Systematic Review and Meta-Analysis

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LncRNA PCAT-1 in gastrointestinal cancers

A meta-analysis

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Abstract

Background: Prostate-cancer-associated ncRNA transcript 1 (PCAT-1), a newly discovered lncRNA, was implicated in the progression of multiple tumors. We conducted a systematic review and meta-analysis to determine its prognostic potential for gastrointestinal cancers.

Methods: A literature survey was conducted by searching the PubMed, Web of Science, Cochrane Library, Embase together with Wanfang, and China National Knowledge Infrastructure (CNKI) database for articles published as of October 15, 2017. Hazard ratio (HR) or odds ratio (OR) with 95% confidence intervals (95% CIs) were calculated to demonstrate prognostic value of PCAT-1 using Stata 12.0 software.

Results: A total of 6 studies with 961 cases were pooled in the analysis to evaluate the prognostic value of PCAT-1 in gastrointestinal cancers. Increased PCAT-1 expression was significantly correlated with poor overall survival (OS) (HR = 1.04, 95% CI: 1.02–1.06). Statistical significance was also observed in subgroup meta-analysis stratified by cancer type, histology type, sample size, and analysis type. Additionally, high expression of PCAT-1 was significantly associated with deeper tumor invasion (OR = 4.46, 95% CI: 3.00–6.63), positive lymph node metastasis (OR = 3.76, 95% CI: 1.39–10.16), and advanced clinical stage (OR = 4.09, 95% CI: 1.55–10.82).

Conclusion: High expression of PCAT-1 was related to poor prognosis and could be a promising biomarker of clinicopathologic features in gastrointestinal cancers. More studies will be necessary to verify and strengthen the clinical value of PCAT-1 in gastrointestinal cancers.

Abbreviations: 95% CI = 95% confidence interval, CNKI = China National Knowledge Infrastructure, CRC = colorectal cancer, ESCC= esophageal squamous cell carcinoma, GC = gastric cancer, HCC = hepatocellular carcinoma, HE = high expression, HR = hazard ratio, MVA = multivariate analysis, NA = not available, NOS = Newcastle–Ottawa scale, OR = odds ratio, OS = overall survival, PCAT-1 = prostate-cancer-associated ncRNA transcripts 1, RT-qPCR = real-time quantitative polymerase chain reaction.

Keywords: gastrointestinal cancer, lncRNA, meta-analysis, prostate-cancer-associated ncRNA transcript 1, prognosis

1. Introduction

Gastrointestinal malignancies are the major and complex diseases in the world, which have caused a great burden on human health, families, and society.[1,2] They originated from the gastrointestinal tract or accessory organs of digestion. The patients diagnosed with such kind cancers usually have an unfavorable prognosis, especially for 5-year survival rate. Over the past few decades, many studies have focused on searching promising novel biomarkers for gastrointestinal cancers.[3–5] Exploring early diagnosis and prognosis tumor-markers is important and also in urgent need.

Long noncoding RNAs (lncRNAs), as rising stars in recent years, have attracted mountains of attention for their vital roles in diverse biologic processes.[6–8] Through lncRNAs are a group of noncoding RNAs with over 200 nucleotides in length but without protein-coding ability, more and more lncRNAs were identified and reported to function as oncogene or tumor suppressor factor in tumorigenesis and cancer progression.[9–11] Furthermore, they might act as diagnostic, prognostic biomarkers, or therapeutic targets in human cancers.[12–15]

Prostate-cancer-associated ncRNA transcript 1 (PCAT-1) was a novel identified lncRNA. It was firstly found in prostate cancer and was reported playing an active role in promoting prostate cancer cell proliferation.[16] In recent years, abnormal expression of PCAT-1 was found in multiple cancers and involved in the progression of various tumors, such as breast cancer, bladder cancer, glioblastoma, and nonsmall-cell lung cancer.[17–20] Notably, the role of PCAT-1 in gastrointestinal cancers has aroused considerable interest. Researchers reported that PCAT-1 was implicated in tumor invasion and metastasis,[21–23] and found correlations between PCAT-1 expression and clinical outcomes in multiple gastrointestinal malignancies.[24–26] However, until now there was no meta-analysis systematically elucidating the prognostic value of PCAT-1 in gastrointestinal tumors, and considering the limitations associated with specimen

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sizes or study methodology of the single study. Consequently, we performed this study to explore the clinical values of PCAT-1 in gastrointestinal cancers by gathering all related published data.

2. Materials and methods

2.1. Publication search

Since this is a meta-analysis, ethical approval was not needed. To identify potentially eligible articles, a comprehensive literature search of PubMed, Web of Science, Cochrane Library, Embase together with Wanfang, and China National Knowledge Infrastructure (CNKI) database was performed prior to October 15, 2017. The following keywords were used for searching: PCAT-1 or PCA1 or PCAT1 or prostate-cancer-associated ncRNA transcript 1 or prostate-cancer-associated transcript-1. The reference lists of relevant literature were manually searched for additional eligible articles.

2.2. Inclusion and exclusion criteria

Inclusion criteria are as the following: studies detecting the expression of PCAT-1 in gastrointestinal cancers; the association between PCAT-1 expression and overall survival (OS) was investigated; sufficient survival data were provided for the hazard ratio (HR) with 95% confidence interval (CI); and patients were divided into 2 groups.

The following studies were excluded: duplicate publications; those on non gastrointestinal tumors, or animal experiments; studies investigating the molecular structure and functions of PCAT-1 without survival outcome; and reviews, letters, case reports, conference abstracts, or editorials.

2.3. Data extraction

The following data and information were collected from all eligible studies: publication information: name of first author, publication year, country; Patients' characteristics: cancer type, number of patients, expression pattern, follow-up duration, gender, histologic grade, tumor depth, lymph node metastasis, distant metastasis and clinical stage; PCAT-1 expression measurement and cut-off value; and HRs of PCAT-1 for OS as well as their 95% CIs and *P*-values.

If only Kaplan–Meier curves were available, we extracted data from the graphical survival plots and estimated the HRs. If a study reported the data in multivariate analysis or/and univariate analysis for OS, the former was directly applied.

The Newcastle–Ottawa quality assessment scale (NOS) used to assess the quality of enrolled studies, with the score ranging from 0 to 9 points in the method. A study with a score ≥6 was considered high quality.

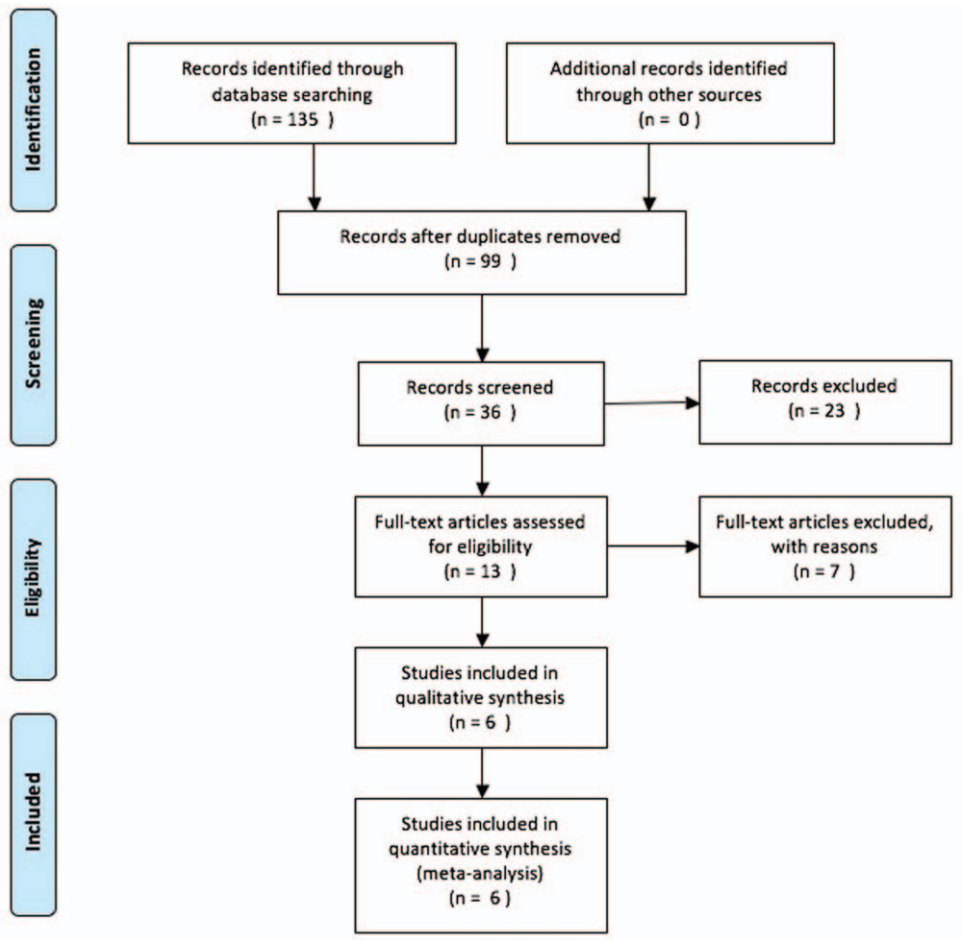

Figure 1. Flow diagram of the study search and selection process.

2.4. Statistical analysis

All statistical analyses were executed using STATA statistical software version 12.0 (STATA, College Station, TX) in this meta-analysis. Heterogeneity across studies was quantified with the I^2 statistics and Cochran Q test. The random-effects model was conducted to analyze the relationship between PCAT-1 expression and clinical outcomes when calculated I^2 values $>50\%$ or/and $P_h < .1$. If there was no significant heterogeneity among studies, the fixed effects model was applied. Probable publication bias was displayed by constructing a funnel plot and conducting Begg test. Sensitivity analysis was used to evaluate the robustness of the pooled results. A P-value of $<.05$ was considered statistically significant.

3. Results

3.1. Included literatures

As shown in the flow diagram (Fig. 1), a total of 135 studies were initially identified as appropriate from PubMed, Web of Science, Cochrane Library, Embase, Wanfang, and CNKI database. After excluding duplicates, 36 records were reserved. And after carefully screening those titles and abstracts, 23 irrelevant articles were removed. From the 13 remaining articles, 7 were excluded because of incomplete data or absence of survival outcome. Ultimately, a total of 6 studies[24–29] were included in this meta-analysis according to the selection criteria.

3.2. Characteristics of the enrolled studies

The main features of the 6 enrolled studies are summarized in Table 1. All those publications were written in English with the released period from 2013 to 2017. There were totally 961 patients with median sample size of 160.2, with a wide range from 108 to 321. Four different kinds of gastrointestinal cancers were evaluated in this meta-analysis: 2 esophageal squamous cell carcinoma (ESCC), 2 gastric cancer (GC), 1 colorectal cancers (CRCs), and 1 hepatocellular carcinoma (HCC). All detected samples were fresh or frozen tissues from the patients without any preoperative treatments. The expression of PCAT-1 was measured by RT-qPCR. All are retrospective studies regarding relevance between PCAT-1 expression and gastrointestinal cancers prognosis. In this meta-analysis, the quality scores of all eligible studies were varied from 6 to 9, with a mean value of 7.5.

3.3. Results of the meta-analysis

3.3.1. Relationship between lncRNA PCAT-1 and OS. All included studies comprising 961 patients reported the relationship between lncRNA PCAT-1 and OS in gastrointestinal cancers. No significant heterogeneity across-studies was found ($I^2 = 40.6\%$; $P_h = .135$), so the fixed effects model was used to estimate the pooled HR. The pooled results showed that high expression of PCAT-1 in cancer tissues was significantly correlated with poor OS in gastrointestinal cancers (HR = 1.04, 95% CI: 1.02–1.06, $P < .001$) (Fig. 2). The patients with high PCAT-1 had a worse OS than those with low expression of PCAT-1. PCAT-1 might be a significant prognostic factor of OS for gastrointestinal cancer patients.

3.3.2. Subgroup analysis of PCAT-1 in OS. Subgroup analyses for OS were also performed. As the results showed in Table 2, compared with the merged HR, high PCAT-1 showed a stronger association with unfavorable OS in the subgroups of GC (HR = 1.05, 95% CI: 1.02–1.08, $P < .001$), and ESCC (HR = 1.04, 95% CI: 1.01–1.06, $P < .001$). In addition, the pooled HRs was significantly and consistently >1 in subgroup meta-analysis stratified by the histology type and sample size. Furthermore, PCAT-1 high expression was an unfavorable independent prognostic factor for OS based on multivariate analysis (HR = 1.04, 95% CI: 1.01–1.07, $P < .001$).

3.3.3. Relationship between lncRNA PCAT-1 and clinicopathologic features. Pooled odds ratio (OR) for lncRNA PCAT-1 expression, presented in Table 3, showed that high expression of lncRNA PCAT-1 significantly correlated with depth of tumor invasion (OR = 4.46, 95% CI: 3.00–6.63, $P < .00001$), lymph node metastasis (OR = 3.76, 95% CI: 1.39–10.16, $P = .009$), and tumor stage (OR = 4.09, 95% CI: 1.55–10.82, $P = .004$). However, PCAT-1 expression was not associated with gender (OR = 0.83, 95% CI: 0.60–1.15, $P = .26$), differentiation (OR = 1.20, 95% CI: 0.78–1.83, $P = .40$), or distant metastasis (OR = 1.49, 95% CI: 0.70–3.15, $P = .30$).

3.3.4. Publication bias. Begg test was used to assess the publication bias. Begg funnel plot with pseudo 95% CI was provided (Fig. 3). No significant publication bias affected the analysis of OS ($Pr > |z| = 0.188$).

3.3.5. Sensitivity analysis. As illustrated in Figure 4, the result for sensitivity analysis for OS was negative, revealing that our results were relatively robust.

4. Discussion

The PCAT-1 is located in the chromosome 8q30 gene desert approximately 725 kb upstream of the c-MYC oncogene.[16] As a new identified prostate-cancer-associated lncRNA transcript 1, it was firstly reported to be implicated in prostate cancer progression and contributed to cell proliferation in prostate

Table 1

Main characteristics of the enrolled studies in the meta-analysis.

First author, y	Type	Country	No. of sample	HE (N, %)	Tumor stage	Follow-up	Criterion of HE	Detection method	Outcome measures	MVA	NOS
Bi M, 2017 [24]	GC	China	110	57 (51.82)	I–IV	≥5 y	NA	qRT-PCR	OS	No	6
Cui WC, 2017 [25]	GC	China	175	88 (50.28)	I–IV	≥5 y	Median expression (0.98 fold)	qRT-PCR	OS	Yes	7
Qin HD, 2016 [26]	ESCC	China	321	250 (77.88)	I–IV	≥5 y	NA	qRT-PCR	OS	Yes	9
Shi WH, 2015 [27]	ESCC	China	130	65 (50.00)	0–III	≥5 y	A median value ≥1.93	qRT-PCR	OS	Yes	8
Yan TH, 2015 [28]	HCC	China	117	59 (50.43)	I–IV	≥5 y	PCAT-1 expression ratio ≥ median ratio	qRT-PCR	OS	Yes	7
Ge X, 2013 [29]	CRC	China	108	50 (46.30)	NA	≥5 y	Receiver operating characteristic curves	qRT-PCR	OS	Yes	8

CRC = colorectal cancer, ESCC = esophageal squamous cell carcinoma, GC = gastric cancer, HCC = hepatocellular carcinoma, HE = high expression, MVA = multivariate analysis, NA = not available, OS = overall survival, qRT-PCR = real-time quantitative polymerase chain reaction.

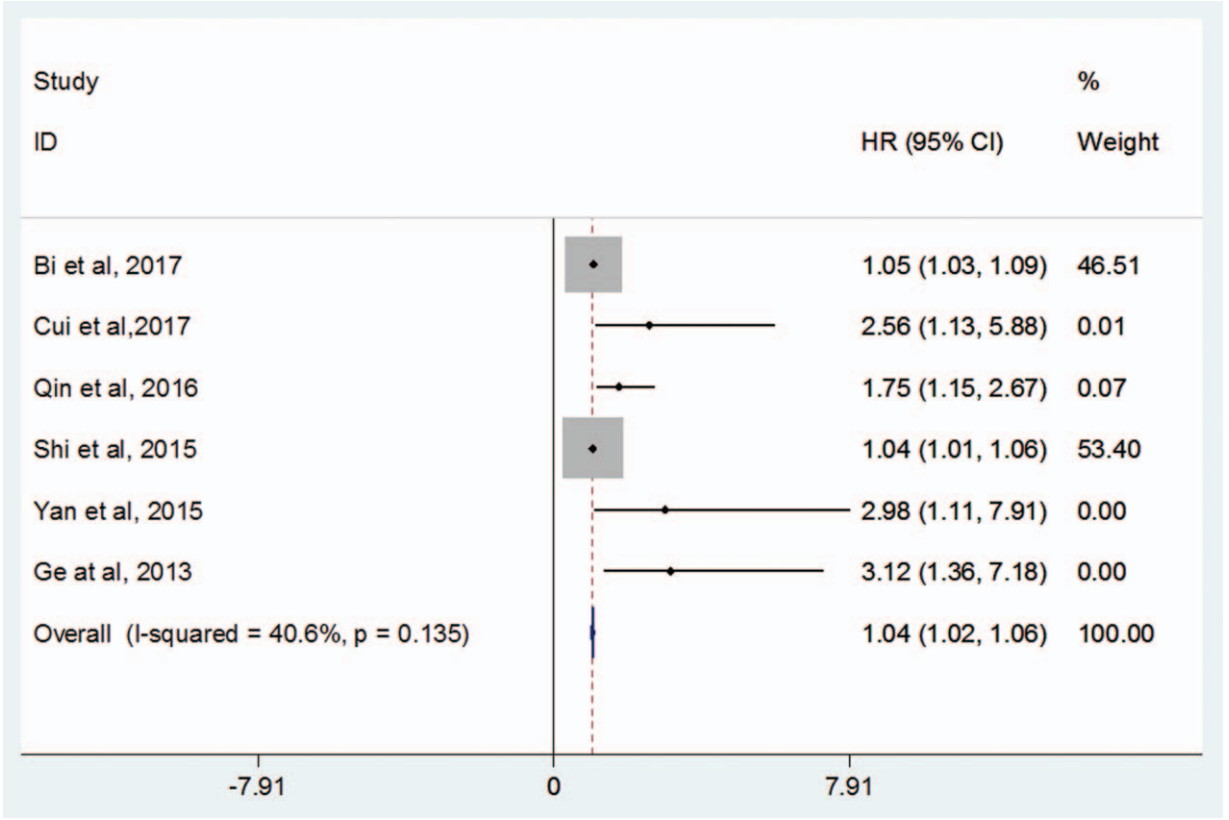

Figure 2. Forest plot of the relationships between prostate-cancer-associated ncRNA transcript 1 (PCAT-1) and overall survival (OS).

Table 2

Results of subgroup analysis of pooled HRs of OS of cancer patients with high PCAT-1 expression.

Stratified analysis	No. of Studies	No. of patients	Pooled HR (95% CI)	*P*	Heterogeneity I^2 (%)	Heterogeneity P_h
Cancer type						
GC	2[24,25]	285	1.05 (1.02–1.08)	<.001	35.5	.213
ESCC	2[26,27]	451	1.04 (1.01–1.06)	<.001	70.5	.066
Histology type						
Squamous cell carcinoma	2[26,27]	451	1.04 (1.01–1.06)	<.001	70.5	.066
Nonsquamous cell carcinoma	4 [24,25,28,29]	510	1.05 (1.02–1.08)	<.001	36.5	.193
Sample size						
≥120	3[25–27]	626	1.04 (1.01–1.07)	<.001	59.7	.084
<120	3[24,28,29]	335	1.05 (1.02–1.08)	<.001	37.0	.204
Analysis type						
Multivariate analysis	5[25–29]	851	1.04 (1.01–1.07)	<.001	51.0	.086

95% CI = 95% confidence interval, ESCC = esophageal squamous cell carcinoma, GC = gastric cancer, HR = hazard ratio.

Table 3

Results of meta-analysis of high PCAT-1 and clinicopathologic features.

Category	Studies (n)	No. of patients	OR (95% CI)	*P*	Heterogeneity I^2 (%)	Heterogeneity P_h	Model
Gender (male vs female)	5[24,25,27–29]	640	0.83 (0.60–1.15)	.26	0	.43	Fixed effects
Differentiation (poorly/undifferentiated vs well + moderately)	3[25,28,29]	400	1.20 (0.78–1.83)	.40	0	.37	Fixed effects
Tumor depth (T3–4 vs Tis-2)	4[24,25,27,29]	523	4.46 (3.00–6.63)	<.00001	44	.15	Fixed effects
Lymph node metastasis (+ vs −)	4[24,25,27,29]	523	3.76 (1.39–10.16)	.009	85	.0002	Random effects
Distant metastasis (+ vs −)	5[24,25,27–29]	630	1.49 (0.70–3.15)	.30	57	.05	Random effects
Tumor stage (III–IV vs 0–II)	4[24,25,27,28]	532	4.09 (1.55–10.82)	.004	82	.001	Random effects

95% CI = 95% confidence interval, OR = odds ratio.

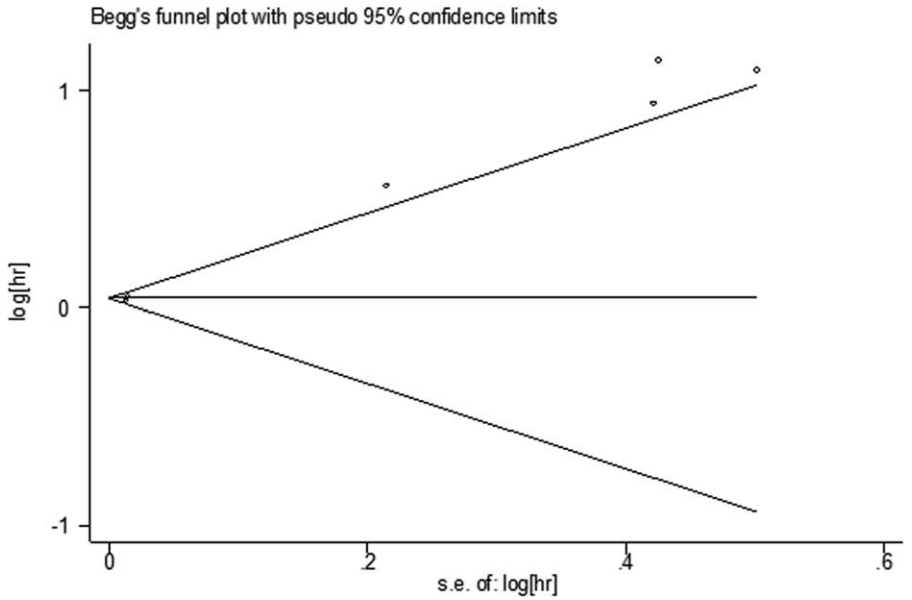

Figure 3. Funnel plots for publication bias test for overall survival (OS).

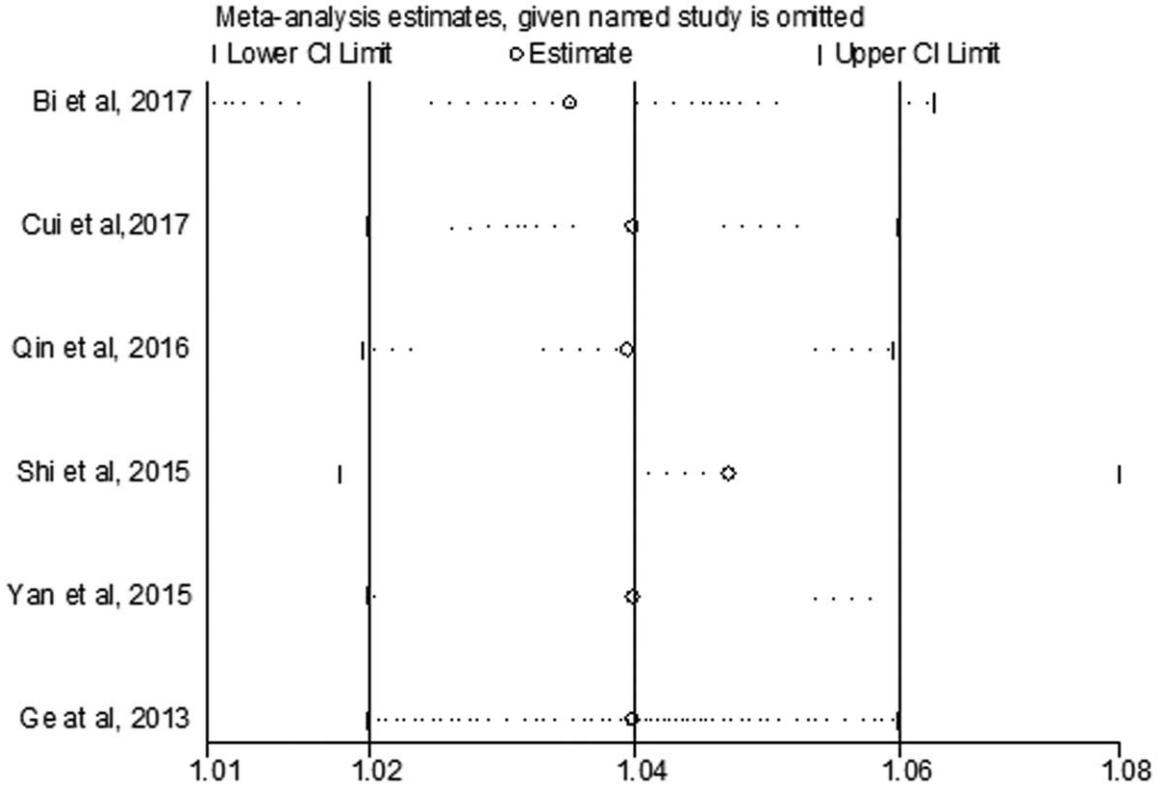

Figure 4. Sensitivity analysis for overall survival (OS).[24–29]

cancer.[30,31] In recent years, PCAT-1 has attracted great interest as a result of proof revealing that its abnormal expression in gastrointestinal cancer, such as HCC,[22,32,33] CRC,[21,23,29] GC,[24,25] ESCC,[26] and cholangiocarcinoma.[34] PCAT-1 was considered an oncogenic lncRNA in gastrointestinal tumors, and its overexpression was related to tumorigenesis and progression in various kinds of gastrointestinal cancers. PCAT-1 suppression significantly weakened cell proliferation, migration, and tumor invasion, whereas overexpressing PCAT-1 promoted these biologically aggressive features. Moreover, PCAT-1 could functions as competing endogenous RNA (ceRNA) to contribute tumor progression via several signaling

pathway, such as TP53-miR-215-PCAT-1-CRKL axis,[33] PCAT-1/miR-129-5p/HMGB1,[22] and PCAT1/miR-122/WNT1 axis.[34]

As far as we know, this is the first meta-analysis to comprehensively assess the association of PCAT-1 expression with prognosis and clinicopathologic features in gastrointestinal tumors. A total of 6 qualified studies, comprising 961 cases, were enrolled in this study. The pooled results showed that high expression of PCAT-1 was significantly associated with poor OS in gastrointestinal tumors, the subgroup analysis of PCAT-1 for OS further suggested that PCAT-1 could act as a predictive marker for OS in patients with gastrointestinal cancers. We also found that PCAT-1 was significantly correlated with some clinical features regarding tumor invasion, lymph node metastasis, and tumor stage. However, no obvious relationships were noticed between the high PCAT-1 expression and gender or differentiation or distant metastasis.

Some limitations of this study should be taken into account. First of all, the number of studies and the sample size were relatively small, with only 6 eligible studies with 961 cases were included. And then, all included studies were from China, researches from other countries were none or less, this may impact the broader applicability of our conclusions. Furthermore, there was no consensus on the cut-off value for distinguishing high and low PCAT-1 expression, for it was not easy to get a united threshold value in different studies. However, it is still essential to get a standardized value for PCAT-1 before it could be really applied in clinical practice. Additionally, significant heterogeneity was observed in the analysis of some clinicopathologic features. At last, most studies tended to report positive results rather than negative results, which might cause potential publication bias.

In conclusion, even with the limitations mentioned above, it can be preliminarily concluded that PCAT-1 might serve as a promising biomarker for improving prognosis estimation in gastrointestinal cancers. Notwithstanding, in the future, well-designed multicenter studies with large sample size are warranted to verify and strengthen the prognosis value of PCAT-1 in gastrointestinal cancers.

Author contributions

Conceptualization: Wanwei Liu.
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Formal analysis: Wanwei Liu.
Funding acquisition: Wanwei Liu.
Investigation: Wanwei Liu.
Methodology: Wanwei Liu.
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Software: Jiwei Xu.
Supervision: Wanwei Liu.
Validation: Wanwei Liu.
Visualization: Wanwei Liu.
Writing – original draft: Wanwei Liu.
Writing – review & editing: Wanwei Liu.

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