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Aberrant SNHG expression predicts poor prognosis in esophageal cancer using meta-analysis and bioinformatics analysis

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Abstract

Background Small nucleolar RNA host gene (SNHG) family were reported involved in various biological processes and may be used as a promising prognostic marker in esophageal cancer (EC). A meta-analysis was performed to investigate the relationship between SNHG expression and prognosis of EC in this study.

Methods Relevant databases were browsed to obtain suitable publications. Hazard ratio (HR) with 95% confidence interval (CI) were extracted to explore the association between SNHG expression and EC prognosis. Odds ratio (OR) with 95%CI were extracted to assess the association between SNHG expression and other clinicopathological parameters. Sensitivity analysis and publication bias were performed to explore the reliability and robustness of the results. Bio-informatics has been explored in order to confirm our conclusions more comprehensively.

Results 16 studies comprising 1229 patients were enrolled. The results showed that increasing SNHG expression indicated worse overall survival (HR: 1.392, 95%CI = 0.876–1.908). SNHG2, SNHG5, and SNHG12 were down-regulated, while other SNHGs were up-regulated in EC. In populations with low expression of SNHG2, SNHG5, and SNHG12, increasing SNHG expression predicted a favorable cancer prognosis (HR: 0.511, 95%CI = 0.322-0.700). Conversely, in populations with high expression of other SNHGs, SNHG expression indicated poor prognosis (OR: 2.340, 95%CI = 1.744–2.936). Elevated SNHG expression also implied advanced TNM stage (OR 1.578, 95%CI = 1.273–1.956) and lymph node metastasis (OR: 1.533, 95%CI = 1.205–1.950).

Conclusion Increased expression of SNHG2, SNHG5, and SNHG12, and decreased expression of other SNHGs tended to have a favorable prognosis in patients with EC. These findings suggest that SNHG may serve as a prognostic marker and therapeutic target for EC.

Keywords SNHG, Esophageal squamous cell carcinoma, Prognosis, Meta-analysis, Bioinformatics

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Core tip

The meta-analysis showed Increased expression of SNHG2, SNHG5, and SNHG12, and decreased expression of other SNHGs tended to have a favorable prognosis in patients with EC. These findings suggest that SNHG may serve as a prognostic marker and therapeutic target for EC.

Introduction

Esophageal cancer (EC) is a highly aggressive malignancy that arises from the lining of the esophagus, the muscular tube that connects the throat to the stomach. It is the eighth most common cancer worldwide and the sixth leading cause of cancer-related deaths, with an estimated 604,000 deaths in 2022 [1]. EC patients has a poor prognosis, with a five-year survival rate of only 20–30%, and its occurrence and development are influenced by a complex network of genetic and environmental factors [2, 3]. Over the past decade, advances in high-throughput sequencing have enabled the identification of numerous small molecules that play a significant role in the occurrence and development of EC. Among these molecules, non-coding RNA has emerged as a particularly significant contributor [4].

Non-coding RNA (ncRNA) refers to a class of RNA molecules that do not encode proteins but perform a variety of regulatory functions in the cell [5]. Non-coding RNAs can be broadly classified into two categories: long non-coding RNAs (lncRNAs) and short non-coding RNAs [6]. LncRNAs are RNA molecules that are longer than 200 nucleotides and do not code for proteins [7, 8]. They are transcribed from DNA sequences and play diverse roles in gene expression regulation, chromosome structure organization, and cellular processes such as proliferation, differentiation, and apoptosis [9]. The small nucleolar RNA host gene (SNHG) family is a group of lncRNA molecules that are transcribed from intronic regions of protein-coding genes. The SNHG family includes at least 128 members, and each member is named SNHG followed by a number [10, 11]. These RNA molecules are typically transcribed by RNA polymerase II and undergo post-transcriptional modifications, such as the splicing, polyadenylation, and cap methylation [12].

Recent studies have shown that members of the SNHG family are involved in the occurrence and development of several cancers, including EC [13, 14]. Dysregulation of SNHG family members has been shown to contribute to the pathogenesis of EC by promoting cell proliferation, migration, invasion, and other malignant behaviors. In addition, some SNHG family members have been identified as prognostic markers and potential therapeutic targets for EC [15]. Due to the small sample size of individual studies and the conflicting conclusions among some of them, the purpose of this study is to conduct a meta-analysis to comprehensively investigate the correlation between SNHG expression levels and tubular prognosis.

Materials and methods

Obtain the appropriate publications

In February 2023, based on the Preferred reporting items for systematic reviews and meta-analyses (PRISMA) reporting guidelines [16, 17], six relevant databases including PubMed, Embase, Web of Science, Cochrane Library, Google Scholar and China National Knowledge Infrastructure (CNKI) were searched to obtain related literature. The search strategy of this study is conducted using relevant keywords and MeSH terms as follows: ("long non-coding RNA small nucleolar RNA host gene" OR "small nucleolar RNA host gene" OR "lncRNA SNHG" OR "lnc SNHG" OR "SNHG") AND ("esophageal squamous cell carcinoma" OR "EC" OR "esophageal cancer") OR ("prognosis" OR "survival" OR "survival outcome"). The search may also include a manual search of the reference lists of relevant studies and reviews.

Inclusion and exclusion criteria

The primary literature meeting the following criteria would be included in this meta-analysis: (1) investigate the correlation between SNHG family and prognosis of EC. (2) report hazard ratios (HRs) or odds ratios (ORs) with 95% confidence intervals (CIs). (3) were published in English. (4) The experimental subjects are human beings. Articles meeting the following criteria would be excluded: (1) Duplicate publications. (2) reviews, case reports, letters, or conference abstracts. (3) Insufficient or unavailable data. (4) Experimental subjects are animals.

Quality assessment

The quality of eligible studies was assessed by two researchers via using the Newcastle-Ottawa Scale (NOS) score, which consists of three main components: selection of study groups, comparability of study groups, and ascertainment of outcomes [18]. The quality assessment process involves evaluating the risk of bias in each study based on criteria such as selection bias, performance bias, detection bias, attrition bias, and reporting bias. The NOS score were range from 0 to 9, studies that are of low quality (less than 6) or have a high risk of bias are excluded from the meta-analysis.

Data extraction

Two researchers independently extracted relevant information for each study included in this meta-analysis. The

information included the first author's name, year of publication, expression level of SNHG, sample size, patient's country, SNHG expression level detection method, reference gene and cut-off value. Additionally, the number of events and totals for each clinicopathological parameter were recorded. For survival data, such as overall survival and disease-free survival, HR values and 95% CIs were obtained. In cases where the HR value was not provided but the survival curve was available, the HR value and 95% CI were obtained using the Engauge software curve fitting method.

Sensitivity analysis and publication bias

The sensitivity analysis was conducted to assess the robustness and validity of the results in this research, identify potential sources of bias and heterogeneity, test the sensitivity of the results to different assumptions or methods, provide a more comprehensive and nuanced understanding of the correlation between SNHG family and prognosis of EC. Publication bias was performed by using visual inspection of funnel plots and statistical tests based on the Bgger's regression test. Publication bias occurs when studies with positive results are more likely to be published than studies with negative results, leading to an overestimation of the effect size in the meta-analysis.

Bio-informatics analysis

The GEPIA database (http://gepia.cancer-pku.cn/) was explored to uncovered the difference of SNHG expression in tumor tissue and adjacent normal tissue.

Statistical analysis

The software of the Stata SE 14.0, Revman 5.4.0 and Enguage 4.0 were applied to conducted the statistical analysis of this meta-analysis. Pooling HR with 95%CI was conducted to investigate the association between SNHG expression and the survival outcome of EC patients including OS and DFS. Pooling OR with 95%CI was performed to evaluate the relationship between SNHG expression and clinicopathological parameter including TNM stage, LNM, DM, tumor size, invasion depth, histological grade, age and gender. For less heterogeneous results ($I^2 < 50$, $P\text{-}value > 0.05$), the fixed effects model was implemented. For highly heterogeneous results ($I^2 \geq 50$, $P\text{-}value < 0.05$), the random-effects models were performed, and subgroup analyzes were carried out to explore sources of heterogeneity.

Results

Collection of appropriate publications

After comprehensively searching of six databases based on the PRISMA reporting guide, 125 publications were obtained firstly. 18 papers were excluded for replication, 79 studies were excluded for not exploring the association between SNHG expression and EC prognosis, 3 animal experimental articles were abandoned, 1 meta-analysis and 8 articles with insufficient data were discarded. 16 appropriate original research comprising 1229 patients were finally enrolled [14, 19–33] (Fig. 1).

Basic characteristics of included documents

All study patients are from China, time of publication are range from 2017 to 2022 year, number of cases per study were range from 20 to 128, based on the expression level of SNHG, patients were categorized into two groups: those with high SNHG expression and low SNHG expression, the expression level of SNHG was determined using real-time fluorescent quantitative PCR (qRT-PCR) with GAPDH as the reference gene (Table 1). The NOS score were various 6–9 (Table 2). The cut-off value for categorizing patients into high or low expression groups was set at either the mean or median value. SNHG2 and SNHG5 were down-regulated in EC tissues compare to adjacent normal tissue. SNHG1, SNHG6, SNHG8, SNHG16, SNHG17 and SNHG20 were reported up-regulation in EC tissue compare to adjacent normal tissue, moreover, several studies have reported conflicting findings regarding the expression level of SNHG12 in EC, with some reporting high expression and others reporting low expression of the gene.

Association between SNHG expression and the prognosis of EC

Ten researches with 816 patients had explored the relationship between SNHG expression and EC prognosis [14, 19, 20, 22–24, 27, 29, 31, 34], among these studies, SNHG2, SNHG5 and SNHG12 are down-regulated in EC tissues [22, 23, 27], while the other SNHG family members are up-regulated. In EC population with low SNHG expression (SNHG2, SNHG5 and SNHG12), high SNHG expression predicts well prognosis of EC (HR: 0.511, 95%CI: 0.322–0.700), conversely, in the EC patients with high expression of SNHG, increasing SNHG expression indicating poor EC prognosis (HR: 2.340, 95%CI: 1.744–2.936). The result of pooling all HR value with 95%CI of these 10 studies failed to showed the significant relationship between SNHG expression and the prognosis of EC patients (HR: 1.392, 95%CI: 0.876–1.908) (Fig. 2). Considering the large heterogeneity of combined HR values ($I^2 = 75.4$, $P\text{-}value < 0.0001$), the subgroup based on cut-off value (mean and median), HR extraction (Direct extraction and indirect extraction), Analysis method (Multivariate analysis and Univariate analysis), number of patients (less than 60 and not less than 60 patients of single study), Follow-up (less than or not less than 60

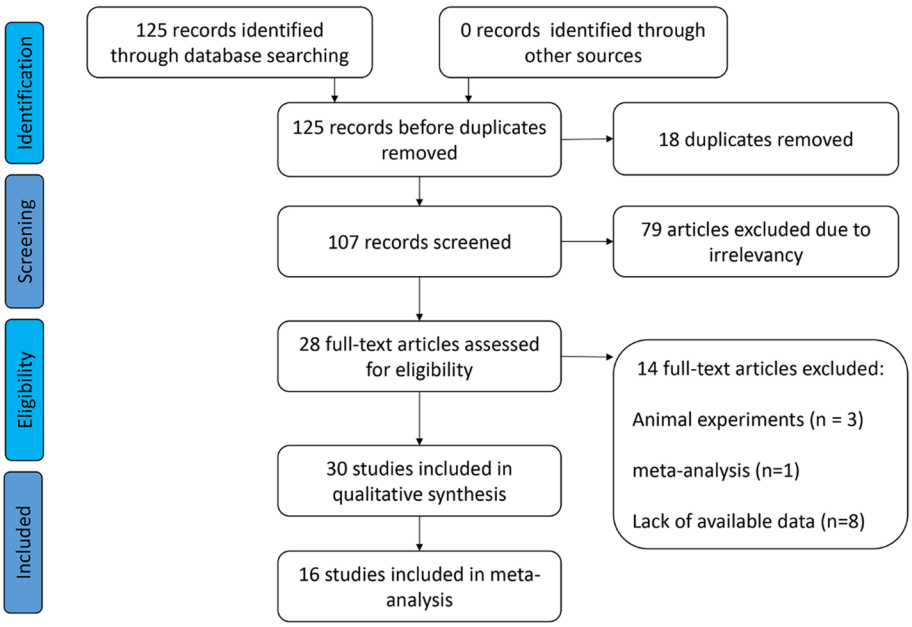

Fig. 1 The flow diagram of the eligible studies

month) and NOS score (NOS score is 9 or NOS score less than 9) was performed. The marked positive correlation between increasing SNHG expression and bad EC prognosis in the subgroup of direction extraction (HR: 2.674, 95%CI: 1.791–3.557), Multivariate analysis (HR: 2.674, 95%CI: 1.791–3.557), less than 60 patients (HR: 2.492, 95%CI: 1.484–3.501), not less than 60 follow-up month (HR: 2.045, 95%CI: 1.111–2.980) and the subgroup of NOS score is 9 (HR: 2.551, 95%CI: 1.804–3.299). but the correlation between SNHG expression and EC prognosis in another subgroup is not significant. In addition, one study assessed the relation between SNHG expression and disease-free survival (DFS), the result indicated that increased SNHG1 expression predicted worse DFS in EC (HR: 3.016, 95%CI: 1.294–4.645) (Table 3).

Association between SNHG expression and TNM stage

Thirteen studies with 1044 patients were enrolled to evaluate the association between SNHG expression and TNM stage in EC patients [14, 20, 21, 23–25, 27–33], and the pooling OR with 95%CI shows that high SNHG expression manifested advanced TNM stage (OR: 1.578, 95%CI: 1.273–1.956), due to the conflicting expression level of different SNHG in EC tissues and various study quality, the subgroup analysis based on SNHG subgroup (patients with high and low SNHG expression) and NOS score (NOS score is 9 or less than 9), the result indicated that in the high SNHG expression subgroup, increased SNHG expression implied advanced TNM stage (OR: 1.779, 95%CI: 1.404–2.253), and in the low SNHG expression subgroup, increasing SNHG expression indicated well TNM stage (OR: 0.820, 95%CI: 0.336–2.001) (Fig. 3). Moreover, high expression of SNHG is associated with advanced TNM stage, regardless of whether the subgroup has a NOS score of 9 or lower than 9 (Table 4).

Association between SNHG expression and LNM

Eleven researches including 878 cases tried to explore the correlation between SNHG expression and the lymph node metastasis in EC patients [14, 21, 23, 24, 26–32]. The significant positive relationship between high SNHG expression and easier lymph node metastasis was uncovered (OR: 1.533, 95%CI: 1.205–1.950). The result based on subgroup analysis showed that, elevated SNHG expression manifested easier lymph node metastasis in the subgroup of patients with high SNHG expression (OR: 1.671, 95%CI: 1.282–2.178) (Fig. 4), the study of NOS score is 9 (OR: 1.730, 95%CI: 1.157–2.585) or less than 9 (OR: 1.432, 95%CI: 1.061–1.934) (Table 4).

Association between SNHG expression and tumor size

Twelve papers comprising 916 patients were enrolled to assess the relation between SNHG expression and tumor size [10, 14, 20, 21, 23, 24, 27–30, 32, 33], the result implied that increasing SNHG expression demonstrated large tumor size (OR: 1.419, 95%CI: 1.134–1.777) (Fig. 5), based on the subgroup analysis, we found that increasing SNHG expression manifests bigger tumor size in the patients with high SNHG

Table 1 Basic characteristics of included studies ($n = 16$)

lnc RNA SNHG	Author and year	Number of patients	Country	SNHG expression	Reference gene	cut-off value	Survival outcome	HR extraction	95% CI	Analytical method	Follow up month	NOS score
SNHG1	Luo DB 2020 [13]	42	China	upregulated	GAPDH	mean	OS	paper	3.342 (1.951–5.064)	Multivariate analysis	60	9
							DFS	paper	3.016 (1.294–4.645)	Multivariate analysis	60	9
SNHG1	Li HM 2020 [18]	53	China	upregulated	GAPDH	mean	OS	Survival curve	1.61 (0.66–3.95)	Univariate analysis	60	7
SNHG1	Chen Y 2021 [19]	36	China	upregulated	GAPDH	mean	OS	survival curve	2.37 (1.02–5.49)	Univariate analysis	60	8
SNHG1	Zhang YJ 2017 [20]	72	China	upregulated	GAPDH	mean	NA	NA	NA	Univariate analysis	60	6
SNHG2	Wang GJ 2018 [21]	112	China	downregulated	GAPDH	median	OS	Survival curve	0.49 (0.3–0.8)	Univariate analysis	48	6
SNHG5	Wei SS 2021 [22]	77	China	downregulated	GAPDH	mean	OS	Survival curve	0.52 (0.27–1.00)	Univariate analysis	60	8
SNHG6	Zhang YL 2019 [23]	75	China	upregulated	GAPDH	median	OS	Survival curve	2.09 (1.08–4.05)	Univariate analysis	60	8
SNHG6	Fan RH 2018 [24]	70	China	upregulated	GAPDH	mean	not reported	NA	NA	NA	NA	6
SNHG8	Wu YH 2022 [25]	20	China	upregulated	GAPDH	mean	OS	not reported	NA	NA	NA	6
SNHG12	Liang M 2020 [26]	85	China	downregulated	GAPDH	mean	OS	Survival curve	0.57 (0.27–1.21)	Univariate analysis	50	8
SNHG12	Wu DG 2020 [27]	70	China	upregulated	GAPDH	mean	not reported	NA	NA	NA	NA	6
SNHG16	Han GH 2018 [28]	128	China	upregulated	GAPDH	median	OS	paper	3.163 (1.213–4.842)	Multivariate analysis	60	9
SNHG16	Ren LH 2022 [29]	55	China	upregulated	GAPDH	mean	not reported	NA	NA	NA	NA	6
SNHG17	Shen SP 2022 [30]	128	China	upregulated	GAPDH	median	OS	paper	1.925 (1.011–3.668)	Multivariate analysis	72	9
SNHG17	Chen WH 2021 [31]	126	China	upregulated	GAPDH	median	not reported	NA	NA	NA	NA	6
SNHG20	Zhang CR 2019 [32]	80	China	upregulated	GAPDH	median	OS	Survival curve	2.24 (1.24–4.05)	Univariate analysis	60	8

GAPDH Glyceraldehyde 3-phosphate dehydrogenase, *OS* overall survival, *SNHG* small nucleolar RNA host gene, *NA* not applicable, *HR* Hazard ratio, *CI* confidence interval, *NOS* Newcastle-Ottawa Scale score

Table 2 The quality evaluation of the included literature is based on the NOS score

Author	Country	Selection				Comparability	Outcome			Total
		Adequate of case definition	Representativeness of the cases	Selection of Controls	Definition of Controls	Comparability of cases and controls	Ascertainment of exposure	Same method of ascertainment	Non-Response rate	
Luo DB 2020 [13]	China	*	*	*	*	*	-	*	*	9
Li HM 2020 [18]	China	*	*	*	*	*	-	*	*	7
Chen Y 2021 [19]	China	*	*	*	*	*	*	*	*	8
Zhang YJ 2017 [20]	China	*	*	*	*	**	-	*	*	8
Wang GJ 2018 [21]	China	*	*	*	*	*	-	*	-	6
Wei SS 2021 [22]	China	*	*	*	*	*	*	*	*	8
Zhang YL 2019 [23]	China	*	*	*	*	*	-	*	-	6
Fan RH 2018 [24]	China	*	*	*	*	*	-	*	-	6
Wu YH 2022 [25]	China	*	*	*	*	*	-	*	-	6
Liang M 2020 [26]	China	*	*	*	*	*	*	*	*	8
Wu DG 2020 [27]	China	*	*	*	*	*	-	*	-	6
Han GH 2018 [28]	China	*	*	*	*	**	*	*	*	9
Ren LH 2022 [29]	China	*	*	*	*	*	-	*	-	6
Shen SP 2022 [30]	China	*	*	*	*	**	*	*	*	9
Chen WH 2021 [31]	China	*	*	*	*	*	-	*	-	6
Zhang CR 2019 [32]	China	*	*	*	*	*	*	*	*	8

Reason for point deduction

1. Lack of clinicopathological data reduces comparability [18, 21, 24, 25]; 2. Univariate analysis may cause statistical bias [19, 22, 23, 26, 32]; 3. Lack of survival data reduces case and control comparability [27, 29, 31]

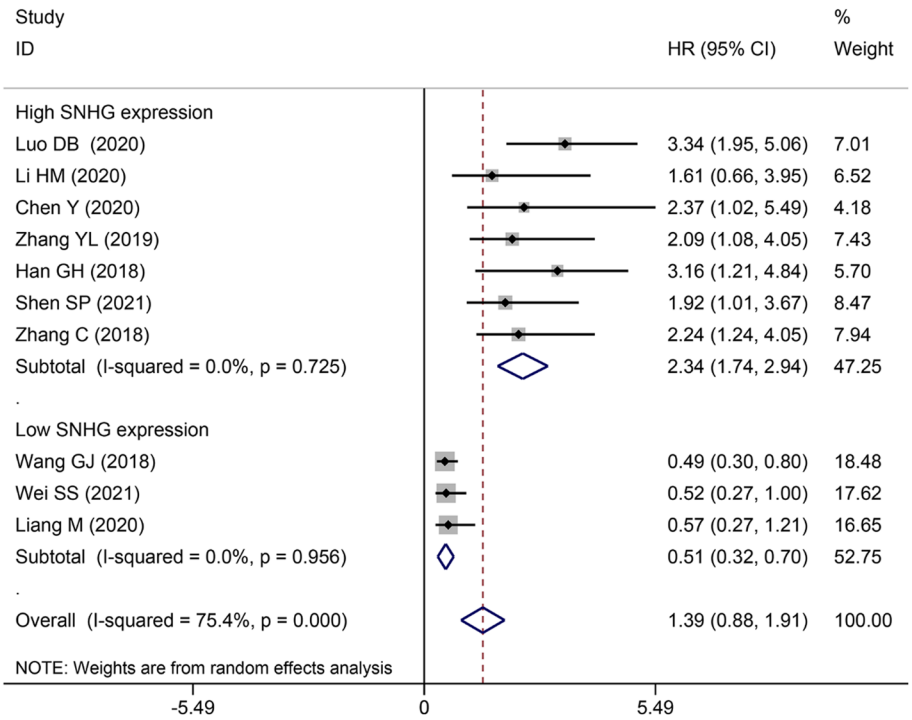

Fig. 2 Forest plot showed the relationship between SNHG expression and overall survival in EC. Note: EC, esophageal cancer

expression (OR: 1.560, 95%CI: 1.216–2.002) and less than 9 of NOS score (OR: 1.351, 95%CI: 1.059–1.723) (Table 4).

Association between SNHG expression and other clinicopathological parameters

Pooling OR with 95%CI suggests that increasing SNHG expression predicted poor histological grade (OR: 1.291, 95%CI: 1.018–1.637) (Fig. 6), deeper invasion of EC cells (OR: 1.565, 95%CI: 1.097–2.232) (Fig. 7). Meanwhile, the association between the expression level of SNHG and distant metastasis (DM) (OR: 1.267, 95%CI: 0.791–2.028) (Fig. 8), age (OR: 1.020, 95%CI: 0.832–1.251) and gender (OR: 0.919, 95%CI: 0.725–1.165) is not significance (Table 4).

Sensitivity analysis and publication bias

Stata 14.0 software was used to test the sensitivity analysis and publication bias of this study. The sensitivity analysis results show that the overall results of OS are robust and reliable (OR: 0.393, 95% CI: −0.115-0.902) (Fig. 9). The results of publication bias showed that there was no obvious publication bias in each result, suggesting that there was no obvious selective publication in each original study (Tables 4 and Fig. 10).

Bioinformatics analysis

Upon exploring the GEPIA database, we discovered that the majority of SNHGs are up-regulated in EC. We did not observe a significant decrease in the expression of SNHG in EC compared to adjacent tissues. This finding further supports the conclusion of our meta-analysis, which suggests that the expression of SNHG is commonly up-regulated in most EC patients (Table 5).

Discussion

Growing evidences have shown that the abnormal expression of SNHG is significantly related to the progression and survival prognosis of esophageal squamous cell carcinoma [35]. However, some SNHG was revealed up-regulating in EC and is significantly associated with poor prognosis of EC [27], other SNHGs are down-regulated in EC, and the high expression of these SNHGs indicated well prognosis of EC [23], leading to the confusion in clinical decision-making and treatment of EC due to inconsistent conclusions between different SNHG. The purpose of this study was to comprehensively explore the correlation between the expression level of all SNHG and the prognosis of EC. This study employed a meta-analysis approach to synthesize data from multiple studies, thereby increasing the statistical power,

Table 3 Subgroup analysis of overall survival based on different subgroups

Subgroup	No. of studies	No. of patients	Pooled HR (95% CI)		Heterogeneity	
			Fixed	Random	I^2(%)	*P*-value
Overall survival	10	816	0.678 (0.498–0.858)	1.392 (0.876–1.908)	75.4	< 0.0001
SNHG expression						
Upregulated	7	542	2.340 (1.744–2.936)	2.340 (1.744–2.936)	0	0.725
Downgerulated	3	274	0.511 (0.322–0.700)	0.511 (0.322–0.700)	0	0.956
cut-off value						
Median	5	523	0.672 (0.435–0.908)	1.836 (0.683–2.988)	80.9	< 0.00001
Mean	5	293	0.686 (0.409–0.963)	1.244 (0.483–2.004)	74.4	0.004
HR extraction						
Directed	3	298	2.674 (1.791–3.557)	2.688 (1.757–3.620)	9.4	0.332
Inderected	7	518	0.579 (0.394–0.764)	0.756 (0.386–1.127)	56.3	0.043
Analysis method						
Multivariate analysis	3	298	2.674 (1.791–3.557)	2.688 (1.757–3.620)	9.4	0.332
Univariate analysis	7	518	0.579 (0.394–0.764)	0.756 (0.386–1.127)	56.3	0.043
No.of patients						
Less than 60	3	131	2.492 (1.484–3.501)	2.485 (1.406–3.565)	11.6	0.323
Not less than 60	7	685	0.618 (0.435–0.801)	1.046 (0.571–1.521)	72.1	0.001
Follow-up (month)						
Not less than 60	8	619	3.342 (1.951–5.064)	2.045 (1.111–2.980)	76.4	< 0.0001
Less than 60	2	197	0.508 (0.287–0.728)	0.508 (0.287–0.728)	0	0.768
NOS score						
NOS score is 9	4	378	2.551 (1.804–3.299)	2.551 (1.804–3.299)	0	0.481
NOS score less than 9	6	438	0.562 (0.377–0.748)	0.657 (0.343–0.971)	41.3	0.13

OS overall survival, *SNHG* small nucleolar RNA host gene, *No.* number, *HR* Hazard ratio, *CI* confidence interval, *NOS* Newcastle-Ottawa Scale score, *Fixed* Fixed effect model, *Random* Random effect model

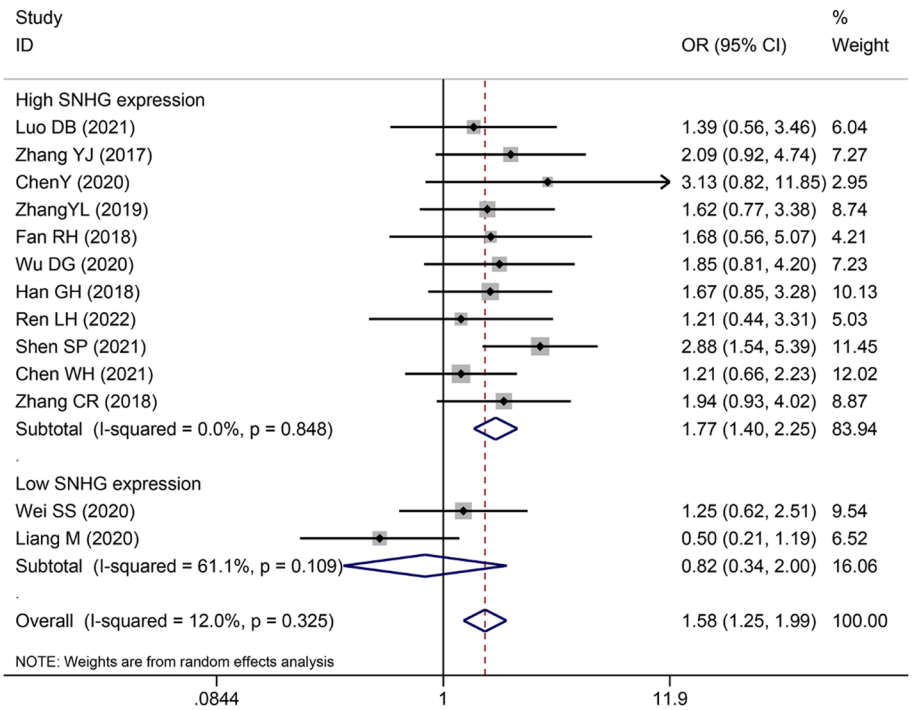

Fig. 3 Forest plot about the relationship between SNHG expression and TNM stage in EC. Note: EC, esophageal cancer

Table 4 Subgroup analysis of different clinicopathological parameters based on different subgroups

Characteristics	No. of studies	No. of patients	Odds ratio (95% CI)		*P*	Heterogeneity	
			Fixed	Random		I^2(%)	*P*-value
Age	22	1830	1.020 (0.832–1.251)	1.020 (0.831–1.252)	0.85	0	0.997
Gender	13	987	0.919 (0.725–1.165)	0.922 (0.725–1.173)	0.486	0	0.756
TNM (III-IV vs. I-II)	13	1044	1.578 (1.273–1.956)	1.578 (1.248–1.994)	< 0.0001	12	0.325
SNHG expression							
High expression	11	882	1.779 (1.404–2.253)	1.772 (1.397–2.247)	< 0.0001	0	0.848
Low expression	2	162	0.858 (0.503–1.465)	0.820 (0.336–2.001)	0.663	61.1	0.109
NOS score							
9	3	298	2.041 (1.358–3.067)	2.020 (1.315–3.103)	0.001	7.7	0.338
Less than 9	10	746	1.427 (1.108–1.838)	1.436 (1.101–1.872)	0.008	5.2	0.393
LNM (present vs. absent)	11	878	1.533 (1.205–1.950)	1.538 (1.205–1.963)	0.001	0	0.574
SNHG expression							
High expression	9	716	1.671 (1.282–2.178)	1.663 (1.274–2.171)	< 0.0001	0	0.961
Low expression	2	162	1.002 (0.557–1.802)	0.916 (0.263–3.184)	0.89	75	0.045
NOS score							
9	3	298	1.730 (1.157–2.585)	1.730 (1.157–2.585)	0.008	0	0.997
Less than 9	8	580	1.432 (1.061–1.934)	1.444 (1.032–2.023)	0.032	12.9	0.329
DM (present vs. absent)	7	504	1.267 (0.791–2.028)	1.352 (0.712–2.570)	0.325	31.7	0.186
SNHG expression							
High expression	6	427	1.617 (0.965–2.711)	1.560 (0.915–2.658)	0.068	0	0.554
Low expression	1	77	0.240 (0.049–1.169)	0.240 (0.049–1.169)	0.077	-	-
NOS score							
9	2	170	1.046 (0.510–2.147)	1.044 (0.506–2.154)	0.907	0	0.455
Less than 9	5	334	1.462 (0.782–2.733)	1.530 (0.568–4.122)	0.401	47.6	0.106
Tumor size (big vs. small)	12	916	1.419 (1.134–1.777)	1.424 (1.084–1.870)	0.002	24.9	0.199
SNHG expression							
High expression	10	754	1.560 (1.216–2.002)	1.573 (1.165–2.124)	< 0.0001	23.4	0.228
Low expression	2	162	0.928 (0.547–1.574)	0.930 (0.547–1.579)	0.782	0	0.57
NOS score							
9	2	170	1.880 (1.044–3.387)	3.515 (0.335–36.932)	0.295	78.4	0.031
Less than 9	10	746	1.351 (1.059–1.723)	1.353 (1.043–1.756)	0.015	8.6	0.363
Histological grade	10	806	1.291 (1.018–1.637)	1.287 (1.014–1.634)	0.035	0	0.933
SNHG expression							
High expression	9	721	1.347 (1.046–1.736)	1.343 (1.041–1.732)	0.021	0	0.949
Low expression	1	85	0.943 (0.474–1.875)	0.943 (0.474–1.875)	0.867	-	-
NOS score							
9	3	298	1.432 (0.928–2.210)	1.422 (0.918–2.202)	0.105	0	0.458
Less than 9	7	508	1.234 (0.929–1.639)	1.234 (0.928–1.640)	0.146	0	0.937
Invasion depth (III-IV vs. I-II)	4	370	1.565 (1.097–2.232)	1.562 (1.094–2.231)	0.013	0	0.677

SNHG small nucleolar RNA host gene, *No.* number, *DM* distant metastasis, *Fixed* Fixed effect model, *Random* Random effect model, *HR* Hazard ratio, *CI* confidence interval, *NOS* Newcastle-Ottawa Scale score, *TNM* Tumor, Node, Metastasis, *LNM* lymph node metastasis

precision, and reliability of the analysis while minimizing bias. By evaluating the potential association between SNHG expression and the prognosis of EC, we were able to provide a more accurate estimate of the effect size and identify potential prognostic markers. Furthermore, this study attempted to identify subgroups of patients who may benefit more from SNHG drug intervention, which could inform personalized medical approaches. Moreover, the implementation of a meta-analysis can provide a more comprehensive understanding of the association between SNHG expression and the prognosis of EC by pooling data from various studies and reducing the risk of bias. This is especially important in the case of SNHG family members, as their functions and mechanisms of

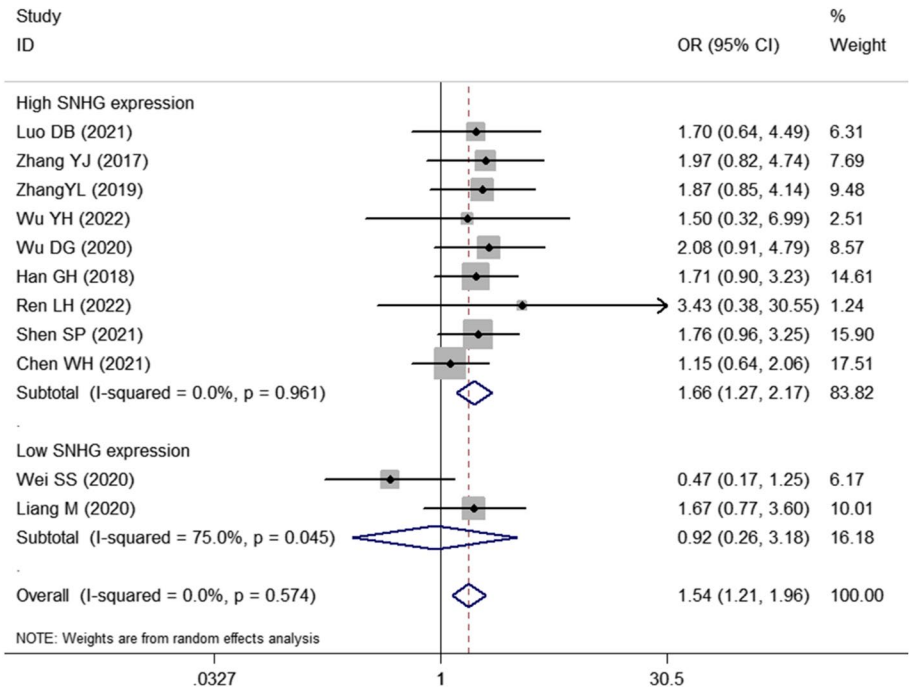

Fig. 4 Forest plot about the relationship between SNHG expression and LNM in EC. Note: EC, esophageal cancer

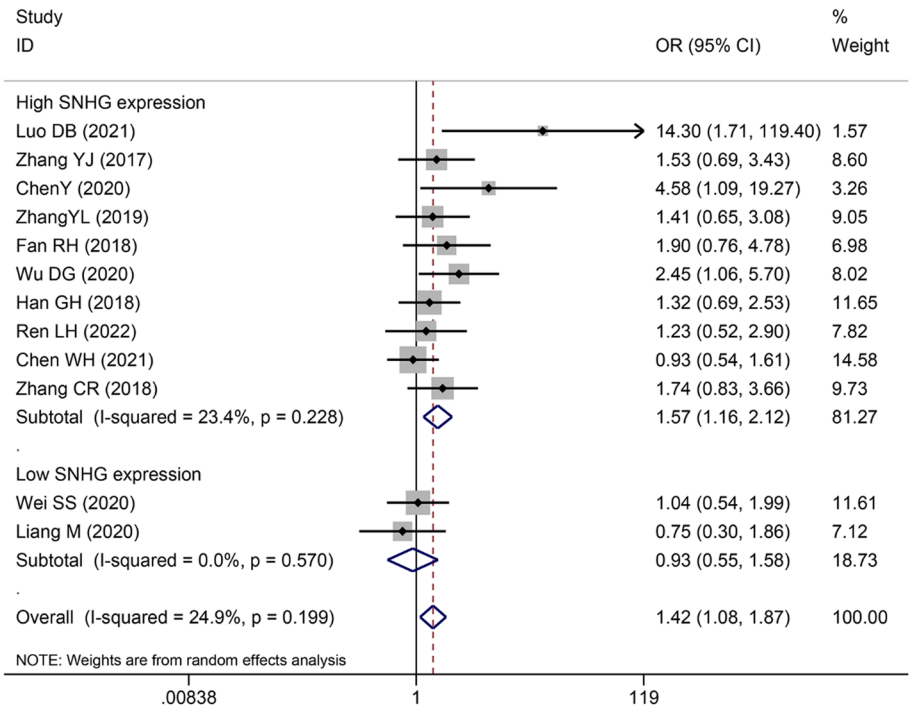

Fig. 5 Forest plot about the relationship between SNHG expression and Tumor size in EC. Note: EC, esophageal cancer

action may differ, and their individual contributions to cancer development and progression may be difficult to ascertain from single studies.

More and more researchers are trying to uncover the molecular biological mechanism of SNHG affecting the progression of EC (Tables 6 and Fig. 11). First, SNHG

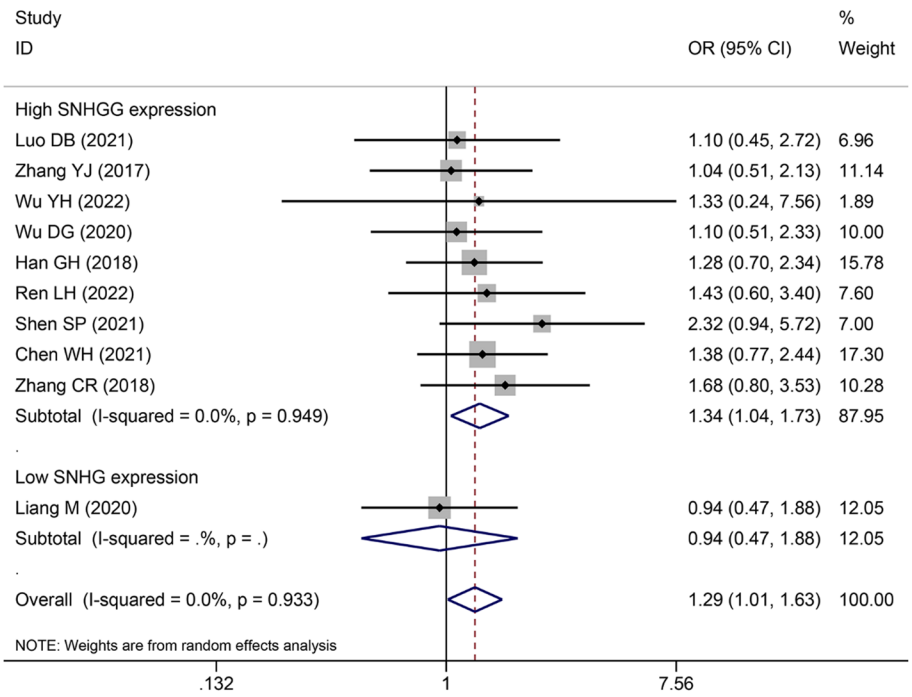

Fig. 6 Forest plot about the relationship between SNHG expression and histological grade in EC. Note: EC, esophageal cancer

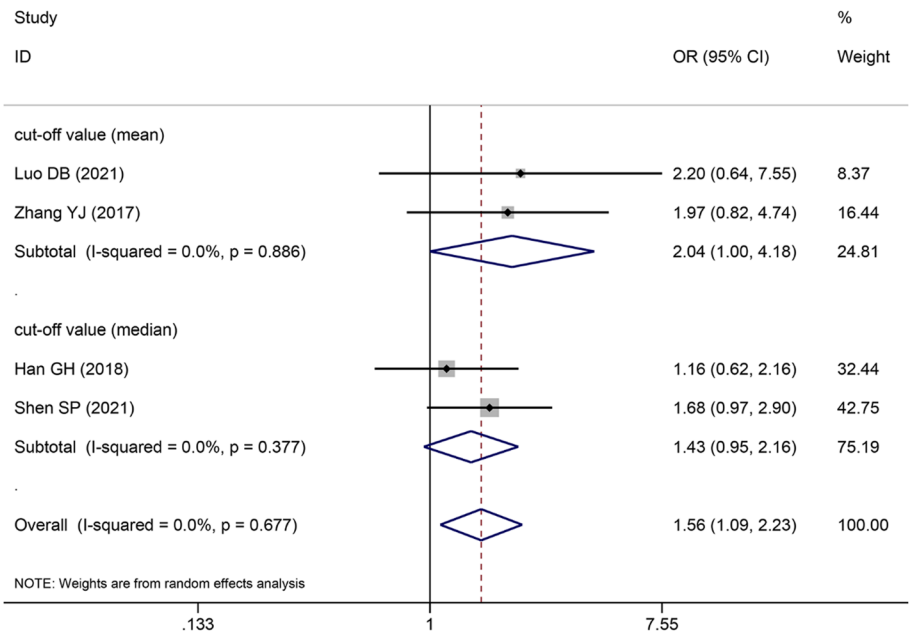

Fig. 7 Forest plot about the relationship between SNHG expression and depth of invasion in EC. Note: EC, esophageal cancer

could act as a tumor promoter gene, promoting the proliferation, migration and invasion of EC cells by directly acting on related signal pathways or proteins in cells. Ren et al. reported that SNHG16 may boost the proliferation and metastasis of EC cells by enriching Ras homologue family member U (RhoU) via down-regulating Eukaryotic translation initiation factor 4A3 (EIF4A3) [30]. Shen et al. uncovered that SNHG17 may facilitate proliferation, lessen apoptosis, elevates migration, invasion and EMT process of Eca-109 cells by decreasing E-cadherin

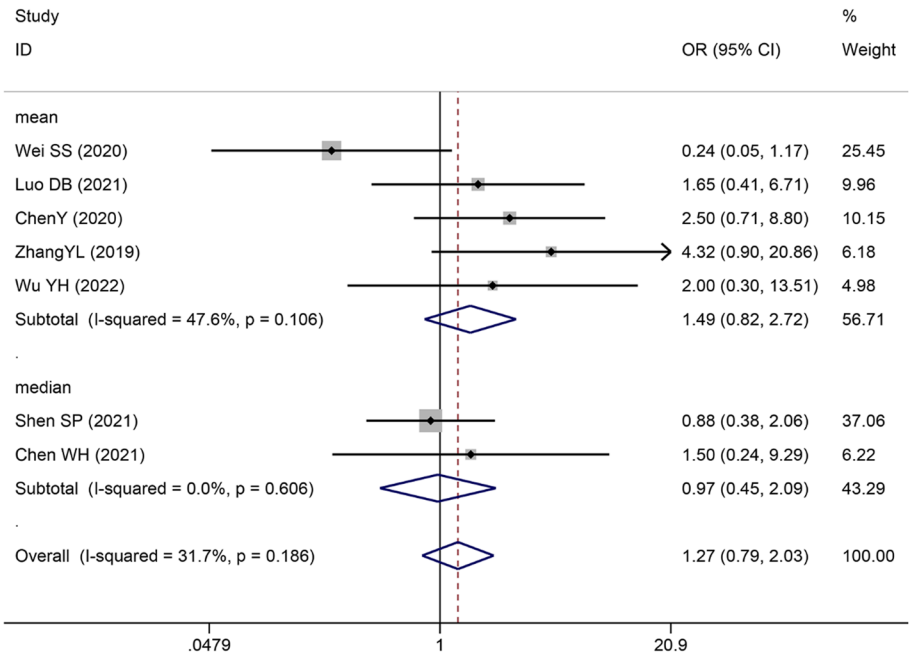

Fig. 8 Forest plot about the relationship between SNHG expression and distant metastasis in EC. Note: EC, esophageal cancer

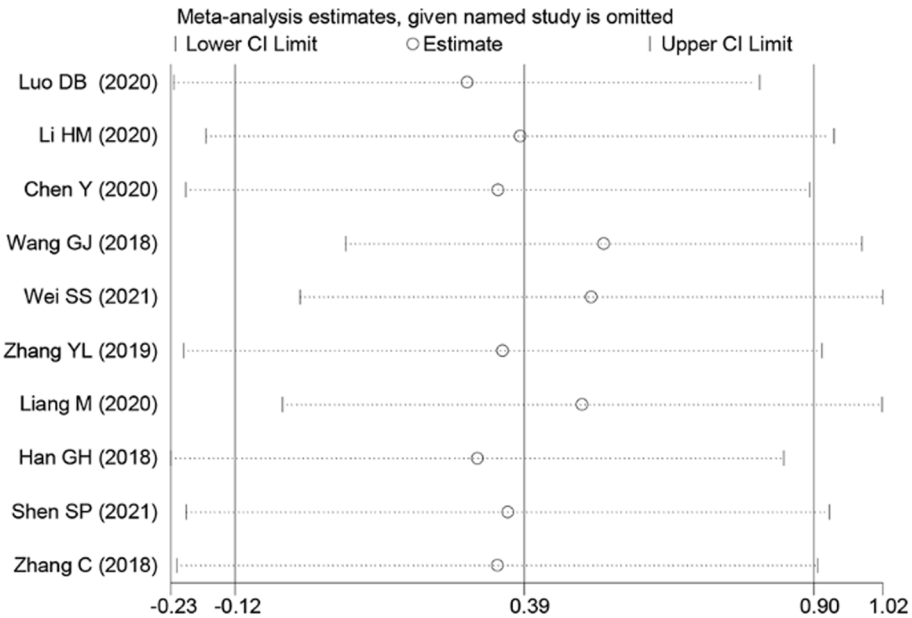

Fig. 9 Sensitivity analysis for SNHG expression with overall survival in EC. Note: EC, esophageal cancer; HR: hazard ratio, CI: confidence interval

expression, increasing N-cadherin and c-Myc expression, and activating PI3K/AKT pathway [31]. Han et al. discovered that SNHG16 promotes the proliferation and invasion of EC-1 cells through the activity of Wnt/β-catenin signaling pathway [29]. Xu et al. revealed that SNHG7 may induce proliferation and inhibit apoptosis of EC9706 cells via suppressing p15 and p16 expression [36]. Zhang et al. implied that SNHG1 boost the proliferation, invasion and EMT process of TE-1 cells via increasing E-cadherin expression, decreasing N-cadherin and Vimentin expression and promoting Notch signaling pathway by increasing the Notch1 and Hes-1 expression [21].

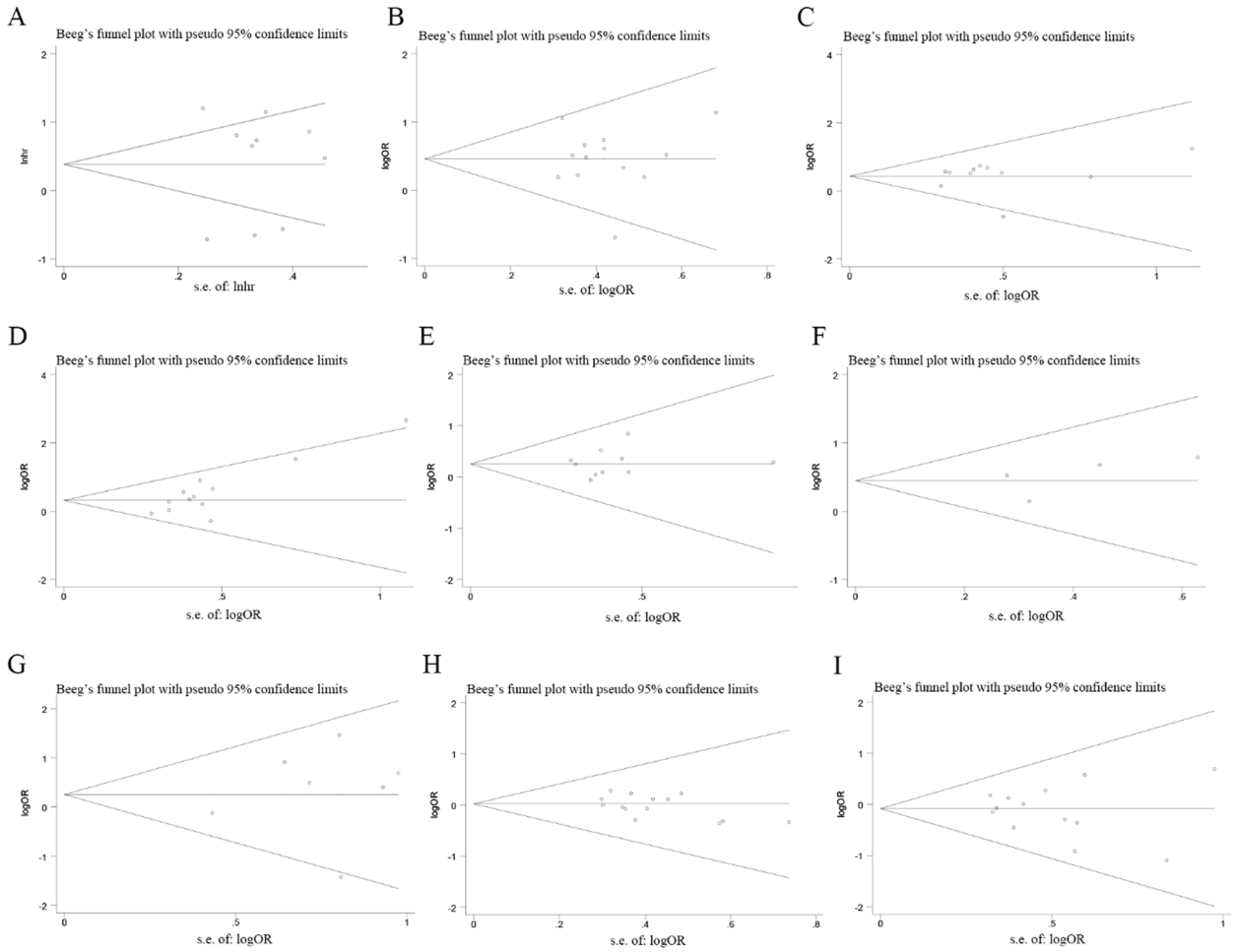

Fig. 10 Funnel plot about the relationship between SNHG expression and survival outcome in EC. **A** OS; **B** TNM stage; **C** LNM; **D** Tumor size; **E** Histological grade; **G** Depth of invasion; **F** DM; **G** Age; **H** Gender. Note: EC, esophageal cancer

In addition to its role in regulating cellular processes, SNHG can also impact the biological behavior of tumor cells through its function as a competitive endogenous RNA (ceRNA) by sponging microRNAs within cells, modulating downstream signaling pathways, leading to altered gene expression and potentially contributing to the development and progression of cancer. For example, Li et al. reported that SNHG1 may drive the proliferation and invasion, inhibit the apoptosis of EC9706 cells by serving as ceRNA, increasing homeobox c8 (HOXC8) expression via decreasing miR-204 [19]. Wu et al. revealed that SNHG12 induced proliferation, invasion and EMT process via post-transcriptional regulation of β-Catenin (CTNNB1) by decreasing miR-6835-3p [28]. Zhang et al. suggested that Differentiation Antagonizing Non-Protein Coding RNA (DANCR, also named as SNHG13) facilitate the proliferation and metastasis of EC cells by up-regulating zinc-finger-enhancer binding protein 1 (ZEB1) expression via sponging and down-regulating miR-33a-5p in EC109 cells [34]. Chen et al. demonstrated that SNHG1 contributes toward cell migration and invasion of TE-1 cells by increasing E-cadherin expression and decreasing N-cadherin and Vimentin expression via sponging and down-regulating miR-195 [20]. Several SNHG could also affect the sensitivity of EC cells to radiotherapy, for example, Lin et al. revealed that Growth Arrest-Specific 5 (GAS5, also named as SNHG2) could enhance the radio-sensitivity of TE-1 cells through the up-regulation of reversion-inducing cysteine-rich protein with Kazal motifs (RECK) expression by sponging miR-21 [36].

In addition, part of SNHG has also been revealed to act as a tumor suppressor gene and inhibit the progression of EC. For example, Liang et al. uncovered that SNHG12 suppressed proliferation, migration, invasion and promoted apoptosis of EC cells through competitively bind to miRNA-195-5p and prevent it from binding to the mRNA of B-cell CLL/lymphoma 9 (BCL9) [27]. This

Table 5 Comparison of the expression of different SNHGs between esophageal carcinoma and adjacent tissues

lncSNHG	Numbers of cancer tissues	Numbers of normal tissues	Expression level (T vs. *N*)	*P* value
SNHG1	182	13	Upregulated	*
SNHG2	182	13	Upregulated	*
SNHG3	182	13	Upregulated	*
SNHG4	182	13	Upregulated	*
SNHG5	182	13	Upregulated	Not significant
SNHG6	182	13	Upregulated	*
SNHG7	182	13	Upregulated	Not significant
SNHG8	182	13	Upregulated	Not significant
SNHG9	182	13	Upregulated	Not significant
SNHG10	182	13	Upregulated	Not significant
SNHG11	182	13	Upregulated	*
SNHG12	182	13	Upregulated	*
SNHG13	182	13	Upregulated	*
SNHG14	182	13	downregulated	*
SNHG15	182	13	Upregulated	*
SNHG16	182	13	Upregulated	*
SNHG17	182	13	Upregulated	*
SNHG18	182	13	Upregulated	Not significant
SNHG19	182	13	Upregulated	*
SNHG20	182	13	Upregulated	Not significant
SNHG21	182	13	Upregulated	Not significant
SNHG22	182	13	Upregulated	Not significant
SNHG23	182	13	Downregulated	Not significant
SNHG24	182	13	Downregulated	Not significant
SNHG25	182	13	Upregulated	Not significan

T tumor, *N* normal

results in increased expression of BCL9 and its downstream targets. Wang et al. discovered that GAS5 was down-regulation in EC tissue, high GAS5 expression suppressed the proliferation and migration of EC9706 cells by inactivating PI3K/AKT/mTOR pathway [22]. Wei et al. reported that SNHG5 could serve as a tumor suppressor gene, high SNHG5 expression could inhibit the migration and invasion by reversing the EMT process of EC cells via pulling down metastasis-associated protein 2 (MTA2) [23]. Additionally, some SNHG molecules have been shown to up-regulate the expression of the PD-L1 receptor on the surface of EC cells [33]. This interaction between the receptor and immune cells can lead to the inhibition of the immune response, allowing cancer cells to evade detection and destruction by the immune system. These findings suggest that SNHG may play a role in the modulation of immune checkpoints in EC and could have implications for the development of targeted immunotherapies in the future. For example, Zhang et al. Revealed that SNHG20 may promote proliferation and migration of EC cells through the activation of ATM-JAK-PD-L1 pathway [33].

The results of this study suggest a positive correlation between high expression of SNHG and poor prognosis of EC, but the correlation was not statistically significant, more high-quality large sample studies may be needed to support the conclusions of this study. Additionally, while some SNHG molecules such as SNHG2, SNHG5, and SNHG12 are down-regulated in EC and predict a good prognosis, others are up-regulated and associated with poor prognosis. As such, the correlation between the expression levels of all SNHG and EC prognosis may not be significant. To account for this, our study conducted subgroup analysis based on SNHG expression levels and other factors such as the Newcastle-Ottawa Scale score, cut-off value, and number of patients. By doing so, we aimed to identify potential patterns or consistencies in the data that could inform future research and clinical decision-making. The results indicate that in populations with high expression of SNHG2, SNHG5, and SNHG12, improved overall survival is predicted for patients with EC. In contrast, in other SNHG subgroups, high expression of SNHG is associated with poor overall survival. The findings of this study can offer valuable guidance for

Table 6 Molecular biology mechanisms of EC progression influenced by lncRNA SNHG

LncRNA	Expression	Role	Micro-RNAs	Targets/ pathway	Cell line	Involved in functions	References
SNHG1	upregulated	Oncogene	miR-21	-	TE-1, Eca-109, KYSE170, and KYSE150	Cell proliferation	Luo DB 2020 [13]
SNHG1	upregulated	Oncogene	miR-204	HOXC8	EC9706, KYSE450, KYSE150 and Eca109	Migration, invasion, and apoptosis	Li HM 2020 [18]
SNHG1	upregulated	Oncogene	miR-195	EMT pathway	EC109, Het-1, TE-1, Cdc42	Cell migration and invasion	Chen Y 2021 [19]
SNHG1	upregulated	Oncogene	-	Notch and EMT pathway	Eca109 and TE-1	Cell proliferation and invasion	Zhang YJ 2017 [20]
GAS5 (SNHG2)	downregulated	Anticancer gene	miR-21	RECK	TE-1, TE-1-R (radiation resistant cell line)	Cell apoptosis, proliferation, invasion, and cell radio-sensitivity	Lin J 2020 [36]
GAS5 (SNHG2)	downregulated	Anticancer gene	-	PI3K/AKT/mTOR	EC9706 and KYSE510	Cell proliferation and migration	Wang GJ 2018 [21]
SNHG5	downregulated	Anticancer gene	-	MTA2, and EMT pathway	Eca-109, TE-13, TE-1, KYSE-170, KYSE-70, and KYSE-510, KYSE-180 and NE2	-	Wei SS 2021 [22]
SNHG6	upregulated	Oncogene	-	-	EC9706, EC109, EC1 and HET-1 A	Cell migration and invasion	Zhang YL 2019 [23]
SNHG6	upregulated	Oncogene	-	-	HEEC, ECA-109 and TE-1	Cell proliferation and apoptosis	Fan RH 2018 [24]
SNHG7	upregulated	Oncogene	-	p15, p16	Eca109, EC9706, TE-10, TE-11 and HEEC	Cell proliferation and apoptosis	Xu LJ 2018 [35]
SNHG8	upregulated	Oncogene	miR-1270	BACH1	Het-1 A, KYSE30, EC9706 and TE-1	Cell apoptosis and proliferation	Wu YH 2022 [25]
SNHG12	downregulated	Anti-oncogene	miR-195-5p	BCL9	Het-1 A, KYSE140, KYSE510, Eca9706, and Ec109	Cell proliferation, migration, invasion and apoptosis	Liang M 2020 [26]
SNHG12	upregulated	Oncogene	miR-6835-3p	BMI1, CTNNB1	EC9706, EC109, KYSE410, KYSE150 and KYSE450	Cell proliferation, and migration	Wu DG 2020 [27]
DANCR (SNHG13)	upregulated	Oncogene	miR-33a-5p	ZEB1	Het-1 A, EC9706, EC109, EC1 and KYSE150	Cell proliferation and metastasis	Zhang CY 2019 [33]
SNHG16	upregulated	Oncogene	-	Wnt/β-catenin signaling pathway	HECC, TE-13, TE-1, EC-1 and Eca-109	Cell proliferation and invasion	Han GH 2018 [28]
SNHG16	upregulated	Oncogene	-	EIF4A3, RhoU	Het-1 A, Eca109, KYSE30, KYSE140 and KYSE410	Cell proliferation and metastasis	Ren LH 2022 [29]
SNHG17	upregulated	Oncogene	-	c-Myc, PI3K/AKT, TGF-β1, and EMT pathway	TE1, Eca109, Kyse150, Kyse170 and Yes2	Cell proliferation, apoptosis, migration, and invasion	Shen SP 2022 [30]
SNHG17	upregulated	Oncogene	miR-338-3p	SOX4	Het-1 A, Eca109, TE-1 and EC9706	Cell proliferation and invasion	Chen WH 2021 [31]
SNHG20	upregulated	Oncogene	-	ATM/JAK/PD-L1 pathway	KYSE450, KYSE150, EC9706, and EC109	Cell proliferation, migration, invasion and apoptosis	Zhang CR 2019 [32]

EC esophageal cancer, *ZEB1* Zinc finger E-box-binding homeobox 1, *SNHG* small nucleolar RNA host gene, *miR* microRNA, *DANCR* Differentiation Antagonizing Non-Protein Coding RNA, *GAS5* Growth Arrest-Specific 5, *SOX4* SRY (Sex Determining Region Y)-Box Transcription Factor 4, *PD-L1* Programmed Death-Ligand 1, *JAK* Janus Kinase, *PI3K* Phosphatidylinositol 3-Kinase, *AKT* Protein Kinase B, *EIF4A3* Eukaryotic Initiation Factor 4 A-III, *BMI1* B-Cell-Specific Moloney Murine Leukemia Virus Integration Site 1, *CTNNB1* Catenin Beta 1, *EMT* Epithelial-Mesenchymal Transition, *BCL9* B-Cell Lymphoma 9, *HOXC8* Homeobox C8, *RECK* Reversion-inducing Cysteine-rich Protein with Kazal motifs, *EC* Esophageal Squamous Cell Carcinoma

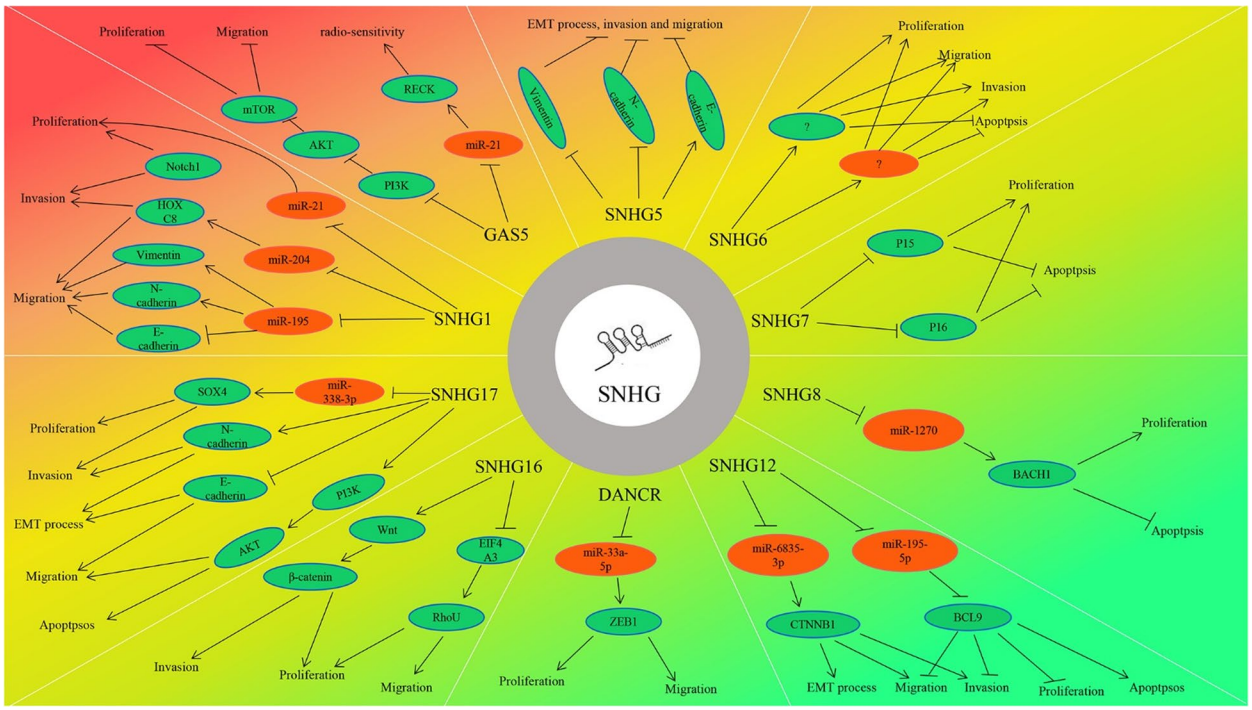

Fig. 11 SNHG involved in a serious of cellular biological role in EC. Note: The "arrow" symbol denotes a promotional relationship. For instance, SNHG16 can enhance the migration of esophageal cancer cells by upregulating the Wnt/β-catenin signaling pathway. The "inhibition line" symbol signifies a suppressive interaction. For example, SNHG8 can increase the levels of BACH1 by downregulating the expression of miR-1270, a process akin to sponge-like absorption, which in turn leads to accelerated proliferation and decelerated apoptosis in esophageal cancer cells. The "?" symbol indicates that it is currently unclear which molecular targets SNHG interacts with to regulate cellular processes such as proliferation, migration, and invasion. For example, SNHG6 has been shown to promote the proliferation and migration of esophageal cancer cells, as well as to inhibit apoptosis. However, the specific downstream molecules that SNHG6 modulates to exert these effects remain to be elucidated

clinical decision-making and the development of targeted treatment strategies for EC patients.

Furthermore, our study investigated the relationship between SNHG expression levels and various clinicopathological parameters. The results revealed that high expression of SNHG is linked to advanced TNM stage, earlier lymph node metastasis, larger tumor diameter, deeper tumor cell infiltration, and worse histological grade. Simultaneously, the overall findings also demonstrated that there was no significant correlation between SNHG expression levels and the occurrence of distant metastasis, nor with the age and sex of the patients. This comprehensive analysis can further contribute to a better understanding of the complex relationship between SNHG expression and EC prognosis, ultimately aiding in the development of more effective, personalized treatment approaches.

This study has some limitations that should be considered. Firstly, the sample population is exclusively Chinese, thus limiting the generalizability of the conclusions to Asian or Chinese patients only, and the results and conclusions of this study are limited to the East Asian population, and we look forward to the implementation and publication of more studies on other ethnic groups. Secondly, certain survival data HR values were derived from original literature while others were obtained through Engauge software, which may have introduced some statistical bias to the results of this meta-analysis. Thirdly, there were relatively fewer high-quality documents and a smaller sample size included in this study. Therefore, to ensure the accuracy and reliability of the findings, it is recommended that future research employs a larger sample size and higher-quality original documents to further support and validate the results and conclusions presented in this article. Finally, within the primary literature encompassed by our study, a significant gap is observed in the research domain, with little to no exploration of the correlation that may exist between the pathological subtypes of esophageal cancer and the expression levels of members within the SNHG family. Furthermore, the literature similarly falls short in investigating the potential relationship that could link these pathological subtypes to patient prognosis. These omissions inherently circumscribe the analytical reach of our study. In conclusion, this study still draws a relatively clear conclusion, that is, the low expression of SNHG2,

SNHG5, and SNHG12 in EC, and the high expression of SNHG2, SNHG5, and SNHG12 indicate a good prognosis of EC. Other SNHGs are highly expressed in EC and predict poor prognosis of EC. SNHG could be used as a potential therapeutic target and prognostic marker for EC. Targeted therapy targeting SNHG for EC requires individualized treatment.

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Authors' contributions
Qiang Guo, Dong-Xiao Ding and Yue-Feng Liu design the project. Dan Li and Wei-Min Luo searched databases and performed literature screen. Ke Shi, Hua-Song Liu and Li-De Huang extracted and analyzed the data, analysis, evaluated the quality of included literature. Dan Li, Li-De Huang and Ke Shi contributed to writing the manuscript. Final draft was approved by all the authors.

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Data availability
All data generated or analyzed during this study are included in this published article or are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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