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LncRNA SNHG1: role in tumorigenesis of multiple human cancers

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Abstract
Small nucleolar RNA host gene 1 (SNHG1) is an important member of the SNHG family. This family is composed of a group of host genes that can be processed into small nucleolar RNAs and play important biological functions. In an oncogenic role, the SNHG1 expression is increased in various cancers, which has immense application prospects in the diagnosis, treatment, and prognosis of malignant tumors. In this review, we have summarized the role and molecular mechanism of SNHG1 in the development of various cancers. In addition, we have emphasized the clinical significance of SNHG1 in cancers in our article. This molecule is expected to be a new marker for potential usage in the diagnosis, prognosis, and treatment of cancer.

Keywords LncRNA, SNHG1, Diagnosis, Prognosis, Treatment, Molecular mechanism

Introduction

Cancer is a major contributor to the global disease burden, and it is gaining prominence as a leading cause of death [1]. Research in the field of genetic and epigenetic modifications is of enormous significance in comprehending the process of tumorigenesis [4]. Although genes are commonly transcribed, the coding genome accounts for <2% of all gene sequences according to transcriptome sequencing data [2], and there exists innumerable and diverse noncoding RNA (ncRNA) sequences in cells [3]. Advances in the study of tumor-associated ncRNAs are likely to guide the identification of new diagnostic and prognostic biomarkers as well as the development of effective therapeutics.

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Aberrant expression of long-noncoding RNA (lncRNA) has been associated with cell migration, invasion, metastasis, gene transcription, and tumorigenesis [5]. LncRNAs can directly bind to RNA, DNA, and proteins to perform their biological functions as tumor suppressor genes or oncogenes. Moreover, lncRNAs can act as competitive endogenous RNA (ceRNA) or microRNA (miRNA) sponges in cells by competitively binding to the latter, which indirectly impacts messenger RNA (mRNA) expression, and, hence, the tumor progression [6].

Small nucleolar RNA host gene 1 (SNHG1) is located in chromosome 11q12.3, which is the host of eight small nucleolar RNAs (snoRNAs) (SNHG1, GenBank accession number: 23,642). Recent investigations have reported that SNHG1 serves as an essential regulator in several diseases. For example, in the treatment of myocardial infarction, a positive feedback loop between SNHG1 and c-Myc is involved in the treatment of heart failure after myocardial infarction [7]. During epilepsy progression, SNHG1 delays epilepsy progression by regulating the miR-181a/BCL-2 axis in vitro [9]. More importantly, SNHG1 is intricately linked to cancer and is abnormally expressed in various cancers, including hepatocellular

carcinoma (HCC) [16], breast cancer (BC) [19], bladder cancer (BLC) [20], esophageal squamous cell carcinoma (ESCC) [23], prostate cancer (PCa) [27], osteosarcoma (OS) [28], non-small cell lung cancer (NSCLC) [33], colorectal cancer (CRC) [38], gastric cancer (GC) [40], and acute myeloid leukemia (AML) [48]. As an essential member of the SNHG family, SNHG1 should be systematically reviewed. This article briefly reviews the progress of research on SNHG1 in the abovementioned cancers in the past 5 years. Furthermore, it summarizes the tumor-promoting mechanism of SNHG1 and the corresponding clinical significance and lists [see Additional File 1] the abnormal expression of SNHG1 in tumor conditions reported in the past 5 years. The research findings are discussed in the ensuing sections.

Discovery of SNHG1

SNHG is the host gene of snoRNAs present in the nucleus and cytoplasm [10]. Of the 22 members of the SNHG family, SNHG1 is crucial and functions as an oncogene that promotes tumor growth [11]. The first article on SNHG1 was published by Chaudhry [8], whose research demonstrated that the gene is induced in TK6 and WTK1 cells. You et al.'s study was the first to show that SNHG1 is associated with cancer. In patients with NSCLC, the expression of SNHG1 was found to be significantly increased in cancer cells [12]. Numerous studies conducted in recent years have noted that SNHG1 plays an oncogenic role in various cancers. Data from the Gene Expression Profiling Interactive Analysis 2 database (Fig. 1) indicated that SNHG1 is highly expressed in pan-cancer. Various clinicopathological features and prognoses of cancer, including tumor lymph node metastasis (TNM) stage, lymph node metastasis, and overall survival (OS), have been observed to be positively correlated with the upregulation of SNHG1. SNHG1 can act as an oncogene, and its upregulation expression promotes cell proliferation, migration, and invasion. In addition, related studies on HCC [13], BC [14], and AML [50] have reported that SNHG1 occurs in exosomes. The biological process of various tumors is regulated by SNHG1. This gene plays a vital role in the occurrence, development, and prognosis of various malignant tumors and may, hence, become a valuable therapeutic target and prognostic biomarker for several cancers. Therefore, the function and molecular mechanism of SNHG1 (Fig. 2) warrant in-depth investigation.

SNHG1 in various cancers

Hepatocellular carcinoma

SNHG1 expression has been demonstrated to be elevated in HCC tissues and cell lines compared with adjacent normal liver cell lines and non-neoplastic tissues [15–17]. Moreover, SNHG1 is expressed in both the nucleus and

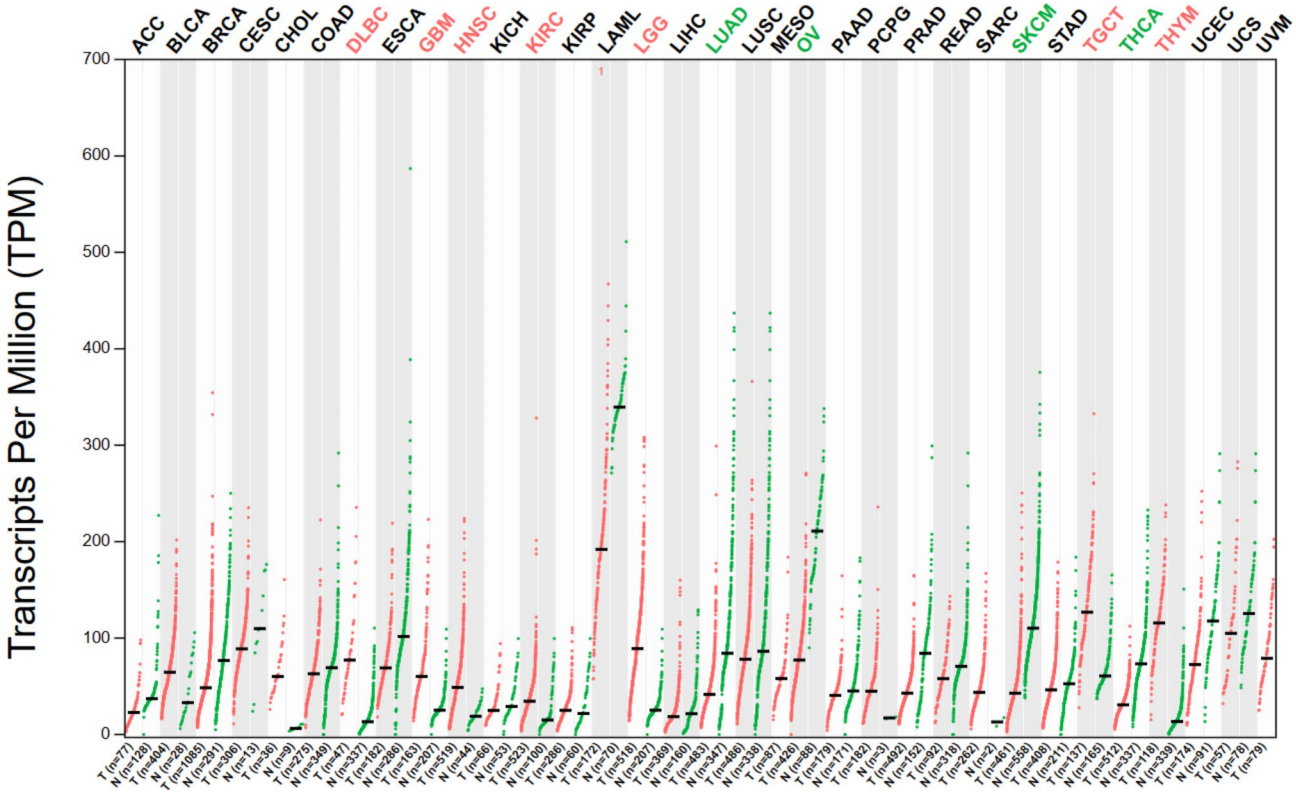

Fig. 1 The SNHG1 expression in different cancers. Data from the GEPIA2 database indicates that SNHG1 was overexpressed in various tumors

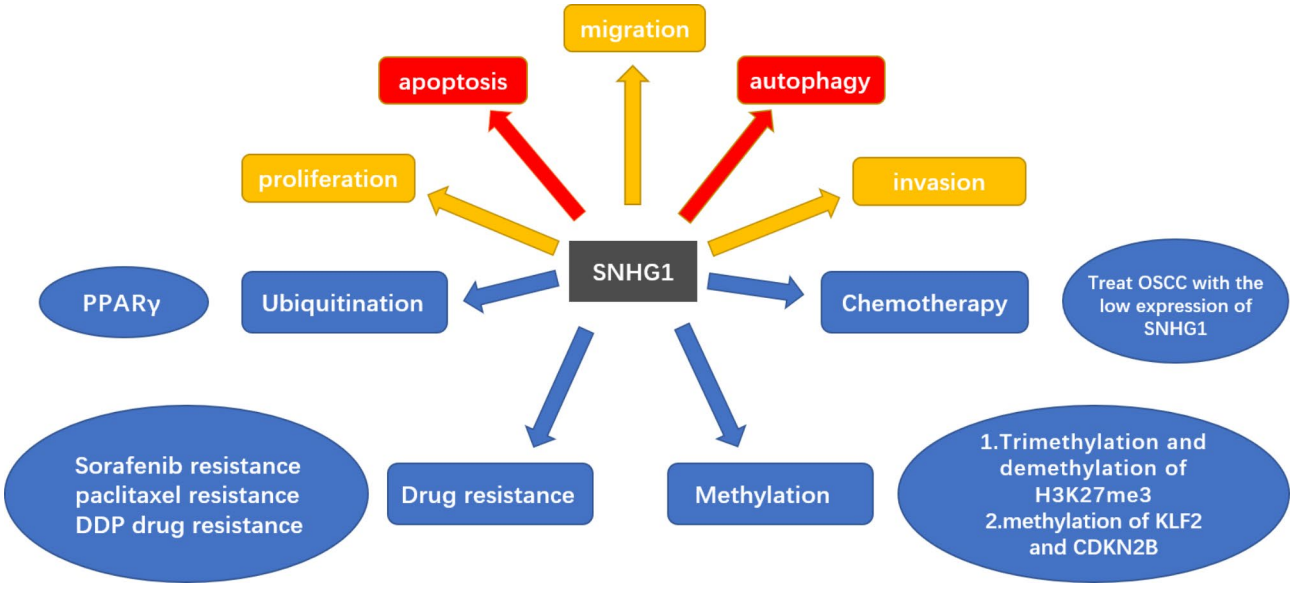

Fig. 2 The function of lncRNA SNHG1 in tumor cells. SNHG1 can regulate tumorigeneses, such as cell proliferation, invasion, and migration in cells, as well as inhibit apoptosis and autophagy. Meanwhile, SNHG1 plays a role in the processes of ubiquitination, methylation, drug resistance, and chemotherapy, among others

the cytoplasm [17]. It was reported that SNHG1 increases the expression of AEG-1 protein in HCC cells by sponging miR-195, thereby promoting the development of HCC [18]. According to the experimental results of Dong et al. [16], Sorafenib promotes the expression of SNHG1 by inducing the translocation of miR-21 into the nucleus. This process leads to the upregulation of SLC3A2, which in turn upregulates the Akt pathway to promote sorafenib resistance. Independently, miR-21 activates the Akt pathway by downregulating PTEN expression. Moreover, SNHG1 has little effect on miR-21 expression. A past study reported that SNHG1 promotes the progression of HCC by epigenetically silencing CDKN1A and CDKN2B in the nucleus and competing with CDK4 mRNA to bind miR-140-5p in the cytoplasm [17]. These two molecular mechanisms of SNHG1 promote the progression of HCC. These data collectively propose SNHG1 as a new potential target in HCC treatment, especially in terms of its role in facilitating the overcoming of sorafenib resistance in the future.

Breast cancer

It has been reported that SNHG1 is significantly overexpressed in BC tissues and cell lines [19–21]. The overexpression of SNHG1 promotes BC cell proliferation, invasion, and migration and is positively linked to reduced patient survival [5, 6, 13]. Zuo et al. [19] demonstrated that under hypoxic conditions, SNHG1 increases in a HIF-1-dependent manner, downregulating miR-199a-3p and upregulating TFAM. Moreover, SNHG1 regulates tumor growth in vivo by regulating the miR-199a-3p/TFAM axis. Another study on BC [20] signified that miR-193a-5p/HOXA1 competes for endogenous RNA regulatory pathways and that SNHG1 acts as a sponge for miR-193a-5p, thereby activating HOXA1 expression. In the field of epigenetics, a past study showed that SNHG1 interacts with EZH2 [21]. EZH2 recruited by SNHG1 triggers trimethylation of H3K27me3, which epigenetically represses miR-381 transcription in these cells. The overexpression of miR-381 does not inhibit tumor progression but rather enhances the sensitivity of cells to DDP (cisplatin). Thus, silencing SNHG1 could aid in overcoming DDP resistance in BC cells. These findings allude that SNHG1 greatly influences BC progression and provides a possible new target for tackling the chemotherapeutic resistance of BC.

Esophageal squamous cell carcinoma

Studies have documented that lncRNA SNHG1 is significantly upregulated in the cells and tissues of esophageal squamous cell carcinoma (ESCC) compared with neighboring noncancerous tissues [22–24]. Increased expression of SNHG1 promotes cell proliferation and decreases the apoptosis rate. In addition, this elevated expression is significantly positively correlated with the depth of invasion, tumor size, TNM stage, lymph node metastasis, and short survival time. It was demonstrated that cell proliferation is promoted under the action of SNGH1 by acting as a nondegradable sponge for miR-338 in ESCC, which targets CST3 directly [22]. Another experimental finding [23] asserted that the expressions of miRNA-21 and SNHG1 are upregulated in the ESCC tissues and serum. miRNA-21 can promote cell proliferation in ESCC via SNHG1, and SNHG1 may be a new downstream target

of miRNA-21, unidirectionally acting on ESCC cells. In addition, a past study [24] reported that the inhibition of SNHG1 expression upregulates E-cadherin, downregulates vimentin and N-cadherin, and inhibits cell proliferation, cell invasion, and epithelial–mesenchymal transition (EMT) in ESCC cells. Furthermore, SNHG1 inhibits the Notch signaling pathway by reducing the expressions of Notch1 and Hes1 in ESCC cells. Thus, SNHG1 may be a potential diagnostic marker and therapeutic target for ESCC.

Prostate cancer

LncRNA SNHG1 is significantly upregulated in PCa [25–27] and is sublocalized in the nucleus [25]. In PCa, cell proliferation and cell cycle progression are closely associated with the overexpression of SNHG1. The investigations of Tan et al. [25] established that positive regulators of EMT are activated by SNHG1 via the hnRNPL-CDH1 axis. SNHG1 interacts competitively with hnRNPL, which affects the translation of CDH1. Thus, the effect of SNHG1 on the EMT pathway is activated, and ultimately, the metastasis of PCa is promoted. Chen et al. [26] stated that the expressions of SNHG1 and EZH2 are positively correlated and that both are upregulated in PCa tissues and cells. SNHG1 regulates the PI3K/AKT/mTOR and Wnt/β-catenin signaling pathways via the EZH2 gene, which affects the proliferation, apoptosis, and autophagy of PCa cells. Furthermore, It was observed that SNHG1 enhances CDK7 expression in PCa by competitively binding to miR-199a-3p, which promotes cell proliferation and cell cycle progression [27]. These data reiterate that SNHG1 is a new diagnostic and therapeutic target for PCa.

Osteosarcoma

Some studies have assessed the expression of SNHG1 using quantitative real-time polymerase chain reaction and reported a significant upregulation of SNHG1 in cancer tissues and cell lines [28, 29]. SNHG1 overexpression impairs the osteogenic differentiation ability of AGS cells, which reduces the osteogenic marker expressions, calcium deposition, and alkaline phosphatase activity [30]. In addition, it has been identified that SNHG1 is associated with OS recurrence [31]. Wang et al. [28] showed that SNHG1 is overexpressed in OS tissues and cell lines and that its high expression predicts poor OS in patients. They found that SNHG1 acts as a competing endogenous RNA to increase human NOB1 by sponging miR-326 to promote cell proliferation, migration, and invasion in OS. Experiments have demonstrated that SNHG1 silencing exerts an inhibitory effect on the growth of xenosarcoma [30]. However, this effect is counteracted by the overexpression of S100A6. SNHG1 acts as a ceRNA and promotes the expression of S100A6 via miR-493-5p sponging. Overexpression of S100A6 stimulates the proliferation of OS cells and also reduces their osteogenic differentiation. Meanwhile, it was confirmed that SNHG1 downregulates the expression of miRNA-101-3p and that the expression of ROCK1 is consequently enhanced [29]. Furthermore, SNHG1 activates the PI3K/AKT pathway and EMT expression. In summary, SNHG1 holds application potential in clinical diagnosis and therapy and can be used to develop new therapeutic strategies for OS.

Non-small cell lung cancer

SNHG1 is overexpressed in NSCLC tissues and cells and further tends to be upregulated in DDP-resistant NSCLC tissues and cell lines [32, 33]. These experiments have indicated that SNHG1 is closely associated with the resistance of NSCLC cells to DDP, which is a commonly used chemotherapeutic drug in NSCLC treatment. Past studies have shown that the expression of SNHG1 is upregulated in DDP-resistant NSCLC tissues and cell lines [34, 35]. In SHI's study [34], it was found that SNHG1 was involved in the formation of DDP resistance in NSCLC partly by regulating the miR-140-5p/W-catenin pathway. Ge et al. [35] later confirmed that SNHG1 silencing augments the sensitivity of NSCLC cells to DDP. SNHG1 increases the DCLK1 expression by sponging miR-330-5p, thereby increasing the DDP resistance and malignancy potential of NSCLC cells. In addition, SNHG1 sponges other miRNAs and plays a role in NSCLC. Functional experiments revealed that SNHG1 regulates the FRAT1 expression by sponging miR-361-3p, which promotes proliferation, represses apoptosis, and enhances migration and invasion of NSCLC cells [32]. Another study proved that SNHG1 upregulates the expression of MTDH by sponging miR-145-5p, thereby accelerating the progression of NSCLC [33]. These findings imply that SNHG1 has potential as a therapeutic target for NSCLC.

Colorectal cancer

Research has revealed that the expression of SNHG1 is significantly increased in both colon cancer tissues and cell lines when compared with that in the normal samples. The overexpression of SNHG1 has been shown to enhance cell proliferation, invasion, migration, and EMT progression in colon cancer. Furthermore, elevated SNHG1 expression is positively correlated with advanced TNM stage, poor prognosis, and shorter OS [36–38]. SNHG1 acts as a sponge for miR-154-5p, miR-137, and miR-181b-5p. It was reported that SNHG1 overexpression attenuates the inhibitory effect of miR-154-5p on CCND2 [36]. Another research observed that SNHG1 promotes CRC cell proliferation, migration, and invasion by targeting the miR-181b-5p/SMAD2 axis [37]. SMAD2 induces EMT and affects CRC progression. In addition, Fu et al. [38] asserted that SNHG1 increases the level of

RICTOR in CRC by sponging miR-137. RICTOR can augment the proliferation and invasion ability of CRC cells, thereby promoting tumorigenesis. In the field of epigenetics, SNHG1 directly interacts with PRC2, regulates histone methylation of KLF2 and CDKN2B in the nucleus, and inhibits the epigenetic modification of KLF2 and CDKN2B. This process promotes the development of CRC [36]. Therefore, SNHG1 may be a potential biomarker for colon cancer and has the potential to serve as a new therapeutic target.

Gastric cancer

The function and expression of SNHG1 in GC differ from those in other cancers. Some studies have demonstrated that SNHG1 is overexpressed in GC [39–41]. Liu et al. [39] stated that SNHG1 accentuates the influence of DCLK1/Notch1 on EMT by regulating the miR-15b expression. A past study [40] proved that SNHG1 acts as a ceRNA to sponge miR-195-5p and thereby upregulates the expression of YAP1, which promotes GC cell proliferation and metastasis. In Xu's study [41], they noted that silencing SNHG1 effectively inhibits GC cell migration and that the gene serves as a sponge for miR-216b-5p to enhance the HK2 expression. This event can make GC cells less sensitive to paclitaxel Contrary to these findings, Wang et al. [42] showed that SNHG1 can regulate the SOCS2/JAK/STAT signaling pathway by upregulating the expression of SOCS2. In the GC tissues, this signaling pathway is often activated. However, their study uncovered that SNHG1 expression was lower in the GC tissues when compared with that in the neighboring noncancerous tissues. Therefore, the migration and invasion of human GC cells will be inhibited. Furthermore, it was reported that SNHG1 suppresses the proliferation of GC cells and promotes their apoptosis in a Notch1 pathway-dependent manner [43]. These results allude that SNHG1 may function as a tumor suppressor gene in GC as well.

Bladder cancer

Recent studies have demonstrated that the expression of SNHG1 is upregulated in BLC tissues and cells, which contributes to the accelerated development of the disease [44–46]. It was currently confirmed that SNHG1 could sponge miR-493-5p [44], miR-9-3p [45], and miR-143-3p [46] and several other micro-RNA to promote the development of BLC. A past study showed that miR-493 binds to the 3′2-UTR of ATG14 mRNA, which affects the expression of the ATG14 protein [44]. This binding has been implicated in the autophagy of BLC cells. In this manner, SNHG1 promotes the proliferation, invasion, and autophagy of BLC cells by targeting the miR-493-5p/ATG14/autophagy pathway. Another study documented that SNHG1 could be used as a ceRNA to reduce the MDM2 expression by sponging miR-9-3p [45]. Moreover, MDM2 induces the ubiquitination and degradation of PPARγ. SNHG1 promotes BLC development by repressing MDM2 expression via splicing. Xiang et al. [46] observed that SNHG1 augments the expression of HK2 by targeting the miR-143-3p/HK2 axis. In addition, they found that SNHG1 serves as a platform for recruiting EZH2 to the initiator subregion of CDH1, thereby catalyzing the trimethylation of H3K27me3 in the CDH1 promoter. SNHG1 inhibits the expression of CDH1 and alters the biological behavior of BLC cells epigenetically. Collectively, these findings suggest that the dysregulation of SNHG1 is involved in the pathogenesis and progression of BLC, implying that it represents a promising target for the development of novel therapeutic strategies.

Acute myeloid leukemia

In recent years, several studies have demonstrated that SNHG1 expression is abnormally upregulated in AML tissues and cells [47–50]. Patients with AML who have high SNHG1 expression tend to have lower OS and shorter recurrence-free survival. Moreover, a high SNHG1 expression has been significantly associated with higher white blood cell counts, lower complete response rates, unfavorable cytogenetics, and higher recurrence rates [47].

A past study showed that SNHG1 negatively regulates miR101 by sponging it, thereby promoting the progression of AML [47]. Another study observed that the SNHG1/miR-489-3p/SOX12/Wnt/β-catenin signaling axis is present in AML cells. SNHG1 inhibits the expression of miR-489-3p, which activates the SOX12/Wnt/β-catenin signaling pathway, thereby promoting the growth of AML cells [48]. It was demonstrated that the expression of SNHG1 is elevated in pAML and THP-1 cells, and it downregulates the miR-488-5p expression by sponging it. SNHG1 promotes the progression of AML via the miR488-5p/NUP205 axis [49]. In addition, Xiao et al. [50], for the first time, successfully isolated plasma exosomes from patients with AML and healthy donors (HDs). Their study revealed that the expression of exosomal lncRNA SNHG1 was significantly upregulated in the plasma of patients with AML (n=65) when compared with HDs (n=20). They demonstrated that SNHG1 could distinguish between those with AML and HDs and was highly stable in plasma exosomes. Exosomal SNHG1 expression was suppressed after alloHSCT treatment. In conclusion, SNHG1 may serve as a potential prognostic biomarker in AML and provide a novel target for the development of new therapeutic strategies for AML.

Conclusions

SNHG1 is involved in the pathogenesis of various human diseases and plays a crucial regulatory role in several tumors. The application value of SNHG1 in

tumor research is worth exploring. SNHG1 is present as an oncogene in most types of cancer and is overexpressed in various cancers. Several clinicopathological features and prognoses, such as OS, TNM stage, and lymph node metastasis of patients, have been shown to be significantly correlated with the abnormal expression of SNHG1. Moreover, SNHG1 plays an essential role in the processes of tumor cell proliferation, invasion, migration, and apoptosis. However, different studies suggest that the carcinogenesis and tumor suppressor effects of SNHG1 in GC remain inconsistent. This disparity could be ascribed to the variations in the gene expressions of different tumors, which remains shrouded in controversy and warrants further research. Collectively, these findings allude that SNHG1 may enhance the response of cancer cells to conventional therapeutic regimens by acting as a target.

SNHG1 utilizes complex molecular functions and cellular mechanisms to play its role in cancer progression. Presently, various signaling pathways linked to the occurrence and development of cancer have been reported to be regulated by SNHG1, including Wnt/β-catenin, PTEN/PI3K/AKT, EMT, Notch, and p53. In terms of molecular mechanisms associated with ceRNA regulation, SNHG1 acts as a molecular sponge by sponging miR-216b-5p, miR-195 5p, and miR-199a-3p as well as several other miRNAs. In conclusion, SNHG1 has the potential to become an emerging tumor diagnostic target and can be used as a prognostic biomarker or therapeutic target. Additional molecular mechanisms of SNHG1 may be involved in tumorigenesis, and the influence of epigenetic and environmental factors on SNHG1 is yet to be further elucidated. Of these, SNHG1 can play its biological role via molecular mechanisms such as methylation [21, 36], demethylation [51], and ubiquitination [45] in epigenetics. In the field of chemotherapy, SNHG1 has research potential [52, 66]. These molecular mechanisms are worthy of in-depth exploration in the future.

Future perspectives

SNHG1 may have potential applications in cancer diagnosis, treatment, and prognosis, but its clinical use is fraught with challenges. The main reason is that most of the current studies are based on SNHG1 in cancer tissues and cells. In the future, SNHG1 needs to be explored in common diagnostic samples, such as blood and other body fluids, to unravel multiple effects between SNHG1 and molecular target markers that can be applied clinically.

As a medium of cell communication, exosomes have become an emerging hot research field. Exosomes can carry proteins and nucleic acids and can exist in some human body fluids, such as blood, saliva, urine, ascites, and cervicovaginal lavage [91]. Several studies in recent years have demonstrated that tumor-derived exosomes can be used as biomarkers for diagnosis, prognosis, or prediction of cancer. Although the current research on SNHG1 in blood, urine, and other body fluid diagnostic samples has achieved some relevant outcomes, for example, related studies on HCC [13], BC [14], and AML [50] have demonstrated that SNHG1 exists in exosomes. Moreover, it was proven that SNHG1 has a high stability in plasma exosomes [50], albeit further in-depth exploration should be continued. In this direction, research around the field of exosomes may be a promising direction.

Furthermore, it is worth noting that, in the field of nontumor diseases, there have been some reports on SNHG1. The molecular mechanism of SNHG1 in nontumor diseases may become a new research hotspot in the future.

Abbreviations

lncRNA	Long noncoding RNA
ceRNA	Competing endogenous RNA
miRNA	MicroRNA
mRNA	Messenger RNA
SNHG1	Small nucleolar RNA host gene 1
ncRNA	Noncoding RNA
snoRNA	Small nucleolar RNA
TME	Tumor microenvironment
HCC	Hepatocellular carcinoma
TNM	Tumor lymph node metastasis
BC	Breast cancer
ESCC	Esophageal carcinoma
EMT	Epithelial–mesenchymal transition
PCa	Prostate cancer
OS	Osteosarcoma
NSCLC	Non-small cell lung cancer
CRC	Colorectal cancer
GC	Gastric cancer
BLC	Bladder cancer
AML	Acute myeloid leukemia
HD	Healthy donor

Supplementary Information

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Supplementary Material 1

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Authors' contributions

HZ wrote and drafted the main manuscript text, and prepared the figures and tables. SZ and WC gathered information or organized graphs. MK and PZ revised and designed the manuscript. All authors reviewed the manuscript.

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Data Availability

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Declarations

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