of apoptosis (20-22). In addition, SNHG7 overexpression correlates with lower patient survival and worse clinical features. SNHG7 may function as a novel diagnostic and prognostic biomarker and as a valuable therapeutic target for cancer treatment.

3. SNHG7 dysregulation in human cancers

SNHG7 in lung cancer. In 2016, She *et al* (23) demonstrated that SNHG7 expression was significantly increased in lung cancer tissues compared with the expression noted in adjacent normal tissues. SNHG7 expression was higher in three human lung cancer cell lines compared with its expression in human bronchial epithelial cells. Silencing of SNHG7 expression inhibited lung cancer cell proliferation, migration and invasion and induced cancer cell apoptosis. Moreover, the expression levels of FAIM2 were upregulated in lung cancer tissues and correlated positively with SNHG7 expression. Knockdown of FAIM2 expression inhibited lung cancer cell proliferation, migration and invasion and induced apoptosis. Therefore, these studies suggested that SNHG7 promoted lung cancer cell proliferation, migration and invasion and inhibited apoptosis by upregulating FAIM2 expression. In 2017, She *et al* (24) demonstrated that SNHG7 was overexpressed in non-small cell lung cancer (NSCLC), whereas the patients with high SNHG7 expression correlated positively with advanced TNM stage and lymph node metastasis and therefore with worse clinical outcomes. SNHG7 interacted with miR193b and exhibited a negative correlation with miR193b. SNHG7 acted as competing endogenous RNA (ceRNA) to sponge miR193b. The expression of FAIM2 correlated negatively with miR193b and its function was antagonized by miR193b in NSCLC cells. Overall, these studies suggested that SNHG7 sponged miR193b to regulate FAIM2 expression and promote tumor growth and metastasis. In 2019, Chen *et al* (25) reported that SNHG7 was overexpressed in 26 cisplatin-resistant NSCLC tumors. Gain and loss of function analyses demonstrated that the high expression of SNHG7 promoted cisplatin-resistance in NSCLC cells. In terms of the mechanism of action, silencing of SNHG7 inhibited the expression of MDR1 (26), BCRP (27) and the activation of the PI3K/AKT/mTOR pathway (28), which have been shown to be associated with drug-resistance. SNHG7 promoted NSCLC cisplatin-resistance by modulating the expression of MDR1, BCRP and by regulating the PI3K/AKT/mTOR pathway.

SNHG7 in osteosarcoma (OS). SNHG7 expression was upregulated in 30 OS tumors compared with its expression noted in normal tissues (29). Patients with high SNHG7 indicated larger tumor size, higher Enneking staging and higher risk of metastasis. Moreover, bioinformatic analysis and dual luciferase reporter assays revealed that SNHG7 directly interacted with miR-34a and exhibited a negative correlation with

miR-34a. Suppression of SNHG7 induced OS cell apoptosis and arrested the cell cycle transition from the G1 to the S phase *in vitro*. SNHG7 further regulated OS cell migration and invasion by sponging miR-34a, which targeted and suppressed the epithelial mesenchymal transition (EMT) by the TGF- β /SMAD4 signaling pathway. In addition, the target genes of miR-34a included Notch1 that is associated with cell proliferation (30), BCL-2, associated with apoptosis (31), CDK6, associated with the cell cycle (32) and SMAD4 (33), associated with EMT. The expression levels of all these targets were promoted by SNHG7. *In vivo*, SNHG7 promoted tumor growth by sponging miR-34a. Overall, SNHG7 functioned as a sponge to inhibit miR-34a expression and to promote tumor growth *in vivo* and *in vitro*.

SNHG7 in bladder cancer (BC). BC is the fourth leading cancer in men and the eighth in women (34). This disease leads to poor patient prognosis in patients treated by radical cystectomy, radiation therapy and postoperative instillation of chemotherapy or immunotherapy (35,36). Zhong *et al* (37) demonstrated that SNHG7 expression was elevated in BC cell lines. Moreover, the expression levels of SNHG7 were increased in BC tissues and exhibited a close association with tumor size, metastasis and clinical stage. Knockdown of SNHG7 inhibited invasion and cell proliferation of BC and induced cell apoptosis. In addition, silencing of SNHG7 inhibited EMT. Chen *et al* (38) further demonstrated that the expression levels of SNHG7 were associated with tumor range, lymph node metastasis and pathological stage. SNHG7 acted as an independent prognostic marker for BC patients. It was also shown that SNHG7-knockdown arrested the cell cycle from the G0/G1 phase to the S phase in BC cells, whereas it was also able to inhibit the activation of the Wnt/ β -catenin pathway. The latter promotes BC cell proliferation, migration and cell cycle. Taken together, the data demonstrated that SNHG7 promoted BC progression by activating the Wnt/ β -catenin pathway. Xu *et al* (39) reported that SNHG7-knockdown enhanced the protein expression levels of Bax, p21 and E-cadherin.

SNHG7 in prostate cancer (PCa). PCa is one of the most common types of male tumors and can be detected by specific screening methods (40). The first study published in 2018 (41) demonstrated that SNHG7 expression levels were significantly increased in PCa tissues and PCa cell lines. This evidence was verified by the fact that PCa patients with high SNHG7 expression exhibited poor prognosis as demonstrated by log-rank test analysis. SNHG7 knockdown inhibited PCa cell proliferation, migration and invasion and induced PCa cell cycle arrest at the G0/G1 phase *in vitro*. *In vivo*, silencing of SNHG7 expression suppressed tumor growth. It was also shown that SNHG7 knockdown could repress EMT in PCa cells. Han *et al* (42) indicated that the lncRNA SNHG7 exhibited a positive correlation with WNT2B via sponging miR-324-3p. In addition, Qi *et al* (41) demonstrated that miR-503 targeted the 3'-untranslated regions (3'-UTR) of SNHG7 and was negatively correlated to SNHG7. miR-503 targeted cyclin D1 and inhibited its expression, suggesting that SNHG7 acted as a ceRNA for miR-503 in order to positively regulate cyclin D1 expression. Previous studies revealed that

SNHG7 acted as a miRNA 'sponge' to regulate the expression and activity of miRNAs and to promote EMT in PCa cell lines.

SNHG7 in breast cancer. Breast cancer is a high incidence malignant tumor originating from luminal epithelial cells and is one of the most commonly diagnosed cancer (43). Luo *et al* (44) focused initially on studying SNHG7 expression in breast cancer in 2018. The authors reported that SNHG7 expression was elevated in 72 breast cancer tissues and that it was associated with poor prognosis of breast cancer patients. SNHG7 correlated positively with tumor stage, lymph node metastasis and distant metastasis in breast cancer patients. Moreover, SNHG7 expression was upregulated in breast cancer cell lines. Knockdown of SNHG7 inhibited breast cancer cell proliferation, migration and invasion *in vitro*. The mechanism of action involved direct targeting of SNHG7 by miRNA-186 (45). Furthermore, SNHG7 expression correlated negatively with miRNA-186 expression. In addition, Sun *et al* (45) verified that SNHG7 could sponge miR34a to inhibit its expression and promote EMT induction and the activation of the Notch-1 pathway. Gao *et al* (46) demonstrated that SNHG7 directly sponged miRNA-381 to promote breast cancer cell proliferation and invasion. Collectively, these findings revealed that SNHG7 may be a new target for breast cancer diagnosis and treatment.

SNHG7 expression in hepatocellular carcinoma (HCC). HCC is one of the most common cancers responsible for approximately 600,000 fatalities every year. Although diagnostic technology, surgical therapy, chemotherapy and molecular targeting therapy are improved, the overall 5-year survival rate of HCC patients is still unsatisfactory due to the recurrence and metastasis of the tumors (47,48). Cui *et al* (49) investigated the expression profile of certain lncRNAs in HCC and normal tissues. The results demonstrated that SNHG7 and PVT1 may participate in HCC cell metastasis. Gain and loss of function analyses demonstrated that silencing of SNHG7 and PVT1 expressions could inhibit HCC cell invasion.

SNHG7 in pancreatic carcinoma (PC). PC is one of the most malignant human cancers of the digestive system and the overall 5-year survival rate of PC is less than 5% due to early metastasis (50,51). SNHG7 expression was significantly increased in 40 PC tissues and 5 PC cell lines (52). Univariate analysis indicated that the patients with high SNHG7 expression were associated with poor prognosis. Knockdown of SNHG7 inhibited PC cell proliferation, migration and invasion *in vitro*. Furthermore, SNHG7 functioned as a ceRNA by directly targeting miR-342-3p in a sequence-specific manner. In addition, ID4 was directly targeted by miR-342-3p and negatively correlated with miR-342-3p expression. This evidence suggested that SNHG7 acted as a sponge by targeting miR-342-3p and that it could abolish the inhibition of miR-342-3p expression. Furthermore, SNHG7-knockdown decreased the tumor volume and weight *in vivo*. miR-342-3p expression was upregulated, while ID4 expression was downregulated. As mentioned above, SNHG7 has been

shown to act as a tumor-promoting factor and may be used as a potential prognostic biomarker and therapeutic target for PC patients.

SNHG7 in esophageal cancer. Xu *et al* (53) demonstrated that SNHG7 expression was upregulated in esophageal cancer tissues and that it was associated with lymph node metastasis. SNHG7 expression was also increased in 4 different esophageal cancer cell lines (Eca109, EC9706, TE10, and TE-11). Furthermore, SNHG7-knockdown could inhibit the proliferation of esophageal cancer cells. Flow cytometry analysis indicated that silencing of SNHG7 expression induced apoptosis and arrested the cell cycle transition from the G1 to the S phase in esophageal cancer cells. Western blotting and reverse transcription-quantitative PCR revealed that SNHG7-knockdown promoted the expression of p15 and p16 which act as tumor-suppressor genes according to previous studies (54,55).

SNHG7 in gastric cancer. Wang *et al* (56) demonstrated that the expression levels of SNHG7 were upregulated in 68 gastric cancer tissues and five gastric cancer cell lines (BGC823, MGC803, SGC7901, N87 and AGS). The CCK-8 assay indicated that interference on SNHG7 expression inhibited gastric cell proliferation. In addition, SNHG7-knockdown increased the apoptotic rate of gastric cancer cells and arrested the cell cycle transition from the G1 to the S phase. *In vivo*, SNHG7-knockdown inhibited tumorigenesis and promoted the expression levels of p15 and p16.

SNHG7 expression in colorectal cancer (CRC). Li *et al* (57) demonstrated that the expression levels of SNHG7 exhibited a significant increase in CRC cells by microarray and GO analyses. The expression levels of SNHG7 indicated a higher increase in CRC cells than in normal cells and were upregulated in CRC tissues compared with those noted in the adjacent tissues. SNHG7 overexpression correlated positively with tumor size, lymphatic metastasis, distant metastasis and tumor stage. Log-rank test analysis indicated that SNHG7 overexpression induced poor prognosis. *In vitro* experiments that investigated gain and loss of function revealed that SNHG7 promoted CRC cell proliferation, migration, invasion and vasculogenic mimicry, while it accelerated the cell cycle progression and inhibited the induction of apoptosis. *In vivo*, SNHG7 promoted CRC cell proliferation and metastasis. Luciferase reporter assays indicated that GALNT7 and SNHG7 were directly targeted by miR-34a. The data indicated that SNHG7 acted as a ceRNA to modulate the expression of GALNT7 by sponging miR-34a. This pathway contributed to CRC progression. Moreover, SNHG7 stimulated the PI3K/AKT/mTOR pathway as determined by KEGG pathway enrichment analysis in CRC cells. Shan *et al* (58) verified that SNHG7 correlated positively with GALNT1 expression and sponged miR-216b, which inhibited growth and metastasis of CRC cells. Taken together, these studies demonstrated that SNHG7 may be considered a novel diagnostic and therapeutic target.

SNHG7 in glioblastoma (GMB). GMB exhibits the highest mortality rate among human cancer types and is one of the

most common primary brain tumors (59). Ren *et al.* (60) demonstrated that SNHG7 expression was increased in 53 GBM tissues and in GBM cell lines. Log-rank test analysis demonstrated that the patients with high SNHG7 expression were associated with poor prognosis. SNHG7 silencing inhibited GBM cell proliferation, migration and invasion of GBM cells, while increasing the rate of cell apoptosis. Moreover, SNHG7 directly targeted miR-5095 and inhibited its expression, which in turn inhibited cell migration, proliferation and invasion and promoted cell apoptosis. Luciferase assays confirmed that the CTNNB1 mRNA expression levels were directly targeted by miR-5095 and correlated negatively with miR-5095 expression. Moreover, SNHG7 overexpression activated the Wnt/b-catenin signaling pathway. Collectively, the data demonstrated that SNHG7 activated the Wnt/b-catenin signaling pathway by directly inhibiting the expression of miR-5095.

SNHG7 expression in other cancer types. SNHG7 expression was elevated in 40 malignant melanoma tissues compared with 30 melanocytic nevus tissues and it was increased in 4 melanoma cell lines (WM266-4, SK-MEL-28, A375, SK-MEL-2) compared with HEMa-LP cells (61). *In vitro*, SNHG7 knockdown inhibited migration and invasion of melanoma cells. In addition, SNHG7 promoted the expression of SOX4. Wang *et al.* (62) demonstrated that the expression of SNHG7 was upregulated in 64 thyroid cancer (TC) tissues and in 4 TC cell lines. In addition, SNHG7 expression correlated positively with increased tumor size and advanced TNM stage. Silencing of SNHG7 inhibited TC cell proliferation and induced apoptosis. Moreover, BDNF expression was significantly increased in TC tissues and exhibited a positive correlation with SNHG7 expression. Recently, Wu *et al.* (63) reported that the expression levels of SNHG7 were elevated in hypopharyngeal cancer (HPC) tissues and were repressed by metformin. Moreover, HPC patients with high SNHG7 expression exhibited a significant correlation with increased tumor size, lower differentiation, lymph node metastasis, distant metastasis, advanced TNM stage, lower overall survival time and resistance to taxol. SNHG7 overexpression improved cell viability and inhibited the induction of cell apoptosis in FaDu cells, which was reversed by metformin. These effects were also confirmed *in vivo*. Metformin led to a hypermethylation of the SNHG7 promoter and increased the activity of SAHH. Moreover, it increased the expression levels of DNMT1, which could regulate the hypermethylation of the SNHG7 promoter and control the expression levels of the downstream molecular targets. These studies suggested that metformin could regulate SNHG7 expression at the epigenetic level. Metformin sensitized FaDu cells to taxol and radiotherapy, while SNHG7 overexpression reversed this effect.

4. Conclusion and future perspectives

Accumulating evidence has verified that the dysregulated expression of lncRNAs may act in an oncogenic or tumor suppressing way in order to regulate tumor progression and tumorigenesis. lncRNAs have potential applications as tumor biomarkers in cancer diagnosis and prognosis and as cancer

therapeutic targets. Recent studies have revealed that SNHG7 is a novel lncRNA. The expression levels of SNHG7 were increased in lung cancer, OS, BC, breast cancer, PCa, HCC, PC, esophageal cancer, gastric cancer, CRC, GMB, melanoma, TC and HPC (Table I). These patterns of expression were associated with specific clinical features, such as tumor size, differentiation, lymph node metastasis, distant metastasis, TNM stage, overall survival time and drug resistance (Table II). SNHG7 acts as an oncogene and regulates cell biological functions including cell proliferation, migration, invasion, inhibition of apoptosis and cell cycle progression. Moreover, SNHG7 acts as a ceRNA to modulate the expression of miRNAs (such as miR-5905) and affect the expression of the downstream genes (such as CTNNB1) or regulate specific classical signaling pathways (Table I). Taken together, these studies verified that SNHG7 plays an important role in tumor development. Therefore, SNHG7 may act as a tumor biomarker in cancer diagnosis and prognosis or as a cancer therapeutic target. The comprehensive assessment of the included studies in the present report revealed that various miRNAs and target signaling molecules were associated with SNHG7 expression depending on the cancer type. It is suggested that one type of lncRNAs can regulate dozens or hundreds of different miRNAs and target signaling molecules. Concomitantly, various miRNAs and target signaling molecules play distinct roles in different tumors due to the complex process of tumor progression and tumorigenesis. Therefore, SNHG7 can regulate different miRNAs and target signaling molecules due to the different nature of the various cancer types. Although the role of SNHG7 has been reported in various cancer types, it has not been previously examined in nasopharyngeal carcinoma and multiple myeloma. Moreover, the exploration of the detailed molecular mechanisms acting upstream and downstream of SNHG7 is still at an early stage. Future studies should focus on exploring the detailed molecular regulatory mechanisms of SNHG7. Higher tumor sample sizes should also be employed in order to validate the findings and promote the potential clinical application of SNHG7.

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Authors' contributions

ZB, WJ, JS and XZ conceived and designed the present review. BX, WH and JJ were involved in designing the review. ZB,

Table I. Functional characterization of SNHG7 in various cancers.

Cancer type	Expression	Role	Biological functions	Related genes	(Refs)
Lung cancer	Up	Oncogenic	Proliferation, migration, invasion, apoptosis	FAIM2, MDR1, BCRP, PI3K/AKT/mTOR, miR-193	(23-25)
Osteosarcoma	Up	Oncogenic	Apoptosis, cell cycle arrest, migration, invasion	MiR-34a, EMT, TGF- β , SMAD4, Notch1, BCL-2, CDK6	(29)
Bladder cancer	Up	Oncogenic	Proliferation, apoptosis, invasion, cell cycle arrest	EMT, Wnt/ β catenin, Bax, p21, E-cadherin	(37-39)
Prostate cancer	Up	Oncogenic	Proliferation, migration, invasion, cell cycle arrest	WNT2B, miR-324-3p, miR-503, Cyclin D1	(41,42)
Breast cancer	Up	Oncogenic	Proliferation migration, invasion	miR34a, EMT, Notch-1, miRNA-381, microRNA-186	(44-46)
Hepatocellular carcinoma	Up	Oncogenic	Invasion	PVT1	(49)
Pancreatic carcinoma	Up	Oncogenic	Proliferation, migration, invasion	MiR-342-3p, ID4	(52)
Esophageal cancer	Up	Oncogenic	Proliferation, cell cycle arrest	P15, p16	(53)
Gastric cancer	Up	Oncogenic	Proliferation, cell cycle arrest, apoptosis	P15, p16	(55)
Colorectal cancer	Up	Oncogenic	Proliferation, migration, invasion, vasculogenic mimicry, apoptosis, cell cycle arrest	PI3K/AKT/mTOR, GALNT1, miR-216b, miR-34a,	(57,58)
Glioblastoma	Up	Oncogenic	Proliferation, migration, invasion, apoptosis	miR-5095, CTNNB1	(60)
Melanoma	Up	Oncogenic	Migration, invasion	SOX4	(61)
Thyroid cancer	Up	Oncogenic	Proliferation, apoptosis	BDNF	(62)
Hypopharyngeal cancer	Up	Oncogenic	Apoptosis, cell viability	SAHH, DNMT1	(63)

UP, upregulated.

Table II. Clinical significance of SNHG7 in diverse cancers.

Cancer types	Overexpression of SNHG7 and clinical features	(Refs)
Lung cancer	TNM stage, lymph node metastases	(23-25)
Osteosarcoma	Tumor size, Enneking staging, distant metastasis	(29)
Bladder cancer	Tumor size, metastasis, clinic stage, lymph nodes, pathological stage	(37-39)
Prostate cancer	Prognosis	(41,42)
Breast cancer	Tumor stage, lymph node metastasis, distant metastasis	(44-46)
Pancreatic carcinoma	Prognosis	(52)
Esophageal cancer	Lymph node metastasis	(53)
Colorectal cancer	Tumor size, lymphatic metastasis, distant metastasis, tumor stage, prognosis	(57,58)
Glioblastoma	prognosis	(60)
Thyroid cancer	Tumor size, TNM stage	(62)
Hypopharyngeal cancer	Tumor size, differentiation, lymph node metastasis, distant metastasis, TNM stage, overall survival time, taxol resistant	(63)

TNM, tumor node metastasis.

WJ, JS, XZ, BX, WH and JJ were involved in the collection of references. ZB and WJ wrote the manuscript. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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