



Review Article

The Prognostic Value of LncRNA SLNCR1 in Cancers: A Meta-Analysis

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Objective. This meta-analysis was performed to identify the prognostic value of SLNCR1 in multiple cancer types. **Methods.** Electronic databases, including PubMed, EMBASE, and Web of Science, Cochrane Library, Medline, BioMed Central, Springer, Science Direct, and China National Knowledge Internet (CNKI), were searched for relevant studies up to August 2021, and the hazard ratios (HR) and 95% confidence intervals (95% CI) were calculated to assess the relationship between SLNCR1 expression and overall survival (OS). **Results.** 12 studies with a total of 1155 patients with 9 different types of cancers were included in this meta-analysis. The pooled HR indicates that high SLNCR1 expression represented poorer prognosis of cancer (HR = 2.11, 95% CI: 1.59–2.80, $I^2 = 0\%$, $P < 0.00001$). Additionally, high SLNCR1 expression was correlated with TNM stage (odds ratio (OR): 1.72, 95% CI: 1.08–2.74, $I^2 = 62\%$, $P = 0.02$), lymph node metastasis (LNM) (OR: 2.42, 95% CI: 1.61–3.64, $I^2 = 55\%$, $P < 0.0001$), and distant metastases (DM) (OR: 2.30, 95% CI: 1.50–3.55, $I^2 = 27\%$, $P = 0.0002$). However, no evidence was found for a relationship between SLNCR1 expression and clinical features such as tumor size (OR: 1.71, 95% CI: 0.93–3.14, $I^2 = 71\%$, $P = 0.09$), age (OR: 0.86, 95% CI: 0.68–1.08, $I^2 = 0\%$, $P = 0.19$), or gender (OR: 1.07, 95% CI: 0.64–1.81, $I^2 = 55\%$, $P = 0.79$). **Conclusion.** Our findings found that high SLNCR1 expression was associated with poor OS, advanced tumor stage, tumor size, LNM, and DM in multiple cancers, indicating that SLNCR1 may serve as a potential prognostic biomarker for cancer patients in China.

1. Introduction

Cancer is expected to rank as the leading cause of death and the most significant barrier to extend human life expectancy worldwide. The incidence and mortality of cancer are expected to grow rapidly with increases in population and age [1]. Although significant achievements have been made in cancer diagnosis and treatment, the five-year survival rate remains dismally low. Numerous scientists remain dedicated to find effective biomarkers for cancer patients [2].

Long noncoding RNA (LncRNA), a novel class of noncoding RNA, commonly refers to RNA transcripts greater than 200 nucleotides in length [3]. Moreover, accumulating studies indicate that LncRNAs may participate

in a wide range of biological functions and act as oncogenes or tumor suppressors in cancer evolution [4, 5].

Steroid receptor RNA activator (SRA)-like noncoding RNA 1 (SLNCR1) is located on human chromosome 17q24.3. Recently, emerging evidence has revealed that SLNCR1 is aberrantly expressed in various cancers, including malignant melanoma [6], non-small-cell lung cancer [7], breast cancer [8], pancreatic cancer [9], and cervical cancer [10], and promotes cancer cell migration, proliferation, and invasion. However, the correlation between SLNCR1 expression and cancer prognosis remains unknown. Therefore, this meta-analysis was performed to bridge this gap in knowledge between the expression of SLNCR1 and prognosis in different kinds of cancers.

2. Materials and Methods

2.1. Literature Search Strategy and Selection. Two of the authors conducted a systematic search of published studies to identify relevant articles on the association of SLNCR1 expression with the prognosis of cancer. English language databases, including PubMed, EMBASE, Web of Science, Cochrane Library, Medline, BioMed Central, Springer, Science Direct, and China National Knowledge Internet (CNKI), were searched for eligible studies published from inception to August 2021. The search keywords were as follows: “LINC00673” or “Lnc00673” or “lncRNA 00673,” “SLNCR1” or “SLNCR,” “ERRLR01,” “cancers,” “prognosis,” “survival” “clinicopathologic feature,” and “OS (overall survival).” In addition, the references of the relevant studies were screened to avoid omitting any potentially eligible studies. The literature screening and study selection process is shown in Figure 1.

2.2. Inclusion and Exclusion Criteria. The criteria for study inclusion were as follows: (1) case-control studies or cohort studies; (2) cancer was definite diagnosis by pathological examination; (3) studies examining prognostic characteristics of SLNCR1 expression in tumors, and patients were grouped in accordance with high or low SLNCR1 expression levels; (4) studies with sufficient data, including survival outcome, Kaplan–Meier curve, metastasis, and clinical features for statistical analysis.

The criteria for exclusion were as follows: (1) nonhuman studies, letters, case reports, and review articles; (2) studies without prognostic outcomes.

2.3. Data Extraction and Quality Assessment. Two of the authors screened all the eligible studies and completed data extraction independently. The data included the first author’s name, publication year, country of origin, cancer type, sample size, age, gender, tumor size, lymph node metastasis (LNM), distant metastasis (DM), TNM stage, cutoff value, and method of detecting SLNCR1. For studies that provided only the Kaplan–Meier curve, Engauge Digitizer version 4.1 was used to extract hazard ratios (HRs) and 95% confidence intervals (95% CIs), and the method described by Tierney et al. was used to obtain survival data [11]. The quality of the included studies was evaluated by the Newcastle–Ottawa Scale [12].

2.4. Statistical Analysis. The strength of the association between SLNCR1 expression and the prognosis of cancer was estimated by calculating HR or odds ratio (OR) and 95% CIs. HR and 95% CIs were extracted from the Kaplan–Meier curves from published studies, and log HR and standard error were used to summarize overall survival (OS). TNM I and II were combined to indicate low tumor stage, and III and IV were combined for representing the advanced tumor stage. The OR was used to estimate the outcome. Tests for heterogeneity assumptions were checked by the Cochran Q statistic and I^2 tests [13]. $I^2 < 50\%$ and $P > 0.05$ indicated no

significant heterogeneity across the studies; therefore, a fixed-effect model was used. $I^2 > 50\%$ and $P < 0.05$ denoted strong heterogeneity for which a random-effect model was used for analysis. Funnel plots were utilized to assess potential publication bias. Sensitivity analyses were performed to identify individual study effects that contributed to pooled results and test the results’ reliability.

3. Results

3.1. Characteristics of Included Studies. This meta-analysis encompassed 12 studies that met the inclusionary and exclusionary criteria, involving 1155 patients with eight different types of cancers: colorectal cancer [14], non-small-cell lung cancer [15, 16], epithelial ovarian cancer [17], gastric cancer [18, 19], thyroid cancer [20], tongue squamous cell carcinoma [21], pancreatic cancer [22], breast cancer [23, 24], and esophageal squamous cell carcinoma [25]. 56 studies associated with the prognosis and metastasis of SLNCR1 and cancers were retrieved from PubMed, EMBASE, Web of Science, Cochrane Library, Medline, BioMed Central, Springer, Science Direct, and China National Knowledge Internet (CNKI). After carefully screening the titles and abstracts, 26 studies were excluded because of duplication. Of the remaining studies, 15 were excluded because they were not case-control or cohort studies or were irrelevant to the present study, and five studies with insufficient data were excluded. Ultimately, 12 articles were selected for the present meta-analysis [14–25]. The quality assessment scores ranged from 6 to 7.

The main features of the ten studies are given in Table 1. All of the studies were conducted in China and published between 2016 and 2021. The sample sizes ranged from 35 to 229 patients. Based on the expression of SLNCR1 detected by quantitative real-time PCR, patients were divided into two groups, referred to as high and low SLNCR1 expression groups. 7 [14, 18–20, 22–24] studies focused on the relationship between SLNCR1 expression and OS, and 10, 5, 10, 12, 11, and 8 on relationships with LNM [14, 15, 17–24], DM [14, 18, 20–22], TNM [15, 17–25], age [14–25], tumor size [14–21, 23–25], and gender [14–16, 18–21, 25], respectively.

3.2. Association between SLNCR1 Expression and OS. A meta-analysis was performed to estimate the relationship between SLNCR1 expression and OS. HR was extracted from the survival curves in 7 [14, 17–19, 21–23] of the studies. As shown in Figure 2(a), a fixed-effect model was used since no significant heterogeneity was observed ($I^2 = 0$, $P = 0.82$). The combined HR was 2.11 (95%nCI: 1.59–2.80, $P < 0.00001$), revealing that OS in cancers was markedly related to SLNCR1 expression, with the high SLNCR1 expression group displaying poorer OS than the low SLNCR1 expression group. No obvious asymmetry was detected by the shape of the funnel plot (Figure 2(b)). Sensitivity analysis demonstrated no significant influence by eliminating any single study on the pooled HR, revealing that the results were stable (Figure 2(c)).

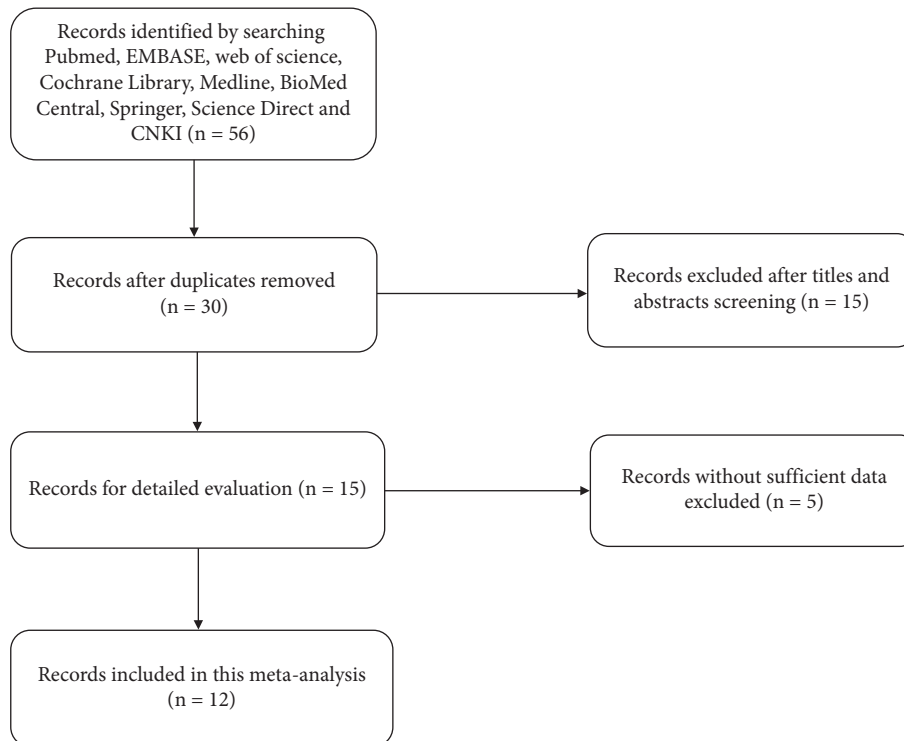


FIGURE 1: Literature screening and study selection process flow diagram.

Two subgroups (digestive system cancers and non-digestive system cancers) were established to assess the HR among different types of cancer (Figure 3). The result suggests that high SLNCR1 expression was associated with poor OS (digestive system cancers: HR: 2.27, 95% CI: 1.62–3.17, $I^2 = 0\%$, $P < 0.00001$; nondigestive system cancers: HR: 1.78, 95% CI: 1.05–3.00, $I^2 = 0\%$, $P = 0.03$) regardless of the subgroup (see combined HR data above).

3.3. Association between SLNCR1 Expression and Clinicopathological Characteristics. 10 [15, 17–25] and 11 [14–21, 23–25] eligible studies reported the state of TNM and tumor size, respectively, based on the expression level of SLNCR1. Compared with the low expression group, the high expression group displayed more advanced TNM stages (OR: 1.72, 95% CI: 1.08–2.74, $I^2 = 62\%$, $P = 0.02$) (Figure 4(a)) with respect to SLNCR1 expression. However, no relationship was found between elevated expression of SLNCR1 and tumor sizes (OR: 1.71, 95% CI: 0.93–3.14, $I^2 = 71\%$, $P = 0.09$) (Figure 4(b)). Other relationships between SLNCR1 expression and clinicopathological characteristics were uninvestigated because of insufficient data.

3.4. Association between SLNCR1 Expression and Metastasis. Data regarding the association between SLNCR1 expression and LNM were collected from the 12 [14–25] eligible studies. The random-effect model was adopted because of significant heterogeneity, and high SLNCR1 expression was correlated with LNM (OR: 2.42, 95% CI: 1.61–3.64, $I^2 = 55\%$, $P < 0.0001$) (Figure 5(a)). Furthermore, a relationship

between SLNCR1 expression and DM was detected in five [14, 18, 20–22] studies for which the fixed-effect model was used based on limited heterogeneity. Subsequently, a significant difference was found between high SLNCR1 expression and DM (OR: 2.30, 95% CI: 1.50–3.55, $I^2 = 27\%$, $P = 0.0005$) (Figure 5(b)).

3.5. Association between SLNCR1 Expression and Clinical Features. The association between SLNCR1 expression and age was examined in 12 [14–25] eligible studies. A fixed-effect model was utilized to calculate the OR because no significant heterogeneity was observed among the enrolled studies. Figure 6(a) shows that elevated expression of SLNCR1 was not correlated with age (OR: 0.86, 95% CI: 0.68–1.08, $I^2 = 0\%$, $P = 0.19$). Moreover, only 8 [14–16, 18–21, 25] studies described in detail the relationship between SLNCR1 expression and gender. As shown in Figure 6(b), no correlation was found between elevated expression of SLNCR1 and gender (OR: 1.07, 95% CI: 0.64–1.81, $I^2 = 55\%$, $P = 0.79$).

3.6. Publication Bias and Sensitivity Analysis. The publication bias of this meta-analysis was estimated by Begg's and Egger's tests (Figure 7). No evidence of publication bias was found in the meta-analysis of OS by Begg's ($P = 0.54$) or Egger's ($P = 0.80$) test. However, sensitivity analysis by elimination of each study to determine its effect on the calculation of overall risk of disease found that two studies significantly affected the analysis of the relationship between SLNCR1 expression and TNM stage. After omitting the

TABLE 1: Characteristics of studies included in the meta-analysis.

Study (year)	Region	Cancer type	Sample size	Age (low/high)	Male (low/high) Female (low/high)	Tumor size (low/high)	LNC00673 high expression		LNC00673 low expression		HR (95% CI)	Cutoff	Method	NOS		
							Total	LNM	DM	Total					LNM	DM
Feng (2018) [14]	China	CRC	71	>60 (19/18) ≤60 (16/18)	20/23 15/13	≤5 cm (23/10) >5 cm (12/26)	36	21	14	35	10	5	2.43 (1.02–5.81)	Median	Q-PCR	6
Shi (2016) [15]	China	NSCLC	80	<65 (17/22) ≥65 (22/19)	31/27 8/14	<5 cm (27/17) ≥5 cm (12/24)	41	23	—	39	13	—	NR	Q-PCR	Q-PCR	6
Tan (2017) [16]	China	NSCLC	76	>60 (17/19) ≤60 (21/19)	32/25 6/13	≤3 cm (6/12) >3 cm (32/26)	—	—	—	—	—	—	Median	Q-PCR	Q-PCR	6
Zheng (2019) [17]	China	EOC	131	<50 (18/23) ≥50 (47/43)	—	<1 cm (57/64) ≥1 cm (8/2)	66	44	—	65	23	—	1.66 (0.23–11.91)	Median	Q-PCR	7
Ba (2017) [18]	China	GC	79	≤55 (18/31) >55 (15/15)	17/31 16/15	<5 cm (23/26) >5 cm (10/20)	46	37	25	33	13	9	1.95 (1.11–3.45)	Median	Q-PCR	6
Huang (2017) [19]	China	GC	73	>65 (18/16) ≤65 (25/14)	23/19 20/11	≤5 cm (28/11) >5 cm (15/19)	30	24	—	40	24	—	3.81 (1.74–8.32)	FC>2	Q-PCR	6
Xia (2018) [20]	China	THCA	60	≤45 (15/11) >45 (15/19)	15/9 15/21	≤10 cm (15/6) >10 cm (15/24)	30	21	1	30	11	3	—	Median	Q-PCR	7
Yu (2016) [21]	China	TSCC	202	≤50 (44/62) >50 (48/48)	76/102 16/8	T1 (19/11) T2–T4 (73/99)	110	50	0	92	28	0	1.79 (1.00–3.21)	NR	Q-PCR	6
Zhang (2018) [22]	China	PC	229	>60 (51/57) ≤60 (58/63)	—	—	120	59	43	109	47	30	1.91 (1.06–3.46)	NR	Q-PCR	7
Qiao (2019) [23]	China	BC	80	<50 (17/21) ≥50 (23/19)	—	≤2 cm (21/10) >2 cm (19/30)	40	30	—	40	23	—	1.81 (0.44–7.46)	NR	Q-PCR	6
Xia (2018) [24]	China	BC	35	≤60 (10/18) >60 (3/4)	—	≤5 cm (13/20) >5 cm (0/2)	22	15	—	13	4	—	Median	Q-PCR	Q-PCR	6
Zhou (2020) [25]	China	ESCC	39	>60 (18/11) ≤60 (29/23)	33/27 14/7	≤5 cm (7/12) >5 cm (40/22)	—	—	—	—	—	—	Median	Q-PCR	Q-PCR	7

CRC, colorectal cancer; NSCLC, nonsmall cell lung cancer; EOC, epithelial ovarian cancer; GC, gastric cancer; THCA, thyroid carcinoma; TSCC, tongue squamous cell carcinoma; PC, pancreatic cancer; BC, breast cancer; LNM, lymph node metastasis; DM, distant metastasis; HR, hazard ratio; NR, not reported; FC, fold change; qRT-PCR, quantitative real-time-polymerase chain reaction; NOS, Newcastle–Ottawa Scale.

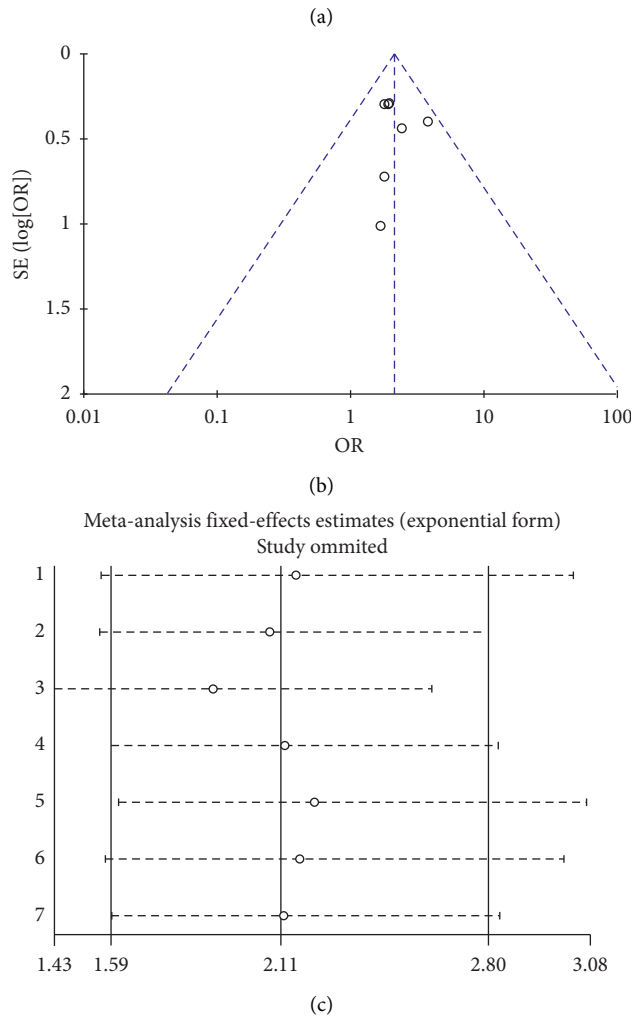
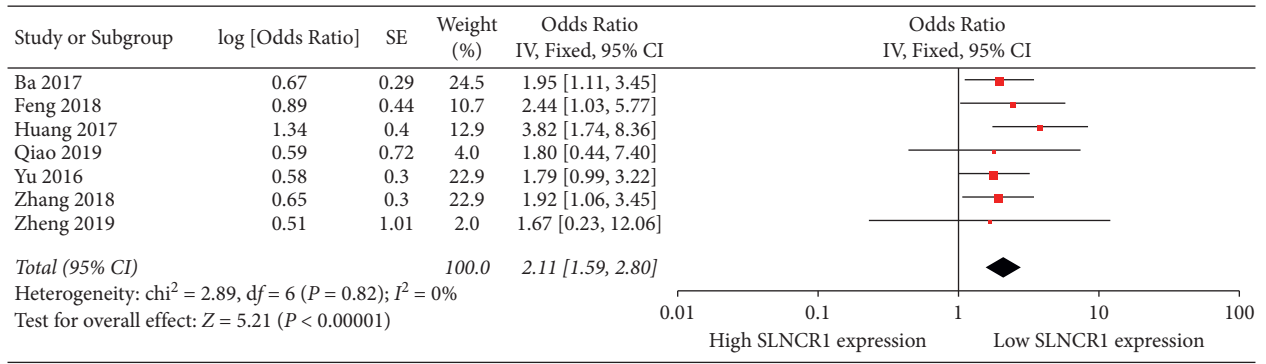


FIGURE 2: The relation between SLNCR1 expression and overall survival. (a) Forest plot. (b) Funnel plot. (c) Sensitivity analysis.

study by Ba et al. [18], the overall risk became worthy of suspicion, the OR was reduced from 1.85 (95% CI: 1.12–3.06) to 1.65 (95% CI: 0.99–2.73), and strong heterogeneity persisted (from $I^2 = 64\%$ to $I^2 = 60\%$). Additionally, the study by Yu et al. [21] deeply impacted the overall risk in the TNM analysis group. The overall risk changed from OR 1.72 (95% CI: 1.08–2.74) to 1.67 (95% CI: 0.96–2.92) after excluding this study, and heterogeneity was almost unchanged (from $I^2 = 62\%$ to $I^2 = 66\%$).

4. Discussion

LncRNAs have been regarded as accidental “transcriptional noise” with little function due to lack of protein-coding capability [26]. Recently, accumulating evidence has shown that LncRNAs may regulate genes or miRNA expression and act as oncogenic or tumor suppressors [27, 28]. Some LncRNAs have been shown to act as competing endogenous RNA by regulating miRNA target genes indirectly. With the

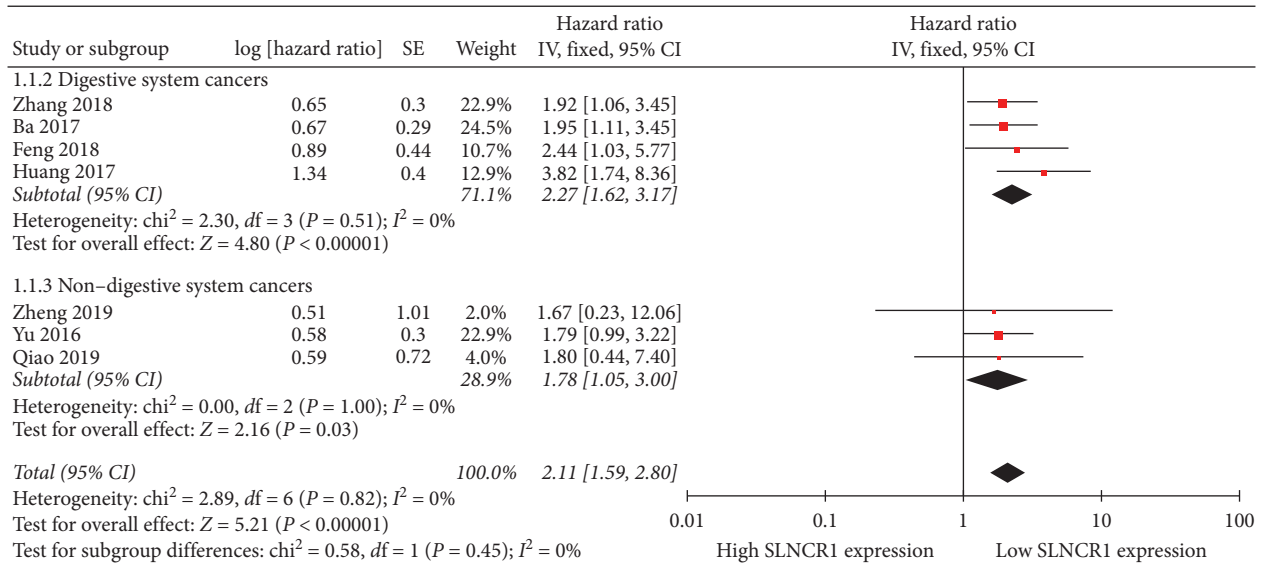


FIGURE 3: Forest plot for the relation between SLNCR1 expression and overall survival based on different types of cancers.

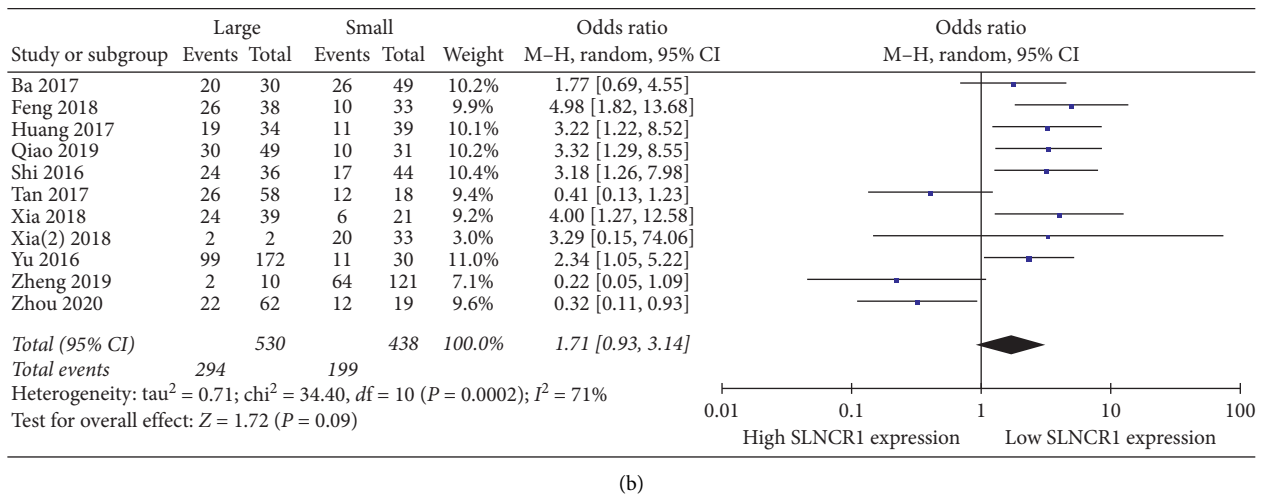
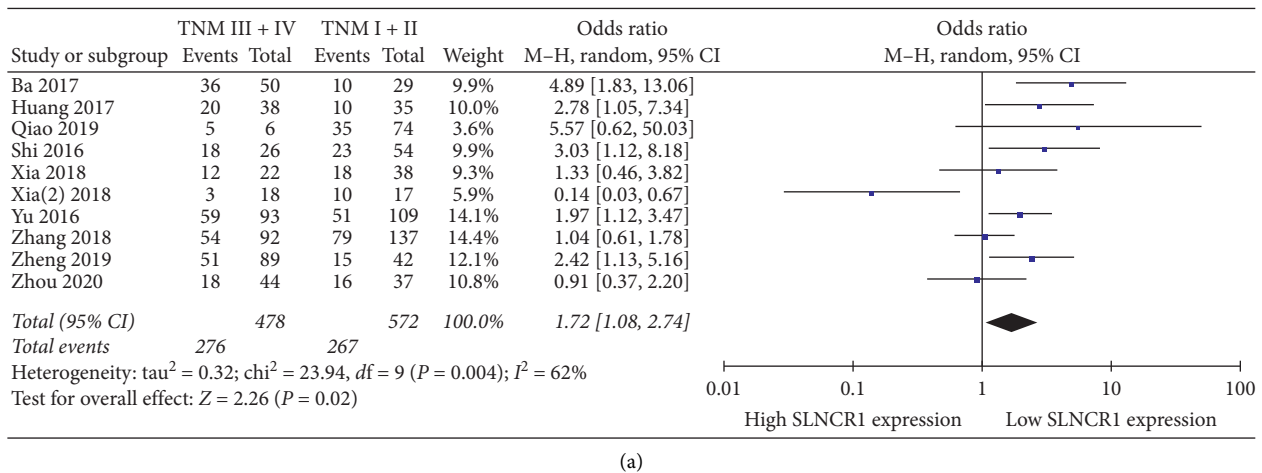
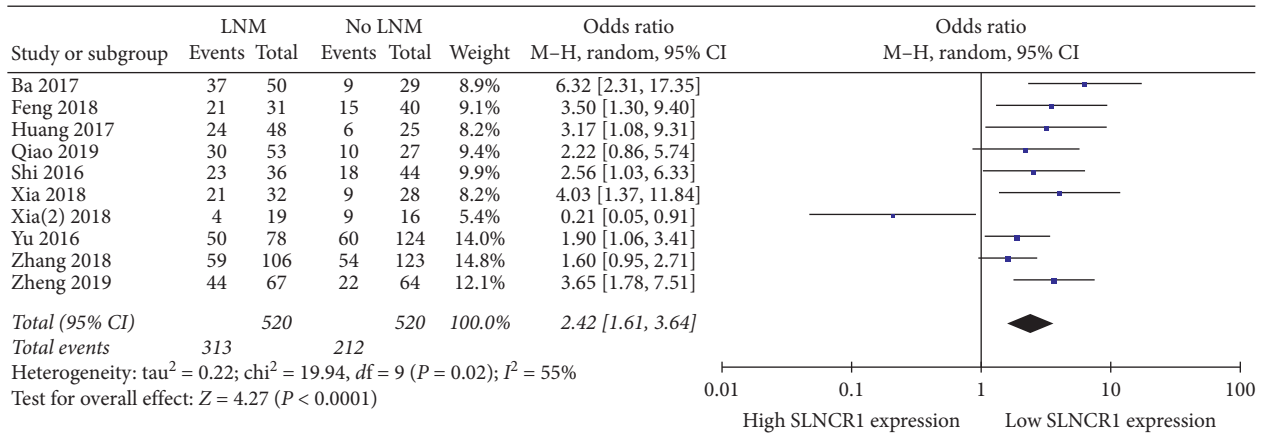
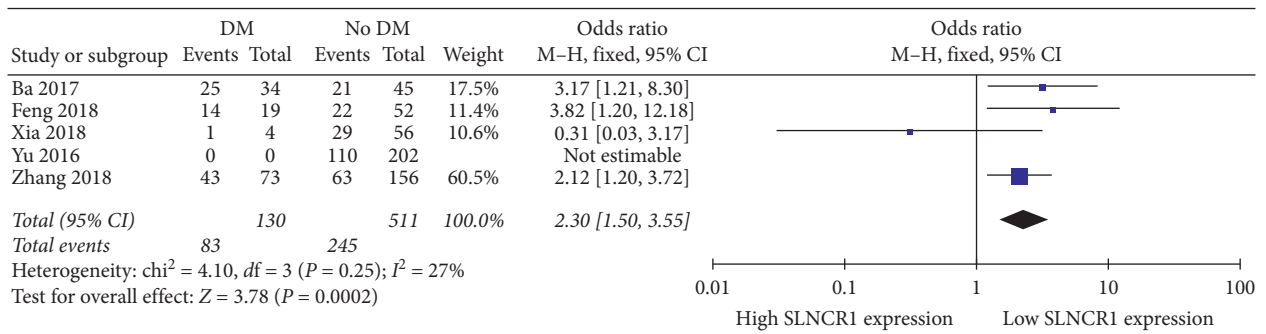


FIGURE 4: Forest plot for the relation between SLNCR1 expression and clinicopathological characteristics. (a) TNM. (b) Tumor size.

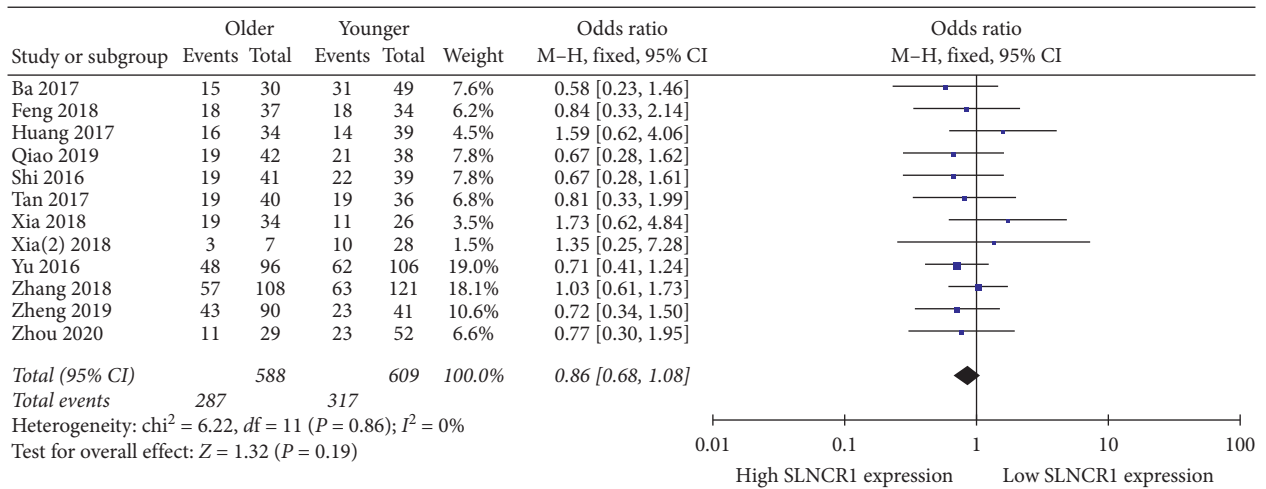


(a)



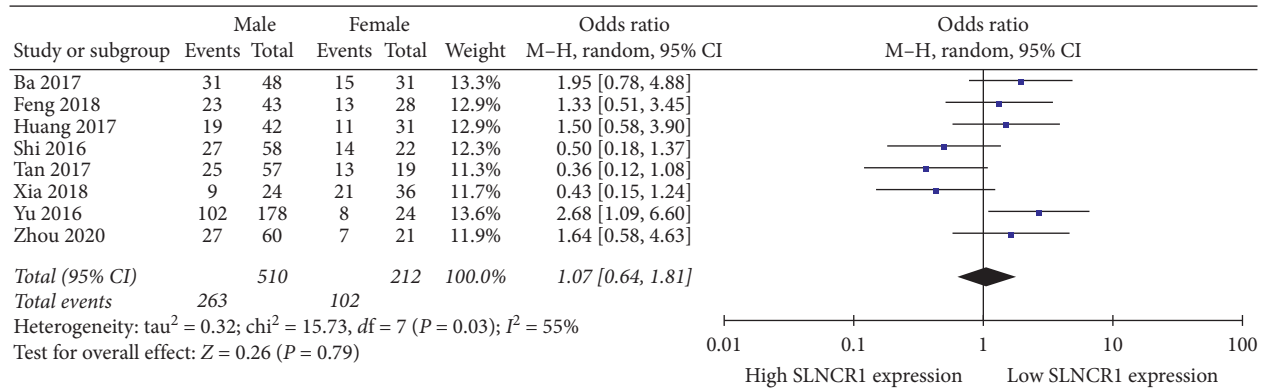
(b)

FIGURE 5: Forest plot for the relation between SLNCR1 expression and metastasis. (a) LNM. (b) DM.



(a)

FIGURE 6: Continued.



(b)

FIGURE 6: Forest plot for the relation between SLNCR1 expression and clinical features. (a) Age. (b) Gender.

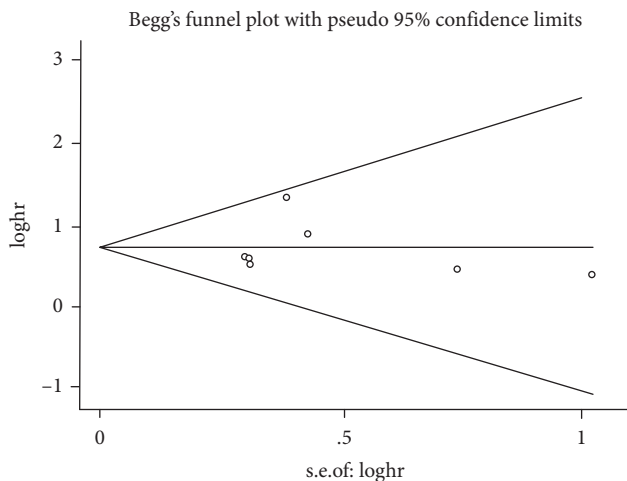


FIGURE 7: Begg's funnel plot for the evaluation of potential publication bias in the impact of SLNCR1 on overall survival.

development of high-throughput genome sequencing technologies, lncRNAs have been identified as new biomarkers for the accurate prognosis of various kinds of tumors due to their functions in tumor proliferation, invasion, migration, and metastasis.

SLNCR1, a lncRNA with high accuracy prognostic value, has been demonstrated to be associated with tumorigenesis and progression and was initially associated with decreased melanoma patient survival. Brain-specific homeobox protein 3a and androgen receptors bind within SLNCR1's conserved region, activating matrix metalloproteinase 9 and subsequently increasing malignant melanoma invasion [6]. Furthermore, SLNCR1 may regulate cell migration, invasion, and stemness through interactions with secretory sPLA2 in nonsmall cell lung cancer [29]. It has been shown to promote the proliferation of breast cancer by sponging miR-515-5p to regulate MARK4 expression and inhibit the Hippo signaling pathway [23]. An increasing number of studies have explored SLNCR1 interaction partners and biological functions in various types of cancers, but the relationship

between the expression of SLNCR1 and tumor progression remains poorly understood.

The current study presents the first meta-analysis to evaluate the relationship between SLNCR1 expression and the prognosis of cancers. The results indicate that patients with high expression levels of SLNCR1 tend to have poorer OS than those with low expression levels. In other words, the high expression level of SLNCR1 is a predictor of a negative prognosis of cancer. Meanwhile, subgroup analysis in the fixed model was performed to assess the role of SLNCR1 in digestive system cancers. The data show that in both digestive and nondigestive system tumors, high SLNCR1 expression was associated with poor prognosis. Furthermore, the results also indicate that tumor stage and the high SLNCR1 expression group were markedly higher, and high SLNCR1 expression was correlated with greater susceptibility to LNM and DM. No relationship between SLNCR1 expression and tumor size and clinical features (age and gender) was observed. Moreover, the cutoff values varied among different studies, which might have caused heterogeneity in the results. In order to clarify the source of heterogeneity, we divided the comparison into subgroups with different cutoff values and analyzed the heterogeneity. As shown in Supplementary Materials (available here), the results showed no heterogeneity changes in OS, TNM, tumor size, LNM, DM, age, and gender. Thus, the cause of heterogeneity remains unclear.

Several limitations regarding this meta-analysis should be taken into account. Initially, the HRs and 95% CIs were extracted from Kaplan-Meier curves; lacking sufficient survival data may have led to extraneous heterogeneity. Second, all of the patients were from China, and the results may not represent the global population.

Furthermore, only 12 studies with 1155 patients were involved in the present meta-analysis. Thus, the small sample size of the study may have reduced the stringency of the conclusion. Finally, age and tumor size were defined by the ranges given in the included studies, and different studies had varying criteria for evaluating these parameters. Thus, more rigorous research studies are needed to confirm our conclusions.

In summary, high SLNCR1 expression was associated with poor OS, advanced tumor stage, LNM, and DM in multiple cancers. Thus, the results of our meta-analysis indicate that SLNCR1 may serve as a prognosis biomarker for cancer patients in China.

Abbreviations

SLNCR1: Steroid receptor RNA activator (SRA)-like noncoding RNA
 HR: Hazard ratio
 lncRNA: Long noncoding RNA
 OR: Odds ratio
 OS: Overall survival
 LNM: Lymph node metastasis
 DM: Distant metastasis.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Consent

Not applicable.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

LLC and HYS conceived and designed the current study. HM and QS conducted data collection and extraction. LLC analyzed the data. LLC, YZ, XLC, and RHJ drafted the manuscript. The author read and approved the final manuscript.

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Supplementary Materials

Subgroups were established to analyze the heterogeneity according to different cutoff values. (*Supplementary Materials*)

References

- [1] L. A. Torre, F. Islami, R. L. Siegel, E. M. Ward, and A. Jemal, "Global cancer in women: burden and trends," *Cancer Epidemiology Biomarkers & Prevention*, vol. 26, no. 4, pp. 444–457, 2017.
- [2] Y. Zhang, R. Yang, J. Lian, and H. Xu, "LncRNA Sox2ot overexpression serves as a poor prognostic biomarker in gastric cancer," *American Journal of Tourism Research*, vol. 8, pp. 5035–5043, 2016.
- [3] J. Wang, Z. Su, S. Lu et al., "LncRNA HOXA-AS2 and its molecular mechanisms in human cancer," *Clinica Chimica Acta*, vol. 485, pp. 229–233, 2018.
- [4] T. R. Mercer, M. E. Dinger, and J. S. Mattick, "Long non-coding RNAs: insights into functions," *Nature Reviews Genetics*, vol. 10, no. 3, pp. 155–159, 2009.
- [5] G. Yang, X. Lu, and L. Yuan, "LncRNA: a link between RNA and cancer," *Biochimica et Biophysica Acta (BBA) - Gene Regulatory Mechanisms*, vol. 1839, no. 11, pp. 1097–1109, 2014.
- [6] K. Schmidt, C. E. Joyce, F. Buquicchio et al., "The lncRNA SLNCR1 mediates melanoma invasion through a conserved SRA1-like region," *Cell Reports*, vol. 15, no. 9, pp. 2025–2037, 2016.
- [7] Y. Wu, Y. Niu, J. Leng et al., "Benzo(a)pyrene regulated A549 cell migration, invasion and epithelial-mesenchymal transition by up-regulating long non-coding RNA linc00673," *Toxicology Letters*, vol. 320, pp. 37–45, 2020.
- [8] U. Abdul-Rahman, B. Györfy, and B. D. Adams, "Linc00673 (ERRLR01) is a prognostic indicator of overall survival in breast cancer," *Transcription*, vol. 9, no. 1, pp. 17–29, 2018.
- [9] J. Zheng, X. Huang, W. Tan et al., "Pancreatic cancer risk variant in LINC00673 creates a miR-1231 binding site and interferes with PTPN11 degradation," *Nature Genetics*, vol. 48, no. 7, pp. 747–757, 2016.
- [10] Y. Wang and T. Luo, "LINC00673 rs11655237 polymorphism is associated with increased risk of cervical cancer in a Chinese population," *Cancer Control: Journal of the Moffitt Cancer Center*, vol. 25, Article ID 1073274818803942, 2018.
- [11] J. F. Tierney, L. A. Stewart, D. Ghersi, S. Burdett, and M. R. Sydes, "Practical methods for incorporating summary time-to-event data into meta-analysis," *Trials*, vol. 8, Article ID 16, 2007.
- [12] G. A. S. B. Wells, D. O'Connell, J. Peterson, V. Welch, M. Losos, and P. Tugwell, *The Newcastle-Ottawa Scale (NOS) for Assessing the Quality of Nonrandomised Studies in Meta-Analyses*, Ottawa Health Research Institute University of Ottawa, Ottawa, Canada, 2000.
- [13] R. DerSimonian and N. Laird, "Meta-analysis in clinical trials," *Controlled Clinical Trials*, vol. 7, no. 3, pp. 177–188, 1986.
- [14] L. M. Feng, D. W. Zhao, S. J. Li, and J. Huang, "Association of the upregulation of LncRNA00673 with poor prognosis for colorectal cancer," *European Review for Medical and Pharmacological Sciences*, vol. 22, pp. 687–694, 2018.
- [15] X. Shi, C. Ma, Q. Zhu et al., "Upregulation of long intergenic noncoding RNA 00673 promotes tumor proliferation via LSD1 interaction and repression of NCALD in non-small-cell lung cancer," *Oncotarget*, vol. 7, no. 18, pp. 25558–25575, 2016.
- [16] Q. Tan, Y. Yu, N. Li et al., "Identification of long non-coding RNA 00312 and 00673 in human NSCLC tissues," *Molecular Medicine Reports*, vol. 16, no. 4, pp. 4721–4729, 2017.
- [17] T. Zheng, J. Qiu, C. Li, X. Lin, X. Tang, and K. Hua, "Long noncoding RNA LINC00673 promotes the proliferation and metastasis of epithelial ovarian cancer by associating with opioid growth factor receptor," *OncoTargets and Therapy*, vol. 12, pp. 6145–6156, 2019.
- [18] M.-C. Ba, H. Long, S.-Z. Cui et al., "Long noncoding RNA LINC00673 epigenetically suppresses KLF4 by interacting with EZH2 and DNMT1 in gastric cancer," *Oncotarget*, vol. 8, no. 56, pp. 95542–95553, 2017.
- [19] M. Huang, J. Hou, Y. Wang et al., "Long noncoding RNA LINC00673 is activated by SP1 and exerts oncogenic

- properties by interacting with LSD1 and EZH2 in gastric cancer,” *Molecular Therapy*, vol. 25, no. 4, pp. 1014–1026, 2017.
- [20] E. Xia, A. Bhandari, Y. Shen, X. Zhou, and O. Wang, “lncRNA LINC00673 induces proliferation, metastasis and epithelial-mesenchymal transition in thyroid carcinoma via Kruppel-like factor 2,” *International Journal of Oncology*, vol. 53, pp. 1927–1938, 2018.
- [21] J. Yu, Y. Liu, Z. Gong et al., “Overexpression long non-coding RNA LINC00673 is associated with poor prognosis and promotes invasion and metastasis in tongue squamous cell carcinoma,” *Oncotarget*, vol. 8, no. 10, pp. 16621–16632, 2017.
- [22] B. Zhang, C. Li, and Z. Sun, “Long non-coding RNA LINC00346, LINC00578, LINC00673, LINC00671, LINC00261, and SNHG9 are novel prognostic markers for pancreatic cancer,” *American Journal of Tourism Research*, vol. 10, pp. 2648–2658, 2018.
- [23] K. Qiao, S. Ning, L. Wan et al., “LINC00673 is activated by YY1 and promotes the proliferation of breast cancer cells via the miR-515-5p/MARK4/Hippo signaling pathway,” *Journal of Experimental & Clinical Cancer Research*, vol. 38, no. 1, Article ID 418, 2019.
- [24] E. Xia, Y. Shen, A. Bhandari et al., “Long non-coding RNA LINC00673 promotes breast cancer proliferation and metastasis through regulating B7-H6 and epithelial-mesenchymal transition,” *American journal of cancer research*, vol. 8, pp. 1273–1287, 2018.
- [25] M. Zhou, Y. Mao, S. Yu et al., “LINC00673 represses CDKN2C and promotes the proliferation of esophageal squamous cell carcinoma cells by EZH2-mediated H3K27 trimethylation,” *Frontiers in Oncology*, vol. 10, Article ID 1546, 2020.
- [26] J. E. Wilusz, H. Sunwoo, and D. L. Spector, “Long noncoding RNAs: functional surprises from the RNA world,” *Genes & Development*, vol. 23, no. 13, pp. 1494–1504, 2009.
- [27] Y. Kondo, K. Shinjo, and K. Katsushima, “Long non-coding RNAs as an epigenetic regulator in human cancers,” *Cancer Science*, vol. 108, no. 10, pp. 1927–1933, 2017.
- [28] N. Li, K. Shi, and W. Li, “TUSC7: a novel tumor suppressor long non-coding RNA in human cancers,” *Journal of Cellular Physiology*, vol. 233, no. 9, pp. 6401–6407, 2018.
- [29] W. Xu, Q. Xu, D. Kuang et al., “Long non-coding RNA SLNCR1 regulates non-small cell lung cancer migration, invasion and stemness through interactions with secretory phospholipase A2,” *Molecular Medicine Reports*, vol. 20, pp. 2591–2596, 2019.