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Clinicopathological and prognostic value of long noncoding RNA SNHG7 in cancer patients: a meta-analysis

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Abstract. – OBJECTIVE: Recent studies have provided evidence that long noncoding RNA SNHG7 is highly expressed and associated with poor clinical outcomes in cancer patients. The meta-analysis is aimed to evaluate the prognostic value of SNHG7 across various cancers.

MATERIALS AND METHODS: Eligible studies about prognosis and clinicopathological features of SNHG7 expression in all kinds of tumors were collected by searching the databases of PubMed, Web of Science, Embase, Cochrane Library from inception through August 13, 2020. Odds ratios (ORs) and hazard ratios (HRs) with 95% confidence intervals (CIs) from eligible studies were extracted and pooled to investigate the association between SNHG7 and survival or clinicopathology by STATA 16.0 software.

RESULTS: A total of 13 studies enrolling 1029 cancer patients met the inclusion criteria in this meta-analysis. Based on the results, over-expressed SNHG7 was associated with deeper tumor invasion (OR: 2.76; 95% CI: 1.98-3.86; *p*: 0.000), earlier lymphatic metastasis (OR: 4.22; 95% CI: 3.04-5.86; *p*: 0.000), more advanced tumor stage (OR: 3.49; 95% CI: 2.45-4.98; *p*: 0.000) and poor histologic grade (OR: 2.23; 95% CI: 1.33-3.74; *p*: 0.002), but not with sex, age, tumor size and distant metastasis. As for prognosis, patients with high expression of SNHG7 were more likely to have shorter overall survival (OS) (HR: 1.64; 95% CI: 1.38-1.94; *p*: 0.000) and disease-free survival (DFS) (HR: 1.37; 95% CI: 1.09-1.71; *p*: 0.006).

CONCLUSIONS: SNHG7 may serve as a novel biomarker in terms of predicting prognosis and clinicopathological characters in various human cancers.

Key Words:
Long noncoding RNA, SNHG7, Cancer, Prognosis, Meta-analysis.

Introduction

The incidence of cancer is increasing quickly and has become the main leading cause of death worldwide. Based on the global cancer statistics by the International Agency for Research on Cancer (IARC), there were 18.1 million new cancer cases and 9.6 million cancer deaths in 2018[1]. Though the treatment of cancer has made great progress, the prognosis is still unsatisfactory. Against this backdrop, it is crucial and urgent to explore new biomarkers for earlier diagnosing, accurately predicting prognosis, and serving as effective therapeutic targets of different kinds of cancers.

Long noncoding RNAs (lncRNAs), consisting of more than 200 nucleotides in length, are emerging as an important type of noncoding RNAs (ncRNAs), and can't encode protein due to the lack of functional open-reading frames[2]. LncRNAs play a vital role in regulating gene expression through binding to DNA, RNA, or proteins[3,4]. In particular, the imbalanced expression of lncRNA may be related to cancer occurrence, progression, metastasis, and drug resistance[3,5-7], suggesting that lncRNAs have a great possibility to be valuable markers for cancer patients.

Small nucleolar RNA host gene 7 (SNHG7) is one of the recognized lncRNAs, which is situated on chromosome 9q34.3 and contains 2157 basepairs[8]. Recently, a variety of studies have provided emerging evidence that SNHG7 is over-expression in different malignancy, such as colorectal cancer[9-11], hepatocellular carcinoma[12], gastric cancer[13], pancreatic cancer[14], breast cancer[15], bladder cancer[16], prostate cancer[17,18], cervical cancer[19], neuroblastoma[20,21], contributing to the worse clinicopathological features and poor

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prognosis. However, the results may be untrustworthy in light of the small sample size and single tumor type in each study. Therefore, we conducted a meta-analysis to further discuss the potential of SNHG7 as a prognostic marker in various cancer patients.

Materials and Methods

Data Sources and Search Strategy

Two independent researchers had a search of the databases, including PubMed, Web of Science, Embase, Cochrane Library from inception through August 13, 2020. The search terms included "Small nucleolar RNA host gene 7" or "SNHG7" or "Long noncoding RNA SNHG7", and "cancer" or "carcinoma" or "tumor" or "neoplasm", and "prognosis" or "prognostic". Besides, we also manually searched references from relevant original articles to ensure all eligible studies were included.

Study Selection

The inclusion criteria were (a) patients of cancers were diagnosed by pathology or histology, (b) testing the expression of SNHG7 in patients through specific methods, (c) the patients were divided into "high SNHG7" and "low SNHG7" groups, (d) studies reported the association between SNHG7 and prognosis as well as clinicopathological features, (e) studies provided sufficient data to calculate HR and 95% CIs.

The exclusion criteria were (a) reviews, abstracts, conference papers, case reports, letters, and animal studies. (b) duplicated studies, (c) insufficient data to estimate HR and 95% CIs.

Data Extraction

The process of data extraction was conducted by two researchers independently. Disagreements were resolved by consensus. We used a predefined and structured form to collect the following data from each eligible study: the first author, publication year, study country, recruitment time, cancer type, sample size, sample type, the detection method for SNHG7, the cut-off value of SNHG7 expression level, extract method of HR, endpoints, HR and 95% CI of OS/PFS/DFS extracted directly or calculated based on survival curves, and clinicopathological parameters including age, sex, tumor size, depth of invasion, lymphatic metastasis, distant metastasis, tumor stage, and histologic grade.

Quality Assessment

Quality assessment was based on the Newcastle-Ottawa Assessment scale (NOS). The NOS scale assesses the quality of a study using a star system in three groups: selection, comparability, and exposure/outcome. The NOS ranges from 0 to 9 stars[22]. We judged studies as high-quality studies if the total scores were at least 6 stars.

Statistical Analysis

We performed the meta-analysis of all eligible studies using STATA 16.0 software. HRs with 95% CIs were pooled for assessing the effect of SNHG7 expression on cancer prognosis. ORs with 95% CIs were combined to investigate the association between SNHG7 expression and clinicopathological characteristics in cancer patients. Between-study heterogeneity was evaluated by Cochran's Q and I^2 tests. A random-effect model was utilized for analysis when I^2 value > 50%; otherwise, a fixed-effect model was applied. Publication bias was assessed by Begg's and Egger's tests. The trim-and-fill method was adopted to further evaluate the robustness of pooled effect size when publication bias was detected.

Results

Literature Search and Characteristics of Included Studies

The flow chart for the study selection process is shown in Figure 1. A total of 88 articles were retrieved initially from the described databases. According to the mentioned inclusion and exclusion criteria, 13 articles enrolling 1029 patients were finally included. All included studies were from China. The sample size of each study ranged from 40 to 162. All studies examined the expression level of SNHG7 by qRT-PCR in tumor tissue, while one study sample was from venous blood. There are nine types of cancers in our meta-analysis, including colorectal cancer, hepatocellular carcinoma, gastric cancer, pancreatic cancer, bladder cancer, prostate cancer, breast cancer, neuroblastoma, cervical cancer. The included studies[9-21] all reported the association between SNHG7 expression and OS. Meanwhile, three studies[9,11,12] also reported the correlation of SNHG7 with DFS/PFS. Only five research articles provided HR and 95% CIs

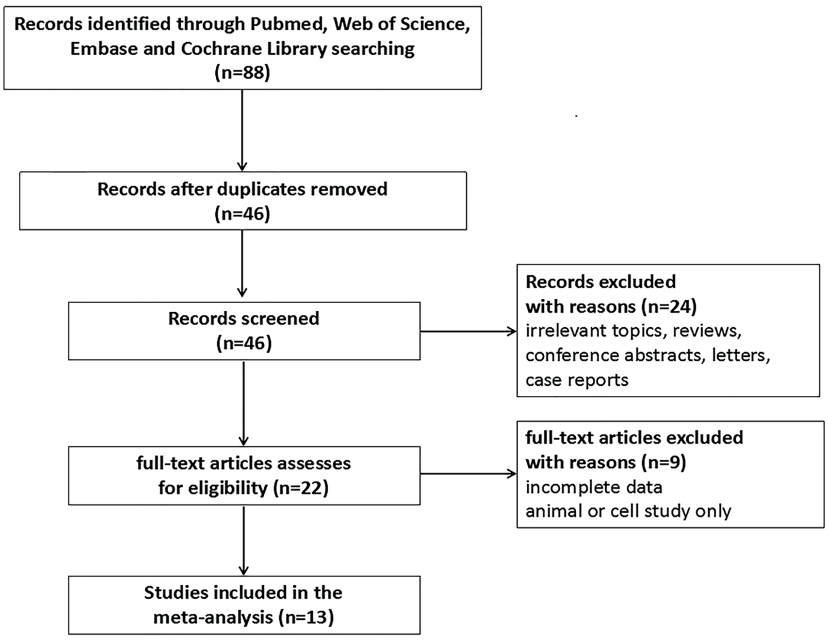

Figure 1. The flow chart of literature search.

for OS directly in the paper and others offered Kaplan-Meier curves for us to extract survival dates using the method published by Tierney et al[23]. The details of included studies were shown in Table I.

The Association Between SNHG7 Expression and Clinical-Pathological Features

Depth of Tumor Invasion

Seven studies[9-11,13,15,17,18] with 600 patients were employed to clarify the relationship of SNHG7 expression with tumor invasion depth. A fixed-effect model was conducted considering low heterogeneity (I^2: 31.0%, *p*: 0.192). The pooled results indicated that patients with high SNHG7 expression were prone to have deeper tumor invasion (OR: 2.76; 95% CI: 1.98-3.86; *p*: 0.000, Table II, Figure 2a).

Lymphatic Metastasis

A total of ten studies[9,10,12,14-19,21] involving 726 patients reported the effect of SNHG7 expression on lymphatic metastasis. We used a fixed-effect model to calculate the pooled OR and 95% CI because there was no obvious heterogeneity among these studies (I^2: 19.8%, *p*: 0.261). The results pointed that up-regulated SNHG7 expression in cancer patients was connected to earlier lymph node metastasis (OR: 4.22; 95% CI: 3.04-5.86; *p*: 0.000, Table II, Figure 2b).

Tumor Stage

To summarize the association between SNHG7 expression and tumor stage, we combined the results of OR and 95% CI from seven studies[9,12-14,18-20]. Since between-study heterogeneity was less than 50% and *p*-value more than 0.1 (I^2: 15.5%, *p*: 0.312), a fixed-effect model was utilized. The pooled OR and 95% CI had statistical significance (OR: 3.49; 95% CI: 2.45-4.98; *p*: 0.000, Table II, Figure 2c), which suggested that high SNHG7 expression had a significant influence on the advanced tumor stage.

Histologic Grade

Four studies[9,11,14,19] containing 249 cancer patients revealed that patients with over-expressed SNHG7 had more possibility to develop poor histologic grade (OR: 2.23; 95% CI: 1.33-3.74; *p*: 0.002, Table II, Figure 2d), applying a fixed-effect model with no heterogeneity detected (I^2: 0.00%, *p*: 0.636).

Besides, we also explore the association between SNHG7 expression and other clinical features, such as age, sex, tumor size, and distant metastasis (Table II). However, no significant associations were found.

Table I. Characteristics of included studies in the meta-analysis.

Author	Year	Country	Recruitment time	Cancer type	Sample size	Sample	Outcome	Detection method	Cut-off value	Extract method of HR	NOS score
Li et al[9]	2018	China	2015-2018	Colorectal cancer	53	Tissue	OS DFS	qRT-PCR	NA	Data in paper Survival curves	8
Luo et al[15]	2018	China	NA	Breast cancer	72	Tissue	OS	qRT-PCR	Mean	Survival curves	6
Qi et al[17]	2018	China	2015-2016	Prostate cancer	42	Tissue	OS	qRT-PCR	Mean	Survival curves	7
Shan et al[10]	2019	China	2012-2016	Colorectal cancer	48	Tissue	OS	qRT-PCR	Median	Survival curves	7
Chen et al[16]	2019	China	NA	Bladder cancer	92	Tissue	OS	qRT-PCR	Median	Survival curves	8
Cheng et al[14]	2019	China	2016-2018	Pancreatic cancer	40	Tissue	OS	qRT-PCR	NA	Survival curves	7
Chi et al[21]	2019	China	NA	Neuroblastoma	92	Tissue	OS	qRT-PCR	Median	Survival curves	8
Zeng et al[19]	2019	China	2014-2018	Cervical cancer	60	Tissue	OS	qRT-PCR	NA	Data in paper	7
Jia et al[20]	2019	China	2009-2011	Neuroblastoma	45	Tissue	OS	qRT-PCR	Mean	Survival curves	7
Shen et al[12]	2020	China	2016-2018	Hepatocellular carcinoma	100	Tissue	OS PFS	qRT-PCR	Mean	Survival curves	8
Xia et al[18]	2020	China	2011-2019	Prostate cancer	127	Tissue	OS	qRT-PCR	Median	Data in paper	7
Zhang et al[7]	2020	China	2013-2015	Synchronous colorectal liver metastasis	96	Blood	OS DFS	qRT-PCR	Median	Data in paper	8
Zhang et al[11]	2020	China	2013-2018	Gastric cancer	162	Tissue	OS	qRT-PCR	Median	Data in paper	7

Abbreviations: OS overall survival, DFS disease-free survival, PFS progression-free survival, HR hazard ratio, NOS Newcastle-Ottawa Scale, NA not available, qRT-PCR quantitative reverse transcription polymerase chain reaction.

Table II. Meta-analysis results for the association of over-expressed SNHG7 with clinicopathological parameters.

Clinicopathological parameters	Number of studies	Number of patients	HR (95% CI)	*p*-value	Heterogeneity			Begg	Egger
					I^2 (%)	*p*-value	Model		
Age (≥ 60 y *vs*. < 60 y)	7	359	1.13 (0.79, 1.61)	0.498	0.00	0.779	Fixed	0.368	0.502
Sex (male vs. female)	10	800	0.77 (0.57, 1.04)	0.085	0.00	0.838	Fixed	0.474	0.400
Tumor size (≥ 5 cm *vs*. < 5 cm)	3	201	1.64 (0.93, 2.89)	0.089	26.4	0.257	Fixed	—	—
Depth of invasion (T3/T4 *vs*. T1/T2)	7	600	2.76 (1.98, 3.86)	0.000	31.0	0.192	Fixed	1.000	0.470
Lymphatic metastasis (yes *vs*. no)	10	726	4.22 (3.04, 5.86)	0.000	19.8	0.261	Fixed	0.283	0.133
Distant metastasis (yes *vs*. no)	7	596	2.20 (0.81, 5.94)	0.120	76.8	0.000	Random	0.764	0.866
Tumor stage (III/IV *vs*. I/II)	7	587	3.49 (2.45, 4.98)	0.000	15.5	0.312	Fixed	0.230	0.011
Histologic grade (poorly *vs*. well/moderately)	4	249	2.23 (1.33, 3.74)	0.002	0.00	0.636	Fixed	—	—

Abbreviations: OR odd ratio, 95% CI confidence interval.

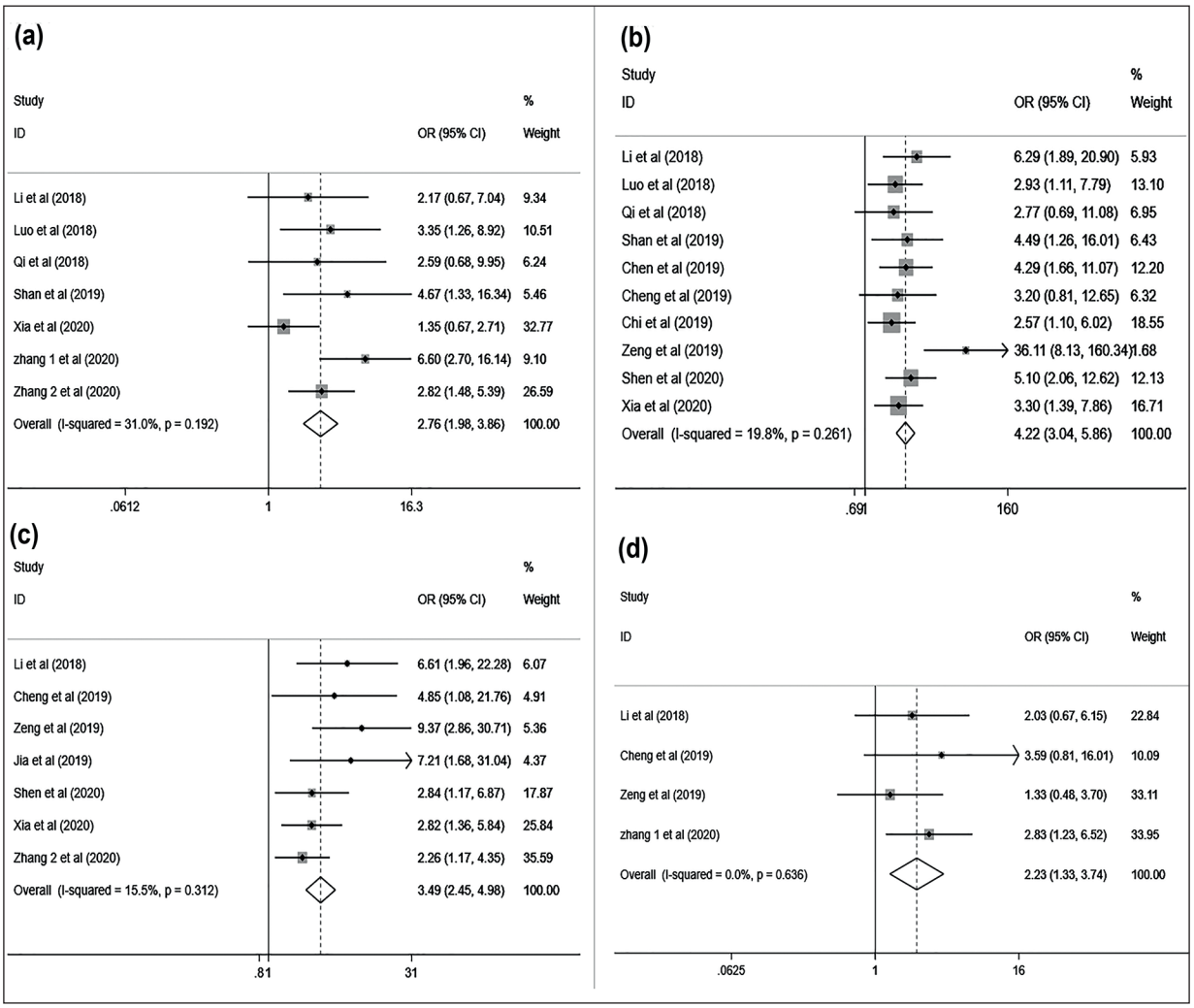

Figure 2. Forest plot for the association between SNHG7 expression and clinicopathological parameters. **(a)** depth of invasion; **(b)** lymphatic metastasis; **(c)** tumor stage; **(d)** histologic grade.

The Association Between SNHG7 Expression and Cancer Prognosis

SNHG7 Expression and Overall Survival (OS)

As is shown in Figure 3, all eligible studies reported the correlation of SNHG7 expression and OS. In light of no existence of heterogeneity (I^2: 0.00%, *p*: 0.487), we adopted a fixed-effect model to combine all results from every included study. The pooled HR and 95% CI had statistical significance (HR: 1.64; 95% CI: 1.38-1.94; *p*: 0.000), which demonstrated that over-expression of SNHG7 in cancer patients were more likely to have the poor OS. Moreover, we discuss the potential value of SNHG7

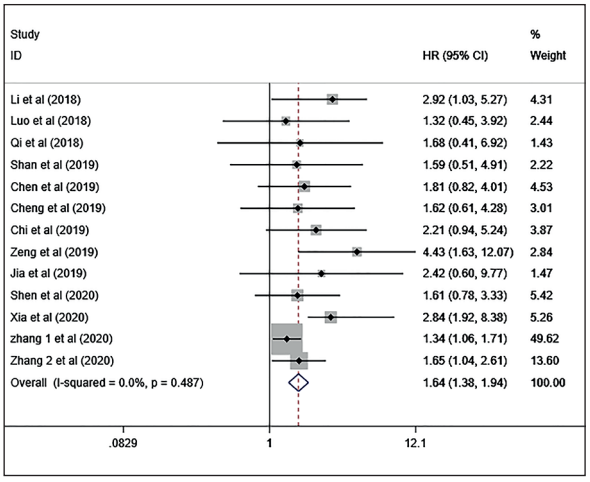

Figure 3. Forest plot for the association between SNHG7 expression and OS.

for OS with subgroup analysis, including cancer type, extract method, sample size, and sample type (Table III, Figure 4). In subgroup of cancer type, up-regulated SNHG7 was significantly related to digestive system cancers (HR: 1.49; 95% CI: 1.23-1.80; *p*: 0.000), reproductive system cancers (HR: 3.00; 95% CI: 1.73-5.18; *p*: 0.000) and neuroblastoma (HR: 2.27; 95% CI: 1.09-4.71; *p*: 0.028), but not associated with breast cancer (HR: 1.32; 95% CI: 0.45-3.90; *p*: 0.615) and urinary system cancers (HR: 1.81; 95% CI: 0.82-4.00; *p*: 0.143). When the studies were classified with extract method of effect size, we concluded that both in the group of date in paper and survival curves, high expression of SNHG7 may lead to worse OS (date in paper: HR: 2.06; 95% CI: 1.37-3.10; *p*: 0.001; survival curves: HR: 1.74; 95% CI: 1.24-2.45; *p*: 0.001). For subgroup of sample size, three groups with different amount of sample size all apparently linked with poor OS (<80: HR: 2.21; 95% CI: 1.48-3.30; *p*: 0.000; 80-100: HR: 1.44; 95% CI: 1.16-1.78; *p*: 0.001; >100: HR: 1.92; 95% CI: 1.30-2.83; *p*: 0.001). As for the subgroup of sample type, most studies detected the expression level of SNHG7 by tumor tissue, while only one by blood. Either group of tissue or blood, patients with high SNHG7 had a shorter OS than those with low SNHG7 (tissue: HR: 1.99; 95% CI: 1.57-2.53; *p*: 0.000; blood: HR: 1.34; 95% CI: 1.06-1.71; *p*: 0.016).

SNHG7 Expression and Disease-Free Survival (DFS)/Progression-free Survival (PFS)

As can be seen from Figure 5, two studies[9,11] consisting of 149 patients proved that SNHG7 had the potential for being a prognostic biomarker in terms of DFS in human cancers (HR: 1.37; 95% CI: 1.09-1.71; *p*: 0.006). One study from Shen et al[12] reported that SNHG7 expression did not influence PFS (HR: 1.57; 95% CI: 0.86-2.87; *p*: 0.142).

Publication Bias

Funnel plots, Begg's and Egger's tests were performed to assess the potential publication bias. With respect to OS, Begg funnel plot presented in Figure 6 seemed to be asymmetrical and the results from Begg's test (*p*: 0.855) and Egger's test (*p*: 0.018) revealed that publication bias may exist in the meta-analysis of OS. For that reason, we conducted a trim-and-fill method using a fixed-effect model[24]. After filling seven fictitious unpublished articles, the funnel plot appeared to be symmetrical, and the pooled HR and 95% CI stayed stable (HR: 1.455; 95% CI: 1.18-1.80; *p*: 0.001). Besides, we also evaluated the publication bias in clinicopathological features, containing age, sex, depth of invasion, lymphatic metastasis, distant metastasis, and tumor stage (Table II). Considering the small

Table III. Overall and subgroup analysis of SNHG7 expression for OS.

Subgroup	Number of studies	Number of patients	HR (95% CI)	*p*-value	Heterogeneity		
					I^2 (%)	*p*-value	Model
OS	13	1029	1.64 (1.38, 1.94)	0.000	0.00	0.487	Fixed
Cancer type							
Digestive system	6	499	1.49 (1.23, 1.80)	0.000	0.00	0.604	Fixed
Breast cancer	1	72	1.32 (0.45, 3.90)	0.615	—	—	—
Reproductive system	3	229	3.00 (1.73, 5.18)	0.000	0.00	0.534	Fixed
Urinary system	1	92	1.81 (0.82, 4.00)	0.143	—	—	—
Neuroblastoma	2	137	2.27 (1.09, 4.71)	0.028	0.00	0.914	
Extract method							
Data in paper	5	498	2.06 (1.37, 3.10)	0.001	61.8	0.033	Random
Survival curves	8	531	1.74 (1.24, 2.45)	0.001	0.00	0.997	Random
Sample size							
< 80	7	360	2.21 (1.48, 3.30)	0.000	0.00	0.669	Fixed
80-100	4	380	1.44 (1.16, 1.78)	0.001	0.00	0.641	Fixed
> 100	2	289	1.92 (1.30, 2.83)	0.001	33.2	0.221	Fixed
Sample							
Tissue	12	933	1.99 (1.57, 2.53)	0.000	0.00	0.853	Fixed
Blood	1	96	1.34 (1.06, 1.71)	0.016	—	—	—

Abbreviations: HR hazard ratio, 95% CI confidence interval.

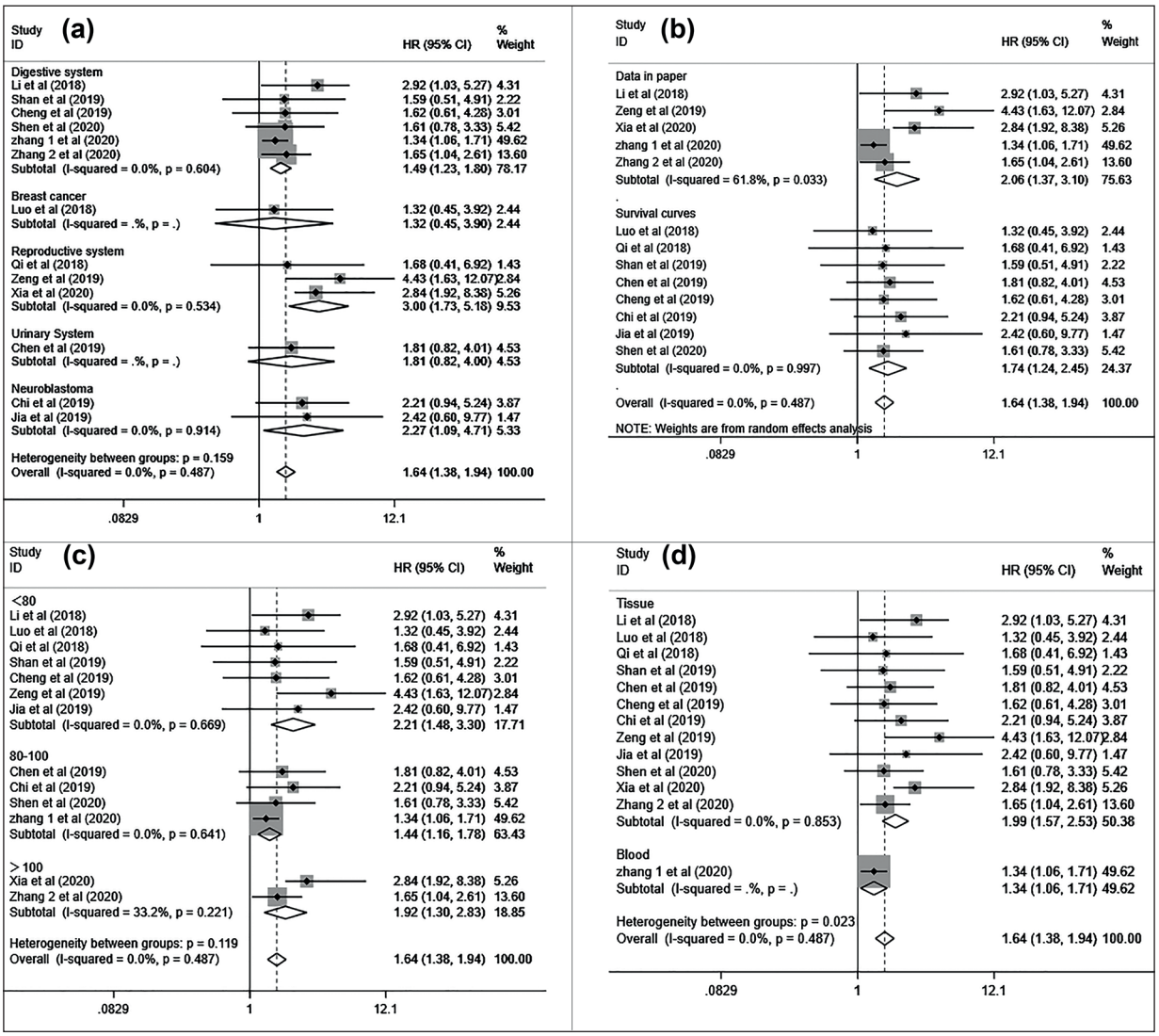

Figure 4. Forest plot of subgroup analysis for the association between SNHG7 expression and OS. **(a)** cancer type; **(b)** extract method; **(c)** sample size; **(d)** sample type.

number of eligible studies in terms of tumor size and histologic grade, Begg's and Egger tests were not carried out.

Discussion

LncRNAs was considered to be "junk gene" for a long time in the past. Currently, more and more evidence has proved that lncRNAs play a great role in different physiological and pathological processes by regulating gene expression, especially in carcinoma through serving as oncogenes or tumor suppressor genes[25]. And with the substantial development of sequencing technology, numerous lncRNAs are identified to have a connection with cancer, such as GHET1[26], H19[27], HOTAIR[28], and so on. Similarly, many studies have demonstrated that SNHG7 may be a promising biomarker for cancer diagnosis and prognosis.

The mechanisms behind how lncRNA SNHG7 modulates the development of human cancers are still widely investigated. Yang et al[29] pointed that SNHG7 could speed up the progression of hepatocellular carcinoma via down-regulating miR-122-5p and strengthening the stability of RPL4. In colorectal cancer proliferation and metastasis, SNHG7 could function as a competing endogenous RNA (ceRNA) for miR-34a and regulate the expression of GALNT7, modu-

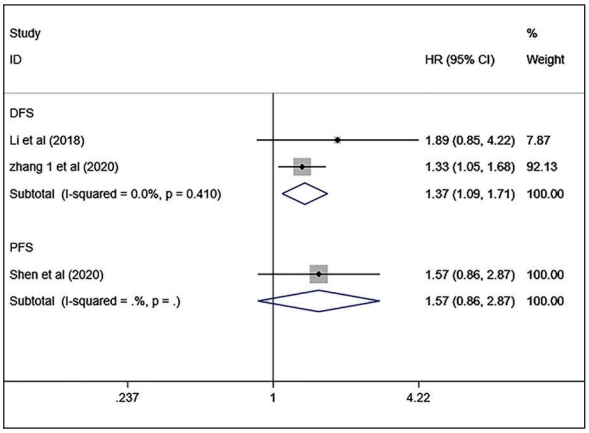

Figure 5. Forest plot for the association between SNHG7 expression and DFS with PFS.

lating PI3K/Akt/mTOR pathway[9]. Additionally, SNHG7 played an important role in other types of cancers by various mechanisms, for example regulating cyclin D1 expression in prostate cancer[17], promoting epithelial-to-mesenchymal transition (EMT) in osteosarcoma, breast cancer, or gastric cancer[13,30,31], over-expression of STAT2 by down-regulating miR-653-5p in neuroblastoma[21], and so on. In the future, more attention should be paid to explore the underlying mechanisms for SNHG7 expression and different carcinomas.

Our meta-analysis including 13 original studies was aimed to summarize the prognostic and clinicopathological value of SNHG7 expression in different kinds of human cancers. It was shown that patients with "high SNHG7" had more possibility to suffer from shorter OS and DFS compared with "low SNHG7". The association between the high expression level of SNHG7 and PFS was not observed possibly because of the limited sample size. Subgroup analysis was conducted to further analyze the association between SNHG7 and OS in different conditions. In terms of cancer type, all other types of cancers included in this analysis had similar outcomes except for breast cancer and bladder cancer. The reason for it may be due to the differences in sex, tumor cell, and the number of studies. As for the subgroup of extract method, sample size, and sample type, all pooled results suggested that over-expressed SNHG7 was related to poor OS. At the same time, we also explore the relationship of SNHG7 expression with various clinical characters. Cancer patients in the group of high expression of SNHG7 were prone to have deeper tumor invasion, positive lymphatic metastasis, more advanced tumor stage, and worse histologic grade. SNHG7 expression had no relevance with several other clinical features containing age, sex, tumor size, and distant metastasis. Taken all these results into consideration, the lncRNA SNHG7 has great potential to be a marker for prognosis in cancer patients.

Some limitations should be acknowledged in our study here. First, all included studies in the meta-analysis were from China, indicating that the results could not represent other countries and caused potential publication bias. Second, the majority of results of HRs and 95% CIs were extracted from survival curves indirectly and may lead to inexactness. Third, the number of sample sizes and studies was fairly small, and a small part of cancers was involved in the analysis. Fourth, the cut-off value for "high or

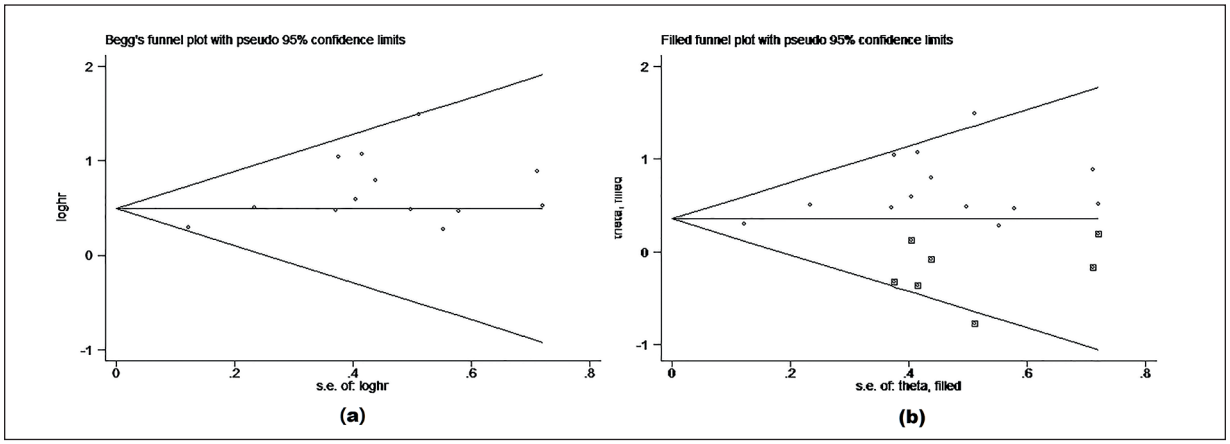

Figure 6. The detection of publication bias for meta-analysis of OS. **(a)** Begg funnel plot; **(b)** filled funnel plot.

low" SNHG7 expression and treatment for different kinds of cancer patients had differences in each study, which may affect the relevant outcomes.

Conclusions

Taken together, our meta-analysis provides evidence that SNHG7 has a significant prognostic value acting as a novel and promising biomarker for multiple human cancers. In the future, well-designed, multi-center, large-scale researches are still needed to confirm our results.

Conflict of Interest

The Authors declare that they have no conflict of interests.

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