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EDITORIAL

Molecular mechanisms underlying roles of long non-coding RNA small nucleolar RNA host gene 16 in digestive system cancers

Ting-Fang Yang, Xin-Rui Li, Mo-Wei Kong

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Abstract

This editorial reviews the molecular mechanisms underlying the roles of the long non-coding RNA (lncRNA) small nucleolar RNA host gene 16 (*SNHG16*) in digestive system cancers based on two recent studies on lncRNAs in digestive system tumors. The first study, by Zhao *et al*, explored how hBD-1 affects colon cancer, *via* the lncRNA *TCONS_00014506*, by inhibiting mTOR and promoting autophagy. The second one, by Li *et al*, identified the lncRNA prion protein testis specific (*PRNT*) as a factor in oxaliplatin resistance by sponging ZNF184 to regulate HIPK2 and influence colorectal cancer progression and chemoresistance, suggesting *PRNT* as a potential therapeutic target for colorectal cancer. Both of these two articles discuss the mechanisms by which lncRNAs contribute to the development and progression of digestive system cancers. As a recent research hotspot, *SNHG16* is a typical lncRNA that has been extensively studied for its association with digestive system cancers. The prevailing hypothesis is that *SNHG16* participates in the development and progression of digestive system tumors by acting as a competing endogenous RNA, interacting with other proteins, regulating various genes, and affecting downstream target molecules. This review systematically examines the recently reported biological functions, related molecular mechanisms, and potential clinical significance of *SNHG16* in various digestive system cancers, and explores the relationship between *SNHG16* and digestive system cancers. The findings suggest that *SNHG16* may serve as a potential biomarker and therapeutic target for human digestive system cancers.

Key Words: Digestive system cancers; Long non-coding RNAs; Small nucleolar RNA host gene 16; Colon cancer

Core Tip: The long non-coding RNA (lncRNA) small nucleolar RNA host gene 16 (*SNHG16*) plays a significant role in the development and progression of various digestive system cancers, including esophageal, liver, pancreatic, gastric, and colorectal cancers. It is involved in processes such as cell proliferation, migration, invasion, apoptosis, and chemoresistance. *SNHG16* acts as a competing endogenous RNA, interacting with microRNAs and regulating target genes, and is associated with a poor prognosis in digestive system cancers. Its expression is influenced by transcription factors and its polymorphisms are linked to cancer susceptibility. *SNHG16* has potential as a biomarker and therapeutic target for digestive system cancers.

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INTRODUCTION

This editorial reviews the molecular mechanisms underlying the roles of the long non-coding RNA (lncRNA) small nucleolar RNA host gene 16 (*SNHG16*) in digestive system cancers based on two recent studies on long lncRNAs in colorectal cancer (CRC), one is by Zhao *et al*[1] and the other by Li *et al*[2]. The study of Zhao *et al*[1] linked hBD-1 to mTOR pathway regulation and autophagy *via* the lncRNA *TCONS_00014506*, highlighting the potential of hBD-1 in cancer cell destruction. The study of Li *et al*[2] identified the lncRNA prion protein testis specific (*PRNT*) as a regulator of oxaliplatin resistance, showing its upregulation in resistant CRC cells and its role in HIPK2 expression, suggesting PRNT as a therapeutic target for CRC treatment. Both of these two articles discuss the mechanisms by which lncRNAs contribute to the development and progression of digestive system tumors.

Digestive system tumors remain a major cause of global mortality, with increasing incidence and mortality rates[1,2]. In 2018, there were approximately 18.1 million new cancer cases and 9.6 million cancer-related deaths worldwide, with digestive system tumors accounting for a significant proportion[3]. Despite significant advances in understanding the potential molecular mechanisms of digestive system tumors[4-7] and substantial progress in their treatment[8,9], the recurrence and mortality rates of digestive system tumors remain dismal[1]. Therefore, it is crucial to seek novel effective biomarkers and therapeutic targets for these tumors.

Recent studies have shown that non-coding RNAs (ncRNAs) play a role in the development of digestive system tumors [10]. ncRNAs are generally divided into two categories based on their length: Small ncRNAs with less than 200 nucleotides (nts) and long ncRNAs (lncRNAs) with more than 200 nts[11,12]. MicroRNAs (miRNAs) are small ncRNAs with a length of 20 to 25 nts, which have been proven to negatively regulate the expression of specific key genes and participate in various aspects of cell biology[13,14]. In recent years, lncRNAs have attracted increasing attention from scholars worldwide. Although lncRNAs represent transcripts without protein-coding potential, they can regulate gene expression at multiple levels, including epigenetic, transcriptional, and post-transcriptional regulation[15-17]. Increasing evidence indicates that many lncRNAs display abnormal expression levels in digestive system tumors[18]. The dysregulation of lncRNAs is often involved in cell events related to tumors, including growth, programmed cell death, metastasis, and stem cell characteristics[19]. The lncRNA *SNHG16* contains 2435 nts and is located on chromosome 17q25.1[20]. *SNHG16* has two splice variants, the long form Nbla10727 (2186 nts) and the short form Nbla12061 (2087 nts)[21]. *SNHG16* was initially described as a strong carcinogenic factor leading to poor prognosis in patients with neuroblastoma [20]. Subsequent studies show that *SNHG16* plays a role in the development and progression of various digestive system tumors, and recent research has made breakthroughs in this field[10-12]. However, there is still a lack of research summarizing the molecular mechanisms of *SNHG16* in the development and progression of digestive system tumors.

This paper aims to systematically review the recent findings on the biological functions, related mechanisms, and potential clinical significance of *SNHG16* in various digestive system cancers, and to explore the close relationship between *SNHG16* and digestive system cancers.

MOLECULAR REGULATORY MECHANISMS OF LNCRNAS AND *SNHG16*

LncRNAs are single-stranded RNA molecules transcribed by RNA polymerase II, with a length of more than 200 nts and lacking protein-coding ability[22]. Overwhelming evidence from numerous studies indicates that lncRNAs play a crucial role in the development, proliferation, migration, and prognosis of various cancers by regulating a series of biological processes, such as interacting with target genes at the transcriptional level, regulating histone modifications and chromatin remodeling, and interacting with miRNAs of approximately 22 nts in length (also known as competing endogenous RNAs [ceRNAs])[22-25]. For example, the lncRNA BC069792 acts as a ceRNA to sponge miR-658 and miR-4739, increasing the expression of the target gene *KCNQ4*, leading to AKT phosphorylation, and thus inhibiting the prolif-

eration and invasion of breast cancer[24,25].

SNHG16 is a member of the SNHG family, located on human chromosome 17q25.1, and consists of four exons. Initially, it was identified as a potent carcinogenic factor that promotes the progression of neuroblastoma[26]. Therefore, *SNHG16* is also known as non-coding RNA expressed in aggressive neuroblastoma. Subsequent studies further revealed the widespread involvement of *SNHG16* in the complex molecular regulatory networks in different human cancers[27-29]. For instance, by regulating the miR-32-5p/SPN axis, the silencing of *SNHG16* inhibits the proliferation and radioresistance of nasopharyngeal carcinoma cells[28]. *SNHG16* may act as an oncogene by binding and recruiting EZH2 to the *p21* promoter, silencing the expression of p21, thereby promoting cell proliferation and reducing apoptosis in bladder cancer cells[29]. *SNHG16* plays a key role in the progression, distant metastasis, and prognosis of ovarian cancer by increasing the expression of MMP9[30]. In addition, in oral squamous cell carcinoma, the expression of *SNHG16* is regulated by a transcription factor called c-Myc, which recruits histone acetyltransferases and induces the clearance of RNA polymerase II[31]. These functions indicate that *SNHG16* plays an important role in the progression, invasion, and carcinogenesis of human cancers through various molecular mechanisms.

MECHANISMS OF *SNHG16* IN DIGESTIVE SYSTEM TUMORS

SNHG16 and esophageal squamous cell carcinoma

Esophageal cancer (EC) is one of the major cancer types worldwide, ranking 7th in incidence (3.1%, 604100 new cases) and 6th in mortality (5.5%, 544076 deaths) among all cancers[2]. There are differences in the incidence and mortality rates of EC across geographic regions[2]. For example, due to economic underdevelopment and dietary habits, the burden of EC in East Asia is heavier, with esophageal squamous cell carcinoma (ESCC) being the predominant histological type[2]. Studies have shown that *SNHG16* is upregulated in EC and closely related to tumor stage, lymph node metastasis, and clinical stage. Silencing of *SNHG16* inhibits the proliferation and invasion of EC-1 and Eca-109 cells by reducing the expression of β-catenin, cyclin D1, and c-Myc proteins, and promotes apoptosis[32]. This study also showed that *SNHG16* is upregulated in ESCC tissue and cell lines, and disrupting *SNHG16* expression promotes apoptosis and inhibits epithelial-mesenchymal transition (EMT) through the miR-140-5p/ZEB1 axis. Another study found that increased expression of *SNHG16* also promotes the growth and metastasis of ESCC, and is related to tumor differentiation and T stage, with the mechanism being that *SNHG16* can bind and recruit EIF4A3 to regulate the expression of RhoU by enhancing the stability of *RhoU* mRNA[33]. These results indicate that the upregulation of *SNHG16* is closely related to the development of ESCC, and *SNHG16* is expected to serve as a marker for ESCC, providing new clues for its clinical treatment and the development of related drugs.

SNHG16 and hepatocellular carcinoma

Hepatocellular carcinoma (HCC) is one of the most common malignant tumors worldwide, accounting for 4.7% of all new cancer cases (906000 cases) and 8.3% of cancer-related deaths (830000 cases), ranking sixth and third among all malignancies, respectively[2]. In most studies, *SNHG16* is considered an oncogene for HCC, and the upregulation of *SNHG16* expression is closely related to the malignant prognosis and tumor stage of HCC. The expression of *SNHG16* in late-stage HCC is significantly higher than that in early-stage HCC[34,35]. Additionally, high expression of *SNHG16* is also associated with tumor size, TNM stage, and vascular invasion in HCC patients[36]. *SNHG16*, as a ceRNA, targets STAT3 and GALNT1 through miR-4500 and miR-let-7b-5p in Huh-7 and HUVEC cells, respectively, promoting the proliferation, metastasis, and angiogenesis of Huh-7 cells and enhancing vascular formation in HUVEC cells[34,36] (Figure 1).

Other studies have shown that downregulating *SNHG16* expression affects the *SNHG16*/miR-195, *SNHG16*/miR-17-5p/P62, and *SNHG16*/miR-302a-3p/FGF19 axes, inhibiting the proliferation, migration, and invasion of HepG2 and Hep3B cells[34,37-39] (Figure 2).

The overexpression of *SNHG16* affects the G2/M transition of HCC cells by sponging miR-let-7b-5p[40]. *SNHG16* is overexpressed in sorafenib-resistant HCC tumor tissues and cells, enhancing the resistance of HCC cells to sorafenib[36]. Conversely, when the expression of *SNHG16* is inhibited, the resistance to sorafenib disappears[41]. Assarzadegan *et al*[42] proposed that *SNHG16* enhances the autophaic function of HCC cells to protect HCC from sorafenib resistance through the miR-23b-3p/EGR1 axis. Moreover, *SNHG16* exosomes can be engulfed by microglia, and through the miR-942-3p/MMP9 axis, they mediate microglia to promote the metastasis of HCC cells[43]. In addition, Hu *et al*[44] found that the overexpression of *SNHG16* promotes the expression of TRAF6 by sponging miR-605-3p, activating NF-κB and thus promoting the development of HCC. Conversely, activated NF-κB can enhance the activity of the *SNHG16* promoter, forming a positive *SNHG16*-NF-κB feedback loop, further exacerbating HCC.

Studies have shown that *SNHG16* regulates a large lncRNA-miRNA-mRNA network in HCC and is closely related to immune cell infiltration, the release of immune regulatory factors, and the expression of chemokines in tumor tissues[45-47]. In addition, researchers have pointed out that the progression of HCC promoted by UBE4B and SEMA3F is regulated by its upstream *SNHG16*/miR-22-3p and *SNHG16*/Let-7c-5p axes[47,48]. Furthermore, Liu *et al*[49] revealed that *SNHG16* can serve as a potential biomarker for poor prognosis in HCC patients. In summary, *SNHG16* is upregulated in HCC and promotes the development of HCC. However, a recent paper presented the opposite view, suggesting that compared to normal liver tissue, the expression of *SNHG16* is reduced in HCC, and the overexpression of *SNHG16* reduces the proliferation of Hep3B and Huh-7 cells by sponging miR-93, inhibiting the development and chemoresistance of HCC[50]. Further research is needed to address the above-mentioned inconsistent findings.

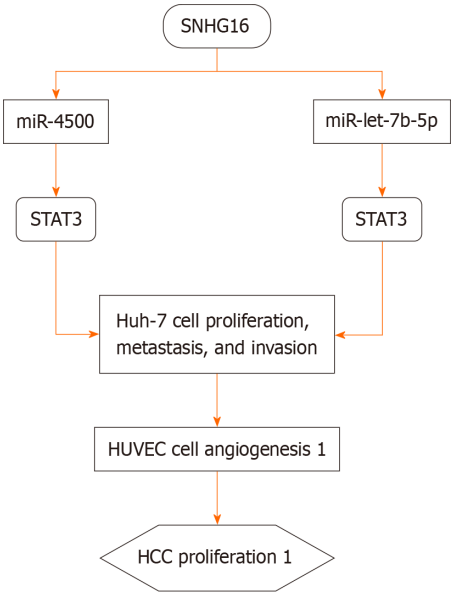

Figure 1 Small nucleolar RNA host gene 16 promotes tumor vascular proliferation. SNHG16: Small nucleolar RNA host gene 16; HCC: Hepatocellular carcinoma.

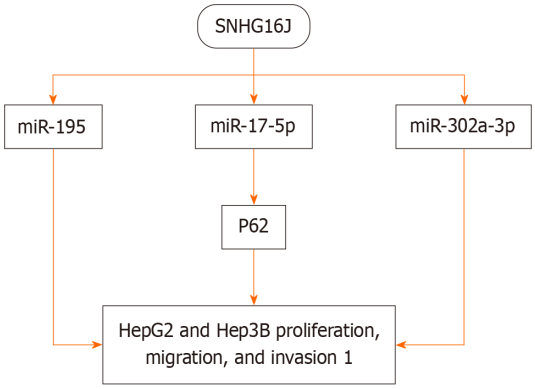

Figure 2 Downregulation of small nucleolar RNA host gene 16 inhibits proliferation, migration, and invasion of HepG2 and Hep3B cells *via* multiple mechanisms. SNHG16: Small nucleolar RNA host gene 16.

SNHG16 and pancreatic cancer

Pancreatic cancer (PC) is one of the most severe malignant tumors in the digestive system. Due to its poor prognosis, the number of PC-related deaths (466000 cases) is almost equivalent to the number of cases (496000 cases), making it the seventh leading cause of cancer death in both genders[2]. Similar to EC, the incidence of PC in high human development index (HDI) countries is 4 to 5 times higher than that in low HDI countries[2]. Studies have shown that *SNHG16* expression is regulated in PC tissue, and the overexpression of *SNHG16* is closely related to patient survival rate, cancer cell differentiation, TNM stage, and lymph node invasion[37]. Altering the expression of *SNHG16* can inhibit the proliferation, migration, invasion, and tumor-forming ability of AsPC-1 cells through miR-218-5p[51], and suppress lipogenesis in AsPC-1 and PANC-1 cells through the miR-195/SREBP2 axis[52]. The overexpression of *SNHG16* is closely related to the resistance of PC cells to gemcitabine. *SNHG16* can interact with EZH2, inhibiting the expression of Smad4 by EZH2 binding to the *Smad4* promoter[53]. The downregulation of SMAD4 reduces its ability to inhibit AKT phosphorylation, thus promoting the resistance of PC cells to gemcitabine[54]. Collectively, all these lines of evidence suggest that *SNHG16* may play a key role in the development of PC and can even be regarded as a marker for poor prognosis in PC.

SNHG16 and gastric cancer

Gastric cancer (GC) remains a significant malignancy worldwide, being the fifth most common malignant tumor (5.6%, over 1000000 new cases) and the fourth leading cause of cancer-related deaths (7.7%, about 769000 deaths)[2]. GC has different characteristics in different parts of the world. The highest age-standardized incidence rate was observed in East Asia, followed by Central and Eastern Europe[2]. Many studies have shown that the expression of *SNHG16* is significantly related to the depth of invasion, lymph node metastasis, TNM stage, histological differentiation, and expression of PTBP1 in GC[55,56]. Inhibiting the expression of *SNHG16* can significantly inhibit GC cell migration and invasion, and

cause cell arrest in the G1 phase[42,43]. In addition, inhibiting the expression of *SNHG16* can reduce the expression of c-Myc and affect the formation of p27/cyclin D1/CDK6, p53/cyclin E1, and cyclin A2/CDK2 complexes[55]. Many GC patients develop resistance to 5-fluorouracil (5-Fu), which is more susceptible to damage than the original GC cells. In GC, blocking the *SNHG16*/miR-506-3p-PTBP1 axis can effectively limit the growth of 5-Fu-resistant GC cell-derived xenograft tumors under 5-Fu treatment[56,57]. PTBP1 stabilizes glycolytic mRNAs by directly binding to the 3′-untranslated region, while *SNHG16* promotes EMT by downregulating the WNT signaling pathway and inhibiting the expression of DKK3[58]. In addition, *SNHG16* activated by CTCF can regulate the proliferation, migration, invasion, and apoptosis of gastrointestinal stromal tumor cells through the miR-128-3p/CASC3 axis[59]. Another study also showed that *SNHG16* can mediate the upregulation of JAK2 and STAT3 by sponging miR-135a, affecting the proliferation, invasion, and apoptosis of GC cells, and *SNHG16* may be regulated by p-STAT3 directly or indirectly[60]. In summary, *SNHG16* is closely related to the occurrence and development of GC and may become a potential marker for poor prognosis in GC.

SNHG16 and CRC

In 2020, there were over 1.9 million new cases of CRC worldwide, with 935000 deaths, ranking third in incidence after breast cancer in women and lung cancer, and second in mortality, close to lung cancer[2]. Increasing evidence indicates that the expression level of *SNHG16* is positively correlated with advanced TNM stage, distant metastasis, and shortened overall survival time in CRC[61-63]. *SNHG16* is mainly present in the cytoplasm, suggesting that *SNHG16* functions as a ceRNA to regulate the activity of multiple miRNAs and target genes. Li *et al*[62] revealed that *SNHG16* is associated with the malignancy and poor prognosis of CRC by sponging miR-200a-3p. Tan *et al*[64] demonstrated that *SNHG16* promotes the proliferation of CRC cells by upregulating its target gene *ABCB1* through interaction with miR-214-3p. It has also been found that *SNHG16* promotes the progression of CRC by activating the expression of USP22 *via* sponging miR-132-3p[65,66]. Ke *et al*[67] discovered that *SNHG16* supports the growth of colon cancer cells by targeting the miR-302a-2p/AKT axis. Chen *et al*[47] showed that the expression level of *SNHG16* in cancerous tissue is higher than that in matched normal tissue and is positively correlated with CRC grade. Moreover, *SNHG16* promotes the proliferation, migration, and EMT of CRC cells *via* the miR-124-3p/MCP-1 axis[48]. Bioinformatics analysis also led to the same conclusion, indicating that *SNHG16* plays an important role in CRC[50,53]. Recent studies have further established the close relationship between *SNHG16* and autophagy in CRC[68,69].

The expression of *SNHG16* is also activated by other proteins such as c-Myc. Christensen *et al*[70] found that in CRC, the expression of *SNHG16* is determined by Wnt-regulated transcription factors, and the inhibition of β-catenin reduces the expression of *SNHG16* and c-Myc. Additionally, the inhibition or overexpression of c-Myc can respectively decrease or increase the expression of *SNHG16*. In another study, Xiang *et al*[71] for the first time discovered a positive feedback loop of *SNHG16*-YAP1/TEAD1 in CRC cells. *SNHG16*, as a ceRNA, physically binds to miR-195-5p, further regulating the expression of YAP1 and promoting tumor progression. YAP1 binds to TEAD1 to form a YAP1/TEAD1 complex, which in turn binds to two sites on the *SNHG16* promoter, activating the transcription of *SNHG16*[71]. The mechanism of *SNHG16*'s involvement in CRC is shown in Figure 3.

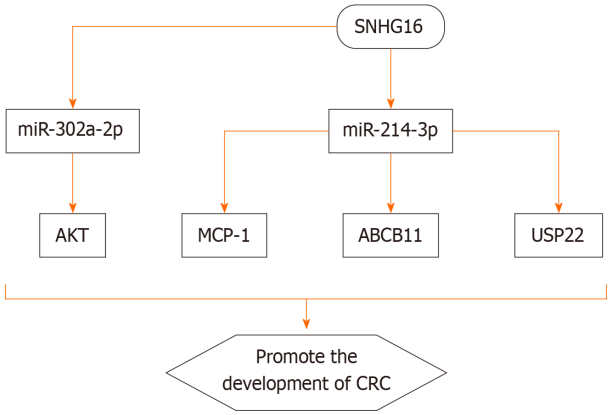

Figure 3 Mechanism of small nucleolar RNA host gene 16's involvement in colorectal cancer. SNHG16: Small nucleolar RNA host gene 16; CRC: Colorectal cancer.

The polymorphisms of *SNHG16* are significantly associated with susceptibility to CRC. The A>G variant at the rs7353 locus of the *SNHG16* gene is associated with a reduced susceptibility to CRC. However, the G>A variant at the rs8038 locus and the A>G and G>A variants at the rs15278 locus may increase the susceptibility to CRC[72].

CONCLUSION

An increasing number of studies indicate that the occurrence of tumors is caused by a combination of genetic and

environmental factors. The focus of this editorial is on the molecular mechanisms underlying the roles of the lncRNA *SNHG16* in the development and progression of digestive system cancers (Figure 3). Existing data suggest that *SNHG16* is closely related to the proliferation, migration, invasion, apoptosis, and poor prognosis of EC, HCC, PC, GC, and CRC. Moreover, *SNHG16* is positively correlated with clinical stage and lymph node metastasis of digestive system cancers. In terms of mechanisms of action of *SNHG16* in digestive system cancers, there are mainly four aspects: (1) Many transcription factors, including CTCF, c-Myc, NF-κB, STAT3, and TEAD1, are positively correlated with *SNHG16*; (2) *SNHG16* directly controls the expression of downstream target genes such as *DKK3*; (3) *SNHG16* can bind and recruit EIF4A3 to regulate the expression of RhoU by enhancing the stability of *RhoU* mRNA, and *SNHG16* can also bind to EZH2 and recruit EZH2 to the promoter of *Smad4*, thereby inhibiting the expression of Smad4; and (4) *SNHG16* can compete with miRNAs, regulate the expression of downstream target genes, and activate different signaling pathways. However, these studies are only preliminary, and the expression levels of *SNHG16* in body fluids and chemical stability have not yet been clarified. In addition, the clinical application of *SNHG16* as a biomarker in digestive system cancers needs further research in the future.

FOOTNOTES

Author contributions: Yang TF and Li XR contributed equally to this work as co-first authors; Kong MW provided crucial suggestions and guidance for manuscript writing; Yang TF wrote the manuscript; Li XR reviewed and revised the manuscript; and all authors read and approved the final manuscript.

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