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REVIEW ARTICLE

Effect of OIP5-AS1 on Clinicopathological Characteristics and Prognosis of Cancer Patients: a Meta-Analysis

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Abstract

Recent research indicates that OPA-interacting protein 5 antisense RNA 1 (OIP5-AS1) played an essential role in a wide variety of carcinomas. Thus, we sought to evaluate the role of OIP5-AS1 in cancer patients to comprehend the OIP5-AS1 function in cancer better. The studies were collected using network databases, while the odds ratios (ORs) or hazard ratios (HRs) were extracted from included articles. The OIP5-AS1 role in cancer was further analyzed by pooled analysis. A total of 18 studies involved 1181 patients who were diagnosed with 13 different types of cancer. Combined analysis showed that OIP5-AS1 overexpressing patients had a poorer overall survival (OS) rate than those with lower expression (pooled HR = 1.541, 95% confidence interval (CI) = 1.351–1.757, $P < 0.001$). Interestingly, elevated OIP5-AS1 expression was found to be linked to cancer's unfavorable clinicopathological aspects, including larger tumor size (pooled OR = 0.274, 95% CI = 0.164–0.455, $P < 0.001$), advanced TNM stage (pooled OR = 0.301, 95% CI = 0.211–0.427, $P < 0.001$), and lymph node metastasis (pooled OR = 0.407, 95% CI = 0.234–0.706, $P = 0.001$). OIP5-AS1 was suggested to be a promising prognostic biomarker and a candidate target for cancer therapeutic strategies.

Keywords OIP5-AS1 · Biomarker · Prognosis · Cancer · Meta-analysis

Introduction

Cancer was considered the second leading cause of death, after cardiovascular complications, and has been regarded as a major complication affecting people's life expectancy worldwide. Furthermore, the variety and complexity of malignancies have progressively increased [1]. Fortunately, with the efforts of researchers, a large number of excellent and effective scientific research achievements have indeed improved the prognosis and quality of life of cancer patients. Personalized immunotherapies based on cancer biomarkers and innovative molecular-targeted therapeutics were expected to be widely used in clinical trials and showed great promise [2]. However, there were many contradicting outcomes of those cancer biomarkers in studies, obstructing the development of an effective predictor and delaying patients' acceptance of effective early treatment.

Long noncoding RNAs (lncRNAs) were found to play a significant role in several important physiological events in various carcinomas. LncRNAs could either suppress or promote tumor onset and progression by altering sequence and spatial structure, epigenetics modulation, expression level regulation, and binding protein interactions [3, 4]. It remained unclear, however, exactly how lncRNAs regulate cancer [5, 6]. To that end, the primary focus of cancer research at the time was to understand better how lncRNAs might be used in preclinical and clinical settings for disease diagnosis and therapy.

OIP5-AS1, called linc-OIP5, was a lncRNA located on the strand opposite the OIP5 gene and played a vital role in several carcinomas [7, 8]. According to mounting evidence, OIP5-AS1 played an oncogenic function in many malignancies. OIP5-AS1 elevated expression enhanced cellular proliferation by sponging miRNAs, including miR-448,

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miR-378a-3p, and miR-137 [9–11]. Moreover, OIP5-AS1 upregulation aided cell proliferation by elevating CDK4, CDK6, SMAD3, and integrin alpha 6 expressions (ITGA6) [10, 12, 13], while downregulation of OIP5-AS1 promoted the apoptotic process by the SOX2 and miR-143-3p/ROCK1 axis targeting, interacting with EZH2, or modulating glyoxalase 1 and NLRP6 expression [14–16]. It has been revealed that depletion of OIP5-AS1 caused cell cycle arrest at the G0/G1 phase and weakened colony formation ability [17]. Furthermore, OIP5-AS1 upregulation enhanced migration, invasion, and metastasis by regulating epithelial-mesenchymal transition (EMT) and angiogenesis, and studies also validated that OIP5-AS1 was involved in ferroptosis and autophagy, thus affecting the progression of cancer [18–20]. Similarly, clinical studies have indicated that an elevated OIP5-AS1 expression was closely associated with worse survival in cancer patients [9, 10, 18]. Conversely, OIP5-AS1 was considered a tumor suppressor gene, downregulated, or even lost in some cancers, showing antitumor activity [21]. Moreover, multiple reported studies have also shown that OIP5-AS1 could slow cellular proliferation, tumor growth, migration, and invasion by regulating miRNAs and tumor suppressor genes [21, 22]. However, some studies revealed no considerable correlation of OIP5-AS1 with the clinicopathological features and prognosis of cancer patients, and even an elevated expression of OIP5-AS1 in some specific cancers suggested a better prognosis [23]. Given these conflicting results, only one comprehensive study has preliminarily explored the clinical characteristics of OIP5-AS1 in malignancies [24]; however, these inconsistencies in OIP5-AS1 results in cancer emphasize the need for more precise and accurate research of OIP5-AS1 in cancer.

For an extensive understanding of OIP5-AS1 role in cancers, we conducted a meta-analysis of reported literature to evaluate the linkage between OIP5-AS1 and clinicopathological features and the prognosis of patients suffering from cancer.

Patients and Methods

Literature Search and Selection of the Study

Beginning in April 2022, we systematically searched for relevant research through May 2023 using Web of Science, PMC, PubMed, and other network databases. We used different combinations of the following terms to search for relevant articles in each database: ("long noncoding RNA" OR "lncRNA" AND "OIP5-AS1" OR "opa-interacting protein 5 antisense transcript 1") AND ("cancer" OR "tumor" OR "neoplasm" OR "carcinoma") AND ("prognosis" OR "Prognostic" OR "survival" OR "outcome"). In addition, two of our authors independently analyzed reference databases of pertinent literature to ensure precision. The entire protocol for this systematic review might be found online at https://doi.org/10.37766/inplasy2022.10.0118 and was registered with INPLASY (INPLASY2022100118). Noteworthy about this meta-analysis was that it strictly adhered to the PRISMA standards and focused only on studies published in English.

Inclusion and Exclusion Criteria

Articles were only considered for inclusion if they satisfied all of the following criteria: (1) patient studies in which a pathology-based diagnosis of cancer was made; (2) scientifically designed and conducted studies; (3) studies that examined OIP5-AS1 expression in cancer patients; (4) studies that grouped patients based on their OIP5-AS1 expression level; and (5) case–control studies that analyzed the prognostic value of OIP5-AS1 and collected enough data to compute HRs and 95% CIs for overall survival (OS). All publications focusing on cells and animals and bioinformatics analyses were also disregarded, as were abstracts, reviews, short reviews, and conference presentations.

Collection of Data and Quality Assessment

Two authors independently gathered research details (year, author, study region, sample source, sample size, cut-off value) and patient details (sex, age, tumor volume, cancer type, lymph node metastases (LNMs), TNM stage, histological grade) and discussed any discrepancies. The final chart was created after a third author checked it over and fixed any remaining problems. Consistent with the previously published prognostic meta-analyses, the included studies' quality was evaluated using the Newcastle–Ottawa Quality Assessment Scale (NOS). Research with a quality score of 6 or above was generally accepted as being of a good standard.

Statistical Evaluations

Statistical analyses were performed using STATA 14.0 (Stata Corporation). The association between OS and OIP5-AS1 expression was estimated by pooling HRs and their 95% CIs, while the correlation between clinicopathological features and OIP5-AS1 expression was assessed by pooling ORs and their 95% CIs. Patients with OIP5-AS1 overexpression had a higher likelihood of a poor outcome or cancer progression (HR and corresponding 95% CI > 1 and OR and corresponding 95% CI < 1). We retrieved the fatalities and sample size in each group from studies that did not include HRs and CIs and used those numbers to determine an HR. Alternatively, information was retrieved from Kaplan–Meier curves using Engauge Digitizer 11.1. The HR was then estimated using the procedure outlined [25]. In addition, the chi-square test and the I^2 statistic were used to analyze the heterogeneity,

and the results informed the adjustments made to the statistical model. To rephrase, we used the random-effects model when the significance level was below 0.10% and/or the heterogeneity index, I^2, was over 50%, and we resorted to the fixed-effects model otherwise. A sensitivity analysis was performed by systematically removing studies to determine the degree to which the main research results changed. To identify publication bias, Begg's and Egger's tests [26] were employed. The test statistic had to have a P-value < 0.05 (two-sided) to be statistically significant.

Results

Features of Current Research and Literature Study

A total of 323 articles were obtained during the initial screening process. After a preliminary review, 155 irrelevant articles were excluded. The abstracts and general content of the remaining articles were further evaluated. Moreover, a total of 34 articles were selected as candidates, of which 16 were excluded for reasons of examining the expression of OIP5 [5], bioinformatic and TCGA studies [$n = 5$], the lack of the survival data [$n = 2$], and cell or animal studies [$n = 4$]). Finally, 18 articles evaluating the prognostic value of OIP5-AS1 in patients (suffering from cancer) were included, and the complete selection procedure of the study was plotted, as shown in Fig. 1. The included studies, which were published between 2018 and 2021, involved 1181 patients with 13 types of cancer, including bladder cancer [27], breast cancer [28], cervical cancer [10], colon cancer [29], gastric cancer [30, 31], hepatic cancer [18, 32], lung cancer [9, 33], malignant melanoma [34], multiple myeloma [23], osteosarcoma [12, 35], ovarian cancer [36], pancreatic cancer [37, 38], and thyroid cancer [39]. These studies were conducted in China, using qRT-PCR analysis for the detection of the OIP5-AS1 expression level. Only one study provided HR by univariate/multivariate analysis, whereas the other studies figured out HR via univariate analysis. Of these, 14 studies selected the OIP5-AS1 expression median value as their cut-off value for grouping patients, while the remaining studies chose the mean value. Furthermore, the

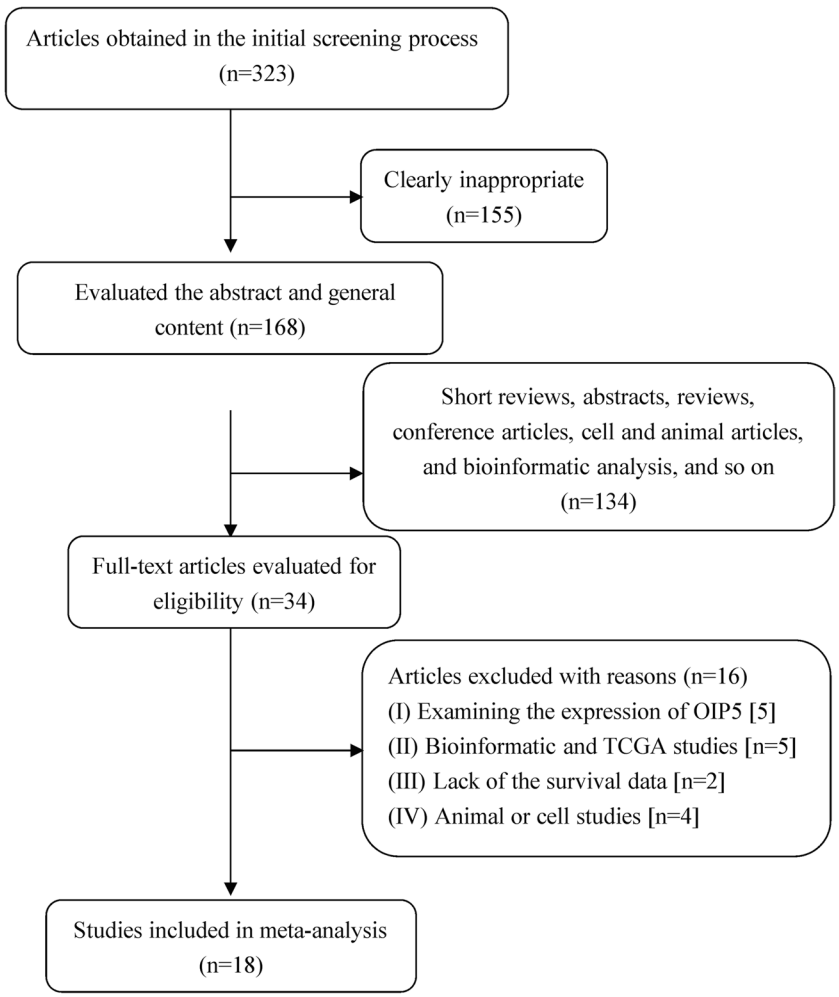

Fig. 1 Flow chart of retrieving relevant articles from the literature

OIP5-AS1 expression in cancer tissues was considerably elevated compared to the adjacent normal tissues in all studies except one. The details are shown in Table 1. Based on NOS evaluations, the included articles were of high quality.

Prognostic Value of OIP5-AS1 for Cancers

As illustrated in Table 2, we indicated that OIP5-AS1 was an effective prognostic indicator in cancer patients. Moreover, patients with an elevated OIP5-AS1 expression had a poorer OS than cancer patients with a low OIP5-AS1 expression (pooled HR = 1.541, 95% CI = 1.351–1.757, $P < 0.001$, Fig. 2). An elevated expression of OIP5-AS1 was related to a poorer outcome in subgroup analyses of individuals with cancer of the digestive system (pooled HR = 1.519, 95% CI = 1.334–1.729, $P < 0.001$, Fig. 3). Furthermore, this prognostic value was significant as well in hepatobiliary pancreatic cancer (pooled HR = 1.413, 95% CI = 1.222–1.633, $P < 0.001$) and cancer of the digestive tract (pooled HR = 2.022, 95% CI = 1.516–2.696, $P < 0.001$). Results also demonstrated that elevated OIP5-AS1 expression was linked with bad outcomes in other cancers

Table 1 Main characteristics of studies included in meta-analysis

Author(year)	Study region	Sample size	Cancer type	Detected Sample	Cutoff values (High/Low)	OS, HR estimation	Analysis method	Detection Method	Quality score
Wang Y(2018)	China	112	Bladder cancer	Tissue	Median	2.13(1.45–3.11)	Univariate analysis	qRT-PCR	6
Dai J(2018)	China	48	Osteosarcoma	Tissue	Median	1.79(1.05–3.03)	Univariate analysis	qRT-PCR	7
Zhang Z(2018)	China	80	Hepatic cancer	Ttissue	Median	1.36(1.03–1.83)	Univariate analysis	qRT-PCR	8
Deng J(2018)	China	64	Lung cancer	Tissue	Mean	4.95(1.62–15.12)	Univariate analysis	qRT-PCR	7
Wang M(2019)	China	80	Lung cancer	Tissue	Median	1.61(1.45–1.96)	Univariate analysis	qRT-PCR	7
Kun-Peng Z(2019)	China	80	Osteosarcoma	Tissue	Median	2.78(1.28–6.04)	Univariate analysis	qRT-PCR	8
Yang J(2019)	China	57	Cervical cancer	Tissue	Median	1.24(1.06–1.45)	Univariate analysis	qRT-PCR	6
Luan W(2019)	China	30	Malignant melanoma	Tissue	Median	3.14(1.17–8.43)	Univariate/multivariate analysis	qRT-PCR	7
Xie R(2020)	China	60	Gastric cancer	Tissue	Median	1.90(1.09–3.32)	Univariate analysis	qRT-PCR	6
Ma YS(2020)	China	54	Hepatic cancer	Ttissue	Median	1.26(1.01–1.57)	Univariate analysis	qRT-PCR	7
Wang Y(2020)	China	38	Multiple myeloma	Bone marrow	Median	0.39(0.19–0.79)	Univariate analysis	qRT-PCR	7
Wu L(2020)	China	110	Pancreatic cancer	Tissue	Median	1.61(1.18–2.21)	Univariate analysis	qRT-PCR	7
Li Q(2020)	China	60	Thyroid Cancer	Tissue	Median	1.30(1.02–1.66)	Univariate analysis	qRT-PCR	7
Tao Y(2020)	China	78	Gastric cancer	Tissue	Mean	2.19(1.39–3.45)	Univariate analysis	qRT-PCR	6
Liu QY(2020)	China	52	Ovarian cancer	Tissue	Mean	1.63(1.14–2.32)	Univariate analysis	qRT-PCR	8
Wang Y(2021)	China	62	Colon cancer	Tissue	Mean	1.93(1.17–3.18)	Univariate analysis	qRT-PCR	8
Li A(2021)	China	86	Pancreatic cancer	Tissue	Median	1.92(1.22–3.03)	Univariate analysis	qRT-PCR	8
Wu Z(2021)	China	30	Breast Cancer	Tissue	Median	1.22(1.03–1.44)	Univariate analysis	qRT-PCR	7

OS overall survival; *HR* hazard ratio. Sample size: the number of patients included in the study. Cutoff values: Grouping patients according to OIP5-AS1 expression level

Table 2 Meta-analysis of OIP5-AS1 expression and prognosis in cancer patients

Categories	Studies (patients)	HR (95% CI)	I^2 (%)	P_h	Z	P
OS	18 (1181)	1.541 (1.351–1.757)	63.9	< 0.001	6.46	< 0.001
Cancer type (digestive system cancer)	7 (530)	1.519 (1.334–1.729)	29.2	0.206	6.33	< 0.001
Cancer type (hepatobiliary pancreatic cancer)	4 (330)	1.413 (1.222–1.633)	14.7	0.319	4.67	< 0.001
Cancer type (digestive tract cancer)	3 (200)	2.022 (1.516–2.696)	0.0	0.905	4.79	< 0.001
Cancer type (other cancers)	11 (651)	1.502 (1.243–1.814)	73.4	< 0.001	4.22	< 0.001
Size ≤ 60	9 (429)	1.318 (1.121–1.550)	59.2	0.012	3.34	0.001
Size > 60	9 (752)	1.698 (1.528–1.888)	27.6	0.199	9.81	< 0.001
Cutoff value (median)	14 (925)	1.458 (1.271–1.673)	64.5	< 0.001	5.38	< 0.001
Cutoff value (mean)	4 (256)	1.933 (1.523–2.454)	23.0	0.273	5.41	< 0.001

OS, overall survival; *HR*, hazard ratio; *CI*, confidence interval; P_h, *P*-value for heterogeneity based on *Q* test; *P*, *P*-value for statistical significance based on *Z* test. Size: the number of patients included in the study, Cutoff value: grouping patients according to OIP5-AS1 expression level. Other cancers: bladder cancer, breast cancer, cervical cancer, lung cancer, malignant melanoma, multiple myeloma, osteosarcoma, ovarian cancer, and thyroid cancer

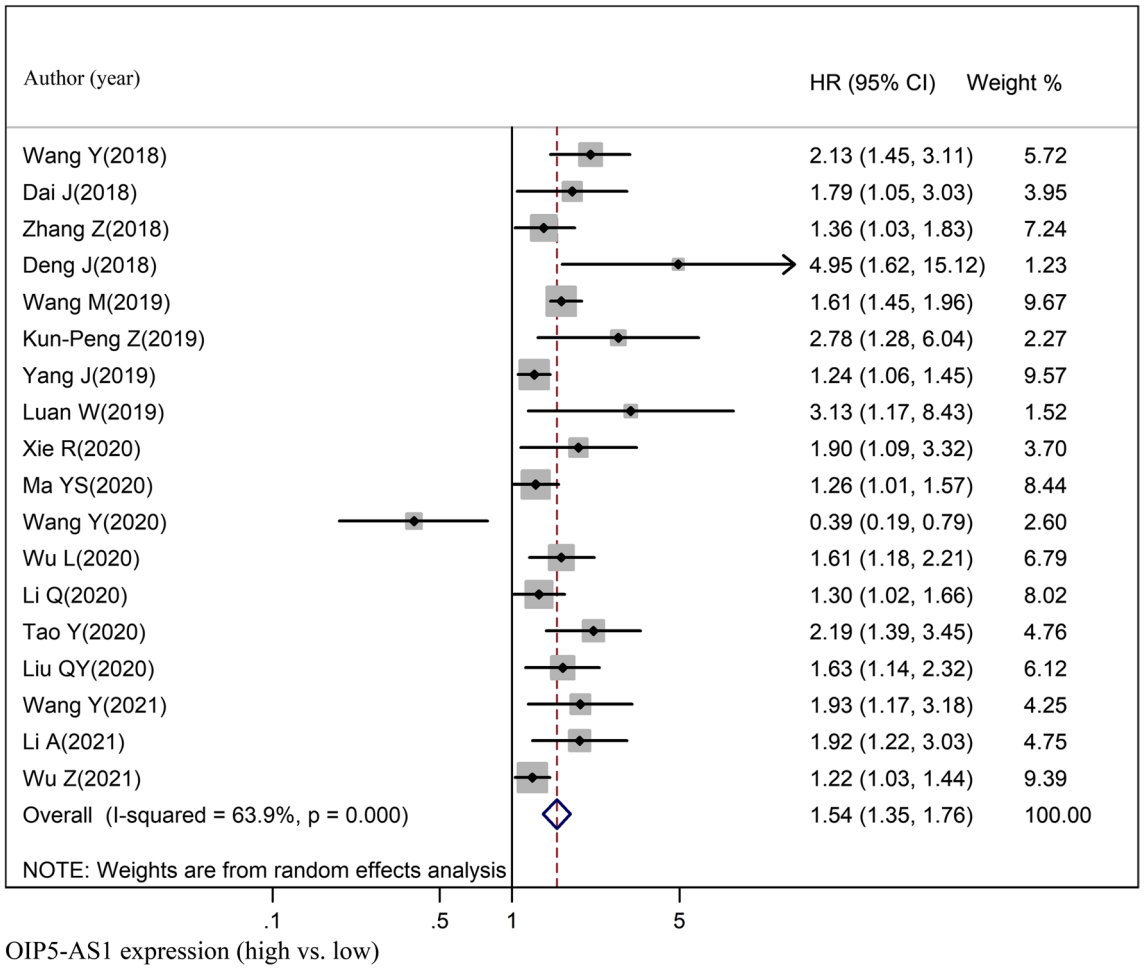

Fig. 2 Forest plot for overall survival in patients with cancers by OIP5-AS1 expression

included in this meta-analysis (pooled HR = 1.502, 95% CI = 1.243–1.814, *P* = 0.001). Moreover, it has been revealed that the prognostic value of OIP5-AS1 did not change with differences in sample size (≤ 60, pooled HR = 1.318, 95% CI = 1.121–1.550, *P* = 0.001; > 60, pooled HR = 1.698, 95% CI = 1.528–1.888, *P* < 0.001, respectively) and cut-off

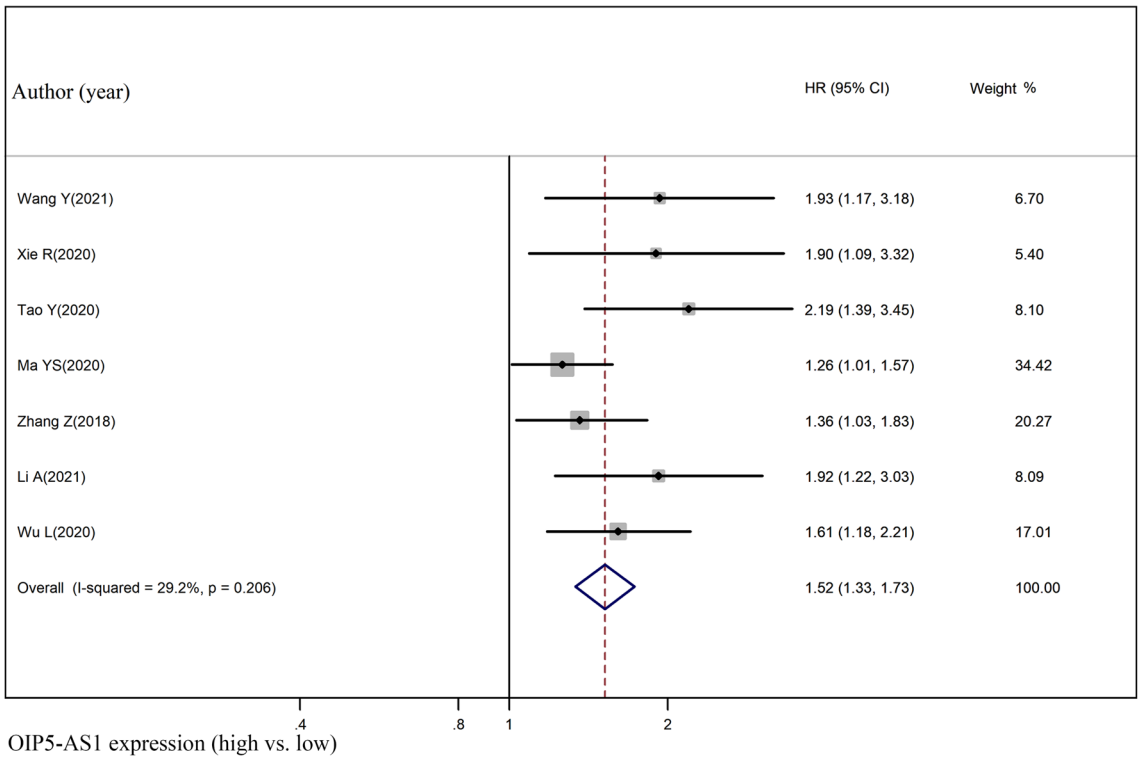

Fig. 3 Forest plot for overall survival in patients with digestive system cancer by OIP5-AS1 expression

value (median, pooled HR = 1.458, 95% CI = 1.271–1.673, $P < 0.001$; Mean, pooled HR = 1.933, 95% CI = 1.523–2.454, $P < 0.001$, respectively).

OIP5-AS1 and Clinicopathological Features in Cancers

Data on clinicopathological characteristics were obtained to determine the implications of OIP5-AS1 in cancer. As described in Table 3, an elevated OIP5-AS1 expression was linked to larger tumor size (pooled OR = 0.274, 95% CI = 0.164–0.455, $P < 0.001$), advanced TNM stage (pooled OR = 0.301, 95% CI = 0.211–0.427, $P < 0.001$), and LNMs (pooled OR = 0.407, 95% CI = 0.234–0.706, $P = 0.001$). The remarkable data demonstrated that overexpression of OIP5-AS1 contributed to cancer progression. However, no significant correlation was exhibited between OIP5-AS1 expression and gender (pooled OR = 1.141, 95% CI = 0.734–1.774, $P = 0.557$), age (pooled OR = 0.993, 95% CI = 0.699–1.412, $P = 0.970$), and histologic grade (pooled OR = 0.724, 95% CI = 0.391–1.339, $P = 0.303$) in cancer patients.

Table 3 Meta-analysis of OIP5-AS1 expression classified by clinicopathological features

Study covariates	Studies (patients)	OR (95% CI)	I^2 (%)	P_h	Z	P
Gender (man/woman)	4(328)	1.141 (0.734–1.774)	0.0	0.732	0.59	0.557
Age (< 60/ ≥ 60)	8(530)	0.993 (0.699–1.412)	0.0	0.712	0.04	0.970
Tumor size (< 5/ ≥ 5 cm)	5(318)	0.274 (0.164–0.455)	0.0	0.587	4.99	< 0.001
TNM stage (I + II/III + IV)	9(582)	0.301 (0.211–0.427)	0.0	0.502	6.70	< 0.001
LN metastasis (absence/presence)	4(247)	0.407 (0.234–0.706)	17.3	0.305	3.20	0.001
Histologic grade (well + moderately differentiated/poorly differentiated)	2(167)	0.724 (0.391–1.339)	0.0	0.429	1.03	0.303

LN, lymph node; *OR*, odds ratio; *CI*, confidence intervals; P_h, *P*-value for heterogeneity based on *Q* test; *P*, *P*-value for statistical significance based on *Z* test

Meta-Analysis on a Cumulative Basis and Sensitivity Analysis

A cumulative meta-analysis and sensitivity analysis was done to determine the consistency of OIP5-AS1's predictive value in patients suffering from cancer. As shown in Fig. 4, the combined HR and corresponding 95% CI tended to stabilize as the year approached and the number of studies increased. Sensitivity analysis plots (Fig. 5) also showed that all point estimates were distributed within the 95% CI range of the combined analysis that assessed the prognostic importance of OIP5-AS1 for all included patients. Taken together, the results generated by this meta-analysis showed consistency and reliability.

Heterogeneity and Publication Bias

The chi-square test and I^2 statistic evaluated heterogeneity. Table 2 reveals that the combined analysis exhibited evident heterogeneity ($I^2 = 63.9\%$, $P_h < 0.001$) for evaluating the impact of OIP5-AS1 expression in patients suffering from cancer. Notably, in the subsequent subgroup analysis, we observed that heterogeneity was reduced in some subgroups; however, the source of heterogeneity remained unexplained. Therefore, a meta-regression analysis was performed. According to the obtained results, neither the type of cancer ($P = 0.720$), publication year ($P = 0.715$), sample size ($P = 0.075$), and cut-off value ($P = 0.131$) nor the analysis method ($P = 0.237$) was the main source of heterogeneity. Additionally, Egger's test ($P = 0.053$, Fig. 6) and Begg's test ($P = 0.064$) pointed out no explicit publication bias in the underlined meta-analysis.

Discussion

The emergence of cancer biomarkers has provided a significant foundation for the establishment of monitoring, treatment, and prediction models and provided an effective basis and guidance for clinicians in cancer therapy [40]. In this meta-analysis, we pointed out that OIP5-AS1 independently predicts the survival outcomes, and an elevated OIP5-AS1 expression could result in poor survival in cancer patients and was expected to become an effective clinical marker.

Recently reported data revealed that OIP5-AS1 contributed to several physiological and pathological processes in which cancer was the subject of great interest. Furthermore, OIP5-AS1 was overexpressed in various carcinomas, and the processes behind this phenomenon have been partially explained [10, 27, 28, 41]. Interestingly, this meta-analysis,

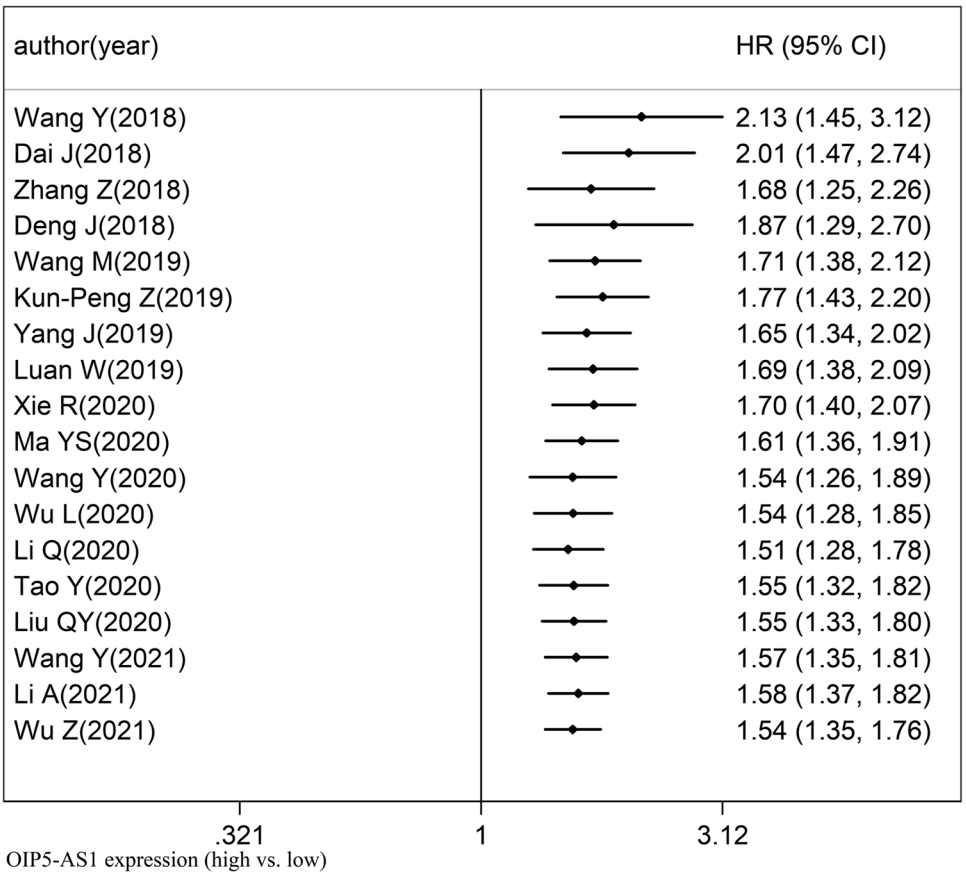

Fig. 4 Cumulative meta-analysis of OIP5-AS1expression and overall survival in cancers

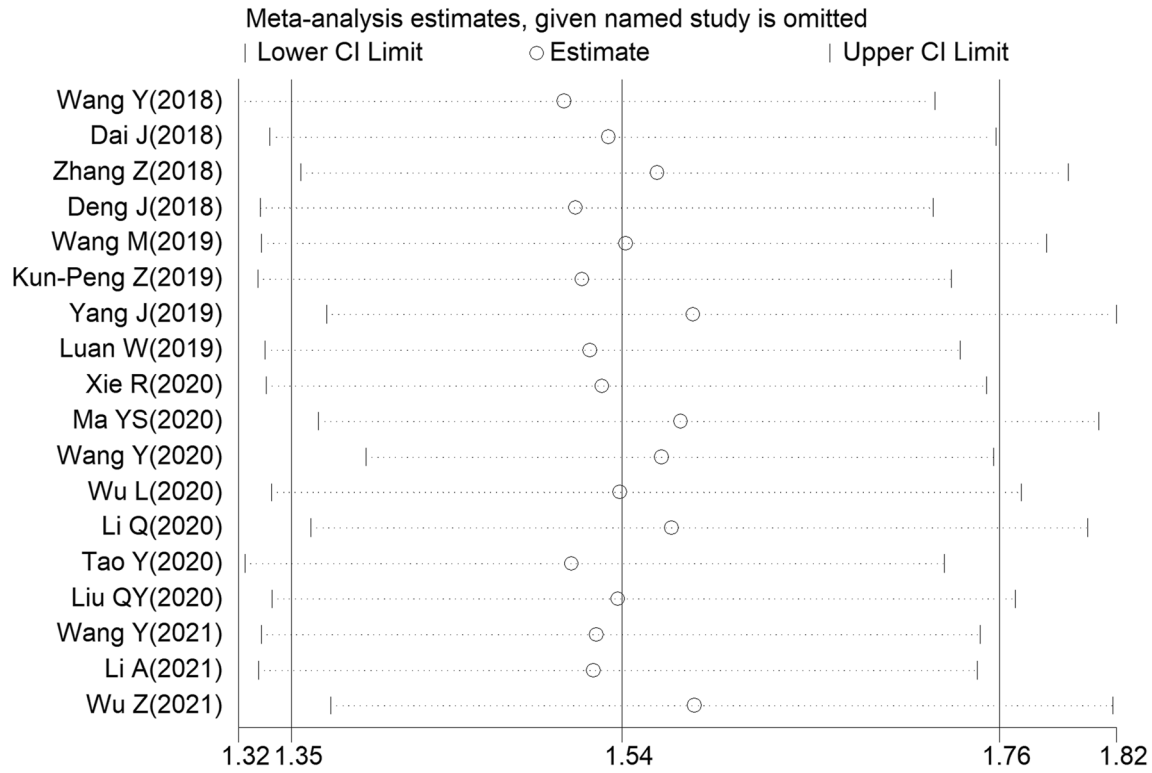

Fig. 5 Sensitive analysis of overall survival for cancer patients

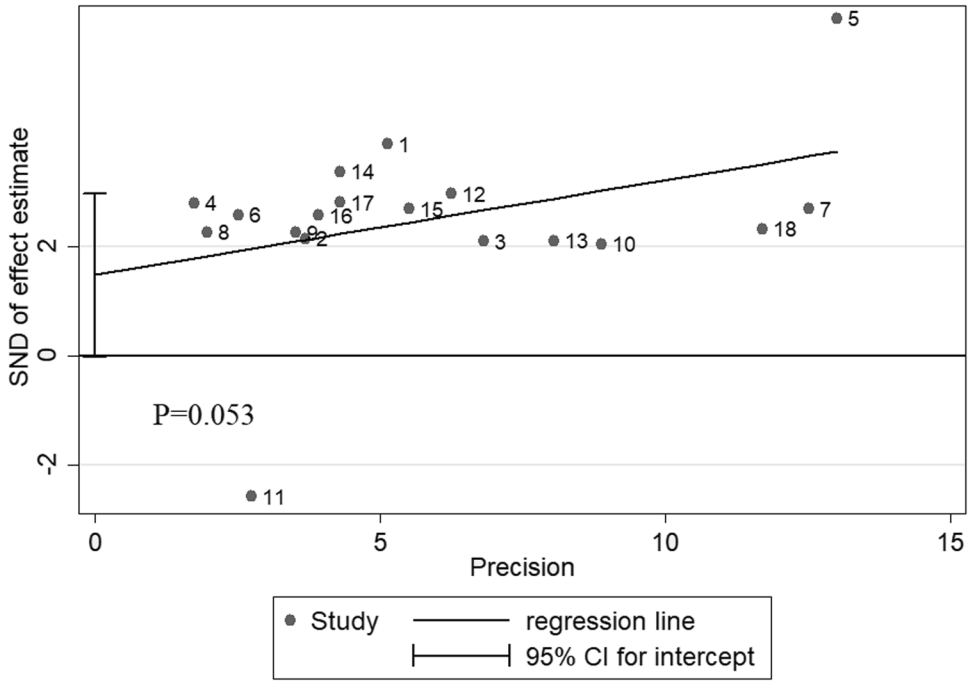

Fig. 6 Assessment of publication bias of included studies in Egger's test

which explored 18 studies in 13 types of cancer involving 1181 patients, showed that OIP5-AS1 overexpression led to adverse outcomes in cancer patients. Similarly, Sun et al. revealed that OIP5-AS1 could modulate G0/G1 phase and facilitate cell cycle progression [17]. OIP5-AS1 knockdown could alleviate the effect of EMT on promoting invasion and metastasis in cancer cells [36]. OIP5-AS1 silencing improved sensitivity to chemotherapeutic and targeted drugs

in cancers [42, 43]. Moreover, OIP5-AS1 was involved in neoplastic transformation by regulating HMGB3 expression via sponging miRNAs [44]. In addition, some signaling cascades related to neoplastic transformation, such as PI3K/AKT/mTOR and VEGF, have also been shown to be regulated by OIP5-AS1 [42, 45]. In view of those, the conclusion in this meta-analysis that OIP5-AS1 was a poor cancer prognostic factor based on the currently available clinical studies remained credible. However, OIP5-AS1 exhibited a tumor-suppressive role in a few carcinomas currently being examined as it influenced a diversity of downstream targets [22, 46]. However, this still needed to be proven in subsequent studies.

In particular, by meta-analysis, we were the first to demonstrate the prognostic importance of OIP5-AS1 in the digestive system, hepatobiliary pancreatic, and digestive tract cancers. This could be because gastrointestinal cancers share similar biological properties, or it could be that OIP5-AS1 played a part in these traits by regulating the signaling axis and pathways essential for colony formation, cell viability, cell cycle process, and cell proliferation in gastrointestinal cancers. It has been shown that OIP5-AS1 regulates and promotes the incidence and development of digestive system cancer by targeting tumorigenic pathways such miR-422a/ANO1 axis [30], hsa-miR-26a-3p/EPHA2 axis [32], and miR-429/FOXD1 axis [38]. Given these findings, OIP5-AS1 showed promise as a universally applicable prognostic marker for patients with cancer.

In addition, the OIP5-AS1 prognostic importance in cancers was the same no matter how the sample size or cut-off value changed. Due to the complexity and diversity of the included articles and cancer types, significant heterogeneity inevitably appeared in the combined analysis. Although heterogeneity was reduced in subgroup analysis, meta-regression analysis verified that cancer type, publication year, sample size, cut-off value, and analysis method were not the main sources of heterogeneity. As a result, we were unable to determine the origin of heterogeneity. A random effect procedure was established to limit the effect of significant heterogeneity on the obtained data. The cumulative meta-analysis and sensitivity analysis showed that the predictive value of OIP5-AS1 in cancer was consistent. Otherwise, in a pooled correlation analysis between OIP5-AS1 and clinicopathological features, we found that an elevated OIP5-AS1 expression was strongly linked to larger tumor size. Xie et al. also pointed out that OIP5-AS1 could stimulate tumor proliferation by the miR-422a/ANO1 axis targeting [30]. Similarly, statistical evidence from this meta-study revealed that OIP5-AS1 had a considerable link with the advanced TNM stage and lymph node metastasis. OIP5-AS1 has been corroborated in ovarian carcinoma to accelerate cancer cells' proliferative and metastatic potential via upregulating ITGA6 [47]. Altogether, OIP5-AS1 led to bad outcomes for clinical patients because it helped cancer get worse, suggesting that OIP5-AS1 had the potential as a prognostic cancer marker. This meta-analysis pooled limited research results and timely and effectively analyzed the oOIP5-AS1 role in cancer, providing more evidence for future research and promoting further development of research in a scientific direction.

The present study had some limitations. First, the number of included articles and sample sizes in several studies were quite small. Second, the study was conducted in a relatively limited geographic area, making it difficult to compile comprehensive statistics. Moreover, despite best efforts, there was still a risk of statistical error because of the lack of uniformity in study methodology among included research. Thirdly, although numerous cancer types were included, the insufficient research on each cancer made it difficult to undertake a precise study of the involvement of OIP5-AS1 in a particular cancer. Fourth, the upstream and downstream molecules of OIP5-AS1 could not be analyzed together in the current study. It was worth mentioning that there was evidence that the level of OIP5-AS1 expression was linked with the oncogenic transcript OIP5 [48]; however, effective data could not be extracted from the articles involved in this meta-analysis. To clarify and evaluate the particular mechanism of OIP5-AS1 in cancer, additional thorough studies should be carried out in more regions and populations.

Conclusion

In this study, OIP5-AS1 was identified as a potential cancer prognostic indicator, and it was discovered that high OIP5-AS1 expression was linked with a poor overall survival due to a combination of large tumor size, lymph node metastases, and an advanced TNM stage. Considerable evidence showed that OIP5-AS1 might soon make important contributions to cancer treatment and might even pave the way for new clinical studies.

Author contribution Study conception and design: CM and JL. Material preparation, data retrieval, and analysis: PY, BL, and CM. Interpretation of the results: BL, CM, and JL. Paper writing: CM and JL. All authors read and approved the final manuscript.

Data Availability All data are available from the corresponding author.

Declarations

Ethics approval and consent to participate Since the meta-analysis was an analysis of previously published studies, therefore, no ethical approval or patient consent was required.

Consent for publication Not applicable.

Competing interests The authors declare no competing interests.

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