

SYSTEMATIC REVIEW

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The association between long noncoding RNA ABHD11-AS1 and malignancy prognosis: a meta-analysis

Guangyao Lin¹, Tao Ye² and Jing Wang^{3*}

Abstract

Background Accumulating evidence has highlighted that lncRNA ABHD11-AS1 plays an essential role in tumorigenesis and is expected to become a new predictive biomarker and ideal target for cancer therapy, whereas some of their findings are conflicting due to the relatively small sample size of individual studies. Thus, this meta-analysis aimed to quantitatively ascertain the association of ABHD11-AS1 with diverse human malignancies.

Methods Eight databases were comprehensively screened for relevant articles on January 1, 2024. The significance of ABHD11-AS1 in malignancies was determined by odds ratios (ORs) or hazard ratios (HRs) with corresponding 95% confidence interval (CI). Subgroup analyses and sensitivity analyses were applied to verify the reliability and robustness of the pooled results. Simultaneously, the GEPIA2021 and UCSC Xena databases were applied to further strengthen the results.

Results Fourteen clinical studies comprising eight kinds of malignancies and 1215 malignancy cases were enrolled into this meta-analysis. The pooled results showed that increased ABHD11-AS1 expression was remarkably associated with lymph node metastasis (OR = 2.73, 95%CI [1.97, 3.77], $I^2 = 0%$, $p < 0.00001$), advanced tumor stage (OR = 3.14, 95%CI [2.34, 4.21], $I^2 = 39%$, $p < 0.00001$), and unfavorable overall survival (OS) (HR = 1.81, 95%CI [1.58, 2.06], $I^2 = 0%$, $p < 0.00001$). Subgroup analyses and sensitivity analyses indicated that the pooled results were reliable and robust. Additionally, ABHD11-AS1 was significantly increased in eight kinds of malignancies according to the validation of the GEPIA2021 database. Meanwhile, the UCSC Xena databases further revealed that elevated ABHD11-AS1 expression was significantly associated with poor prognosis as assessed by progression free interval (PFI), disease free interval (DFI), disease specific survival (DSS), and OS.

Conclusions Current evidence supports the association of elevated ABHD11-AS1 expression with poor prognosis. Thereby, ABHD11-AS1 may be considered as a promising biomarker to screen cancer and predict malignancy prognosis. Also, there is a necessity for larger-scale multicenter studies with uniform study protocols from different countries to further validate the conclusions.

Keywords *LncRNA* ABHD11-AS1, Malignancy, Prognosis, Meta-analysis, Review

*Correspondence:

Jing Wang

lerel-st@hotmail.com

Full list of author information is available at the end of the article



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Introduction

Cancer has been a major health problem worldwide, and almost 10.0 million cancer deaths occurred in 2022 globally [1]. In the United States, there will be 611,720 cancer deaths in 2024, corresponding to 1675 deaths per day [2]. Additionally, in 2020, an estimated number of 3 million cancer-related deaths in China, attributing to 30.2% of all cancer deaths in the world [3]. Remarkably, a growing number of countries are moving with exigency to implement policies on the management of malignancies since the cancer-related burden continues to increase rapidly. For example, China, Australia, and the United States have introduced various public health programs such as Upper Gastrointestinal Cancer Screening Programs, and the Australian Cancer Plan, with a focus on early detection and high-impact prevention of cancer [4–6]. Whereas the cancer incidence and mortality rates have not declined significantly for decades, and there are scarce specific and sensitive biomarkers to detect early-stage cancers [7]. According to the GLOBOCAN 2022 estimates, there will be 35 million new cancer cases worldwide by 2050 [1]. Thus, it is imperative to probe novel biomarkers to screen carcinoma and predict the prognosis of cancer cases.

Long non-coding RNAs (lncRNAs) are more than 200 nucleotides in length and lack protein-coding potential [8]. Generally, diverse biological processes like cancer progression and tumorigenesis were closely associated with the regulation of *lncRNAs*, which might be considered as novel molecular markers for malignancy detection and treatment [9, 10]. For example, ABHD11 antisense RNA 1 (ABHD11-AS1), a recently discovered *lncRNA* which is located on the chromosome 7q11.23 [11]. Emerging evidence has revealed that overexpression of ABHD11-AS1 could promote its oncogenic potential and facilitate the progression of diverse kinds of human malignancies, including digestive system tumors, reproductive system tumors, respiratory system tumors, and other malignancies [11–13]. Clinically, elevated ABHD11-AS1 expression is associated with adverse overall survival (OS), lymph node metastasis (LNM), and advanced tumor stage in papillary thyroid cancer [14–16]. Simultaneously, a recent study also illustrated that ABHD11-AS1 expression is connected to progression free survival (PFS) in colorectal cancer [17]. Moreover, accumulating *in vivo* and *in vitro* studies proved that ABHD11-AS1 contributes to tumor cell growth, migration, and invasion, along with regulates lymphangiogenesis and inhibits tumor cell apoptosis through complex molecular mechanisms [18–20]. Collectively, ABHD11-AS1 strongly captures the attention of researchers and may be explored as a promising predictor of malignancy prognosis.

However, despite the large number of clinical studies investigating the relationship between ABHD11-AS1 expression and malignancy prognosis, several studies have drawn controversial conclusions. For instance, two records [14, 15] suggested that high ABHD11-AS1 expression indicated advanced tumor stage ($p < 0.05$) in papillary thyroid cancer, while another two studies [16–21] held the opposite opinion. Furthermore, Xin, et al. [22], detected the expression of ABHD11-AS1 in gastric cancer by polymerase chain reaction (PCR) and proved that increased ABHD11-AS1 expression was related to larger tumor size ($p < 0.05$), but this was not confirmed by Qiao's study [23]. Owing to the relatively small clinical sample sizes of individual studies, the prognostic significance of ABHD11-AS1 in various cancers may be insufficiently evaluated. Therefore, we performed this meta-analysis to quantitatively assess the association of ABHD11-AS1 with diverse human malignancies.

Methods

This study followed the preferred reporting program of the systematic review and meta-analysis (PRISMA) guidelines [24].

Search strategy

We thoroughly searched eight databases [24, 25], including EBSCO (<https://web.p.ebscohost.com/>), SinoMed (<https://www.sinomed.ac.cn/>), Web of Science (<https://webofscience.clarivate.cn/>), Scopus (<https://www.scopus.com/>), Wanfang (<https://www.wanfangdata.com.cn/>), PubMed (<https://pubmed.ncbi.nlm.nih.gov/>), Cochrane Library (<https://www.cochranelibrary.com/>), and China National Knowledge Infrastructure (CNKI) (<https://kns.cnki.net/>) for relevant studies published from inception to January 1, 2024. The search terms were as follows: ABHD11 antisense RNA 1, *long non-coding RNA* ABHD11-AS1, and ABHD11-AS1. Additionally, a complementary search was also conducted by carefully scrutinizing the references in the retrieved records. The titles and abstracts of all relevant publications were initially checked by two independent researchers (G.Y.L and T.Y) for potential eligibility documents. Subsequently, studies were included after assessing the full texts independently. Any discrepancies were resolved through discussion with the third researcher (J.W) if necessary.

Inclusion and exclusion criteria

The inclusion criteria were as follows: (1) participants were divided into high ABHD11-AS1 group and low ABHD11-AS1 group regardless of cancer types; (2) malignancies were solid tumors; (3) the data of ABHD11-AS1 derived from tissue specimens; (4) studies provided the information on OS or clinicopathologic parameters

like age of patients, tumor stage, and so forth; (5) patients had not been treated with any anticancer therapy before surgery.

The exclusion criteria were: (1) data obtained from public databases; (2) records failed to provide sufficient clinicopathological information related to this meta-analysis; (3) duplicate publications, retracted articles, cellular experiments, reviews, and letters.

Data extraction and quality assessment

Data were independently extracted from eligible documents by two investigators (G.Y.L. and T.Y.) utilizing standardized forms. The following data were extracted: first author, publication year, malignancy types, the number of clinical samples, detection method for ABHD11-AS1, clinicopathologic parameters, the related data for OS, and follow-up time. The Engauge Digitizer 4.1 software was exploited to acquire the corresponding hazard ratio (HR) and 95% confidence interval (CI) data of OS via Kaplan–Meier curves indirectly [26]. Discrepancies were addressed through consensus with corresponding author. Besides, the quality of each included article was appraised utilizing the Newcastle–Ottawa scale (NOS), whose scores ranged from 0 to 9. Studies that scored ≥ 6 were considered as having high quality [27].

Validation of ABHD11-AS1 expression in various malignancies

The expression of ABHD11-AS1 in tumor tissues and normal tissues was further assessed by Gene Expression Profiling Interactive Analysis 2021 (GEPIA2021) (<http://gepia.cancer-pku.cn/>) [28], which consisted of GTEx and TCGA data, and it also had been extensively employed to strengthen meta-analysis results [29–31]. $P < 0.01$ indicated statistically significant. Meanwhile, prognostic indicators such as progression free interval (PFI), disease specific survival (DSS), and disease free interval (DFI) from UCSC Xena (<https://xena.ucsc.edu/>) database were analyzed to comprehensively assess the prognostic value of ABHD11-AS1. $P \leq 0.05$ was considered statistically significant.

Statistical analysis

Review Manager 5.3 [32] and R 4.3.0 software were used for this meta-analysis and prognostic indicators analysis, respectively. Odds ratios (ORs) with 95% CIs were adopted to assess the association between ABHD11-AS1 and clinicopathologic parameters (i.e., tumor stage). Meanwhile, HRs and 95% CIs were applied to clarify the association between ABHD11-AS1 and OS. Forest plots were exploited to display the results of this meta-analysis. The rectangle in the forest plots represented the study weights; the larger the sample size, the larger the

rectangle. The diamond in the forest plots represented the pooled result. When the diamond crossed the vertical line where $OR = 1$ or $HR = 1$, it meant that the result was not statistically significant. Besides, statistical heterogeneity across studies was quantified using the I^2 statistic. When the I^2 statistic exceeded 50%, substantial heterogeneity was considered to be present and random-effects model was performed. Otherwise, a more appropriate fixed-effects model was utilized. Subgroup analyses covering cancer types and sample sizes were performed to further examine the reliability of the results. To ascertain the robustness of the pooled results, sensitivity analysis were carried out by excluding individual studies. $P \leq 0.05$ showed statistically significant.

Results

Included articles

Figure 1 shows the PRISMA study selection flowchart. First, 232 references from our literature search in eight databases were obtained. After removing 154 duplicate publications, 78 records were identified. 60 studies were further excluded since they matched our exclusion criteria. Thereafter, of the remaining 20 records, six studies that failed to provide sufficient clinical data or pooled their data from blood specimens were also removed. Finally, 14 studies investigating the association of ABHD11-AS1 with malignancy prognosis were included in this meta-analysis.

Study characteristics

Table 1 displays the main characteristics of included records according to the order of their publication year. 14 studies involving eight kinds of malignancies with 1215 cancer cases were included. All clinical studies were conducted in different hospitals in China, and the sample sizes in each record ranged from 20 to 248. Besides, four of the 14 studies provided the information on both clinicopathologic parameters and OS. Among these included studies, the follow up duration varied from 40 to 110 months. All the data on HR for OS were extracted from Kaplan–Meier curves. Regarding the study quality, 14 included records were assessed to be of high quality as they were rated ≥ 6 .

Association between ABHD11-AS1 expression and clinicopathologic parameters

Ten studies enrolling 725 patients with cancer explored the association between ABHD11-AS1 expression with LNM. The fixed model was adopted since there was no heterogeneity existing among studies, and the pooled evidence suggested that elevated ABHD11-AS1 expression was significantly associated with early LNM ($OR = 2.73$, 95%CI [1.97, 3.77], $I^2 = 0\%$, $p < 0.00001$)

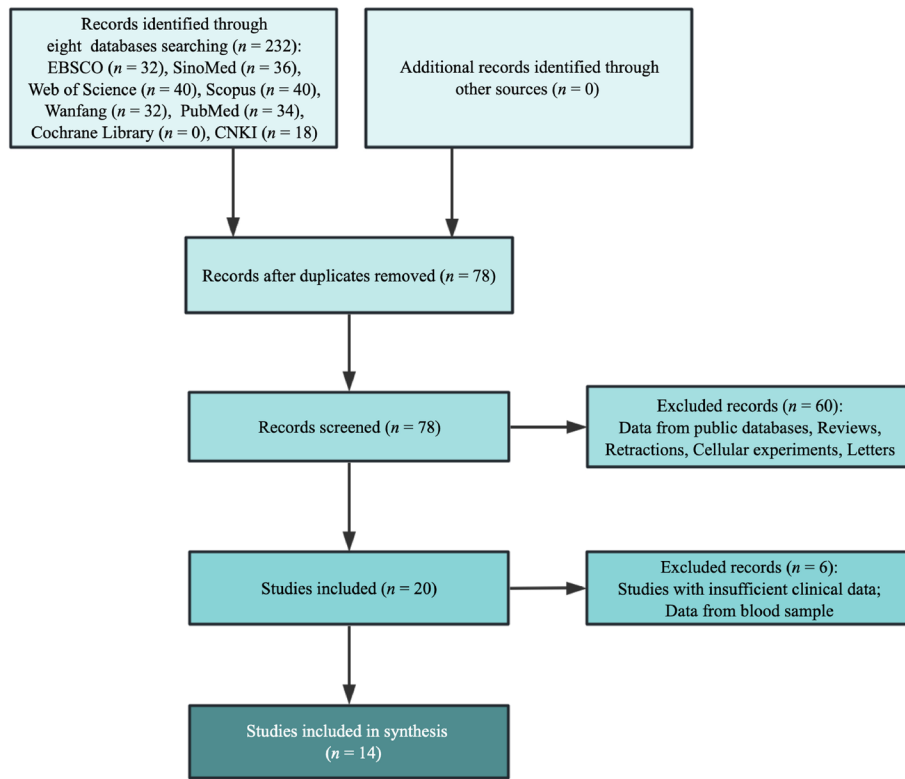


Fig. 1 Documents selection flowchart

Table 1 Main characteristics of included studies

Author	Year	Malignancy types	Sample sizes (n)	Detection method	Outcomes	HR (95% CI) for OS	Follow up time (m)	Data extraction method	NOS
Yang [33]	2023	Cervical carcinoma	20	qRT-PCR	CP	NA	NA	NA	7
Lu [16]	2022	Papillary thyroid carcinoma	98	qRT-PCR	CP	NA	NA	NA	6
Luo [34]	2021	Colorectal carcinoma	60	qRT-PCR	OS	2.24 (0.63, 8.01)	60	Indirectly	7
Zhang [35]	2021	Ovarian carcinoma	50	qRT-PCR	CP, OS	2.24 (0.65, 7.71)	40	Indirectly	6
Zhu [21]	2021	Papillary thyroid carcinoma	98	qRT-PCR	CP	NA	NA	NA	7
Xue [36]	2021	Non-small cell lung carcinoma	40	qRT-PCR	CP, OS	2.12 (0.25, 18.07)	60	Indirectly	8
Xin [22]	2020	Gastric carcinoma	41	qRT-PCR	CP	NA	NA	NA	7
Zhuang [37]	2019	Papillary thyroid carcinoma	80	qRT-PCR	CP	NA	NA	NA	7
Li [38]	2019	Non-small cell lung carcinoma	248	qRT-PCR	CP, OS	1.57 (1.08, 2.29)	110	Indirectly	8
He [39]	2019	Colorectal carcinoma	53	qRT-PCR	OS	2.53 (0.89, 7.17)	NA	NA	7
Wen [40]	2018	Papillary thyroid carcinoma	82	qRT-PCR	OS	2.02 (0.39, 10.62)	60	Indirectly	7
Lei [41]	2018	Colorectal carcinoma	132	qRT-PCR	OS	2.28 (1.15, 4.54)	60	Indirectly	7
Qiao [23]	2018	Pancreatic carcinoma	147	qRT-PCR	CP, OS	1.67 (0.93, 3)	60	Indirectly	8
Chen [42]	2017	Bladder carcinoma	66	qRT-PCR	CP	NA	NA	NA	6

Abbreviations: n number, HR hazard ratio, CI confidence interval, OS overall survival, m months, NOS Newcastle–Ottawa scale, qRT-PCR quantitative reverse transcription polymerase chain reaction, CP clinicopathologic parameters, NA not applicable

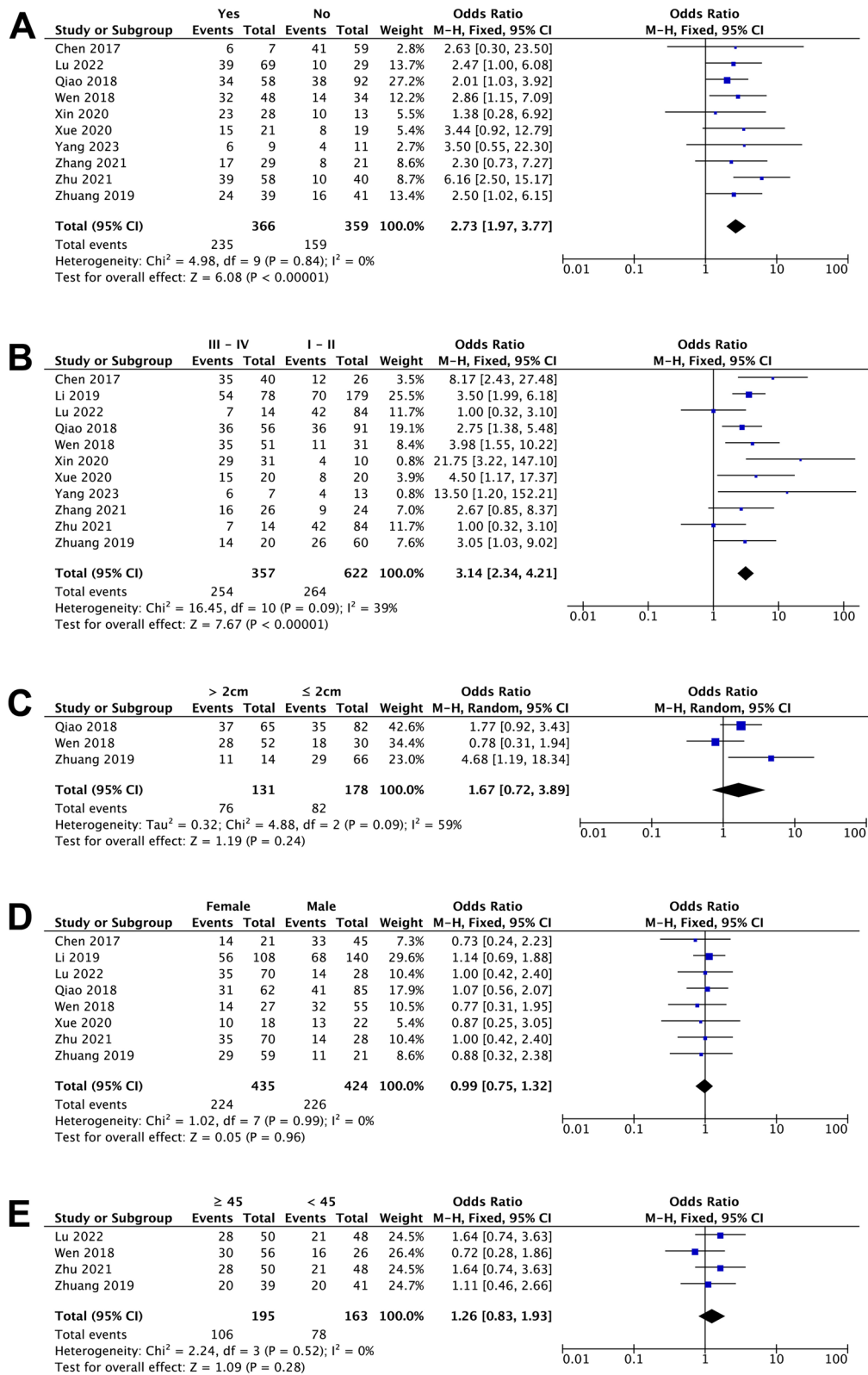


Fig. 2 The forest plots determining the association between ABHD11-AS1 expression and clinicopathological parameters (**A**, lymph node metastasis; **B** tumor stage; **C** tumor size; **D** gender; **E** age). Abbreviations: CI, confidence interval

(Fig. 2A). In addition, regarding the clinicopathologic parameter of tumor stage, 11 studies with 970 cancer cases indicated that high ABHD11-AS1 expression predicted advanced tumor stage (OR=3.14, 95%CI [2.34, 4.21], I²=39%, p<0.00001) (Fig. 2B). Also, the fixed model was applied.

Since all included studies were performed in different places, the cut-off values for tumor sizes and the age of patients were not uniform. Hence, we took tumor sizes ≤2 cm and >2 cm, along with patient age <45 years and ≥45 years as our analytical data, which were adopted by most of the included studies. Besides, we excluded two studies [33, 35] when analyzing the relationship of ABHD11-AS1 expression with patient gender because their cancer types were cervical carcinoma and ovarian carcinoma, respectively. However, there was no clinical evidence illustrating that ABHD11-AS1 expression was related to tumor sizes (OR=1.67, 95%CI [0.72, 3.89], I²=59%, p=0.24) (Fig. 2C), the gender of patients (OR=0.99, 95%CI [0.75, 1.32], I²=0%, p=0.96) (Fig. 2D) and the age of patients (OR=1.26, 95%CI [0.83, 1.93], I²=0%, p=0.28) (Fig. 2E). Moreover, all the pooled results were robust after being estimated by sensitivity analyses.

Association between ABHD11-AS1 expression and overall survival

The association between ABHD11-AS1 expression and OS was determined by eight studies with 812 cancer cases. On account of no heterogeneity existing across articles, the fixed model was adopted for analysis. The pooled results demonstrated that high ABHD11-AS1 expression was significantly associated with poor OS (HR=1.81, 95%CI [1.58, 2.06], I²=0%, p<0.00001) (Fig. 3). Furthermore, the subgroup analyses were carried out based on cancer types (digestive system cancer, respiratory system cancer, and other system cancer) and sample sizes (≥100 cases and <100 cases). As summarized in Table 2, elevated ABHD11-AS1 expression forecasted poor OS, regardless of cancer types and sample sizes (p<0.05).

Validation of the results

To further strengthen our conclusion as broadly as possible, the GEPIA2021 database was applied. As illustrated in Fig. 4, the expression of ABHD11-AS1 was substantially elevated in eight kinds of malignancies including cholangio carcinoma (CHOL), ovarian serous cystadenocarcinoma (OV), colon adenocarcinoma (COAD), pancreatic adenocarcinoma (PAAD),

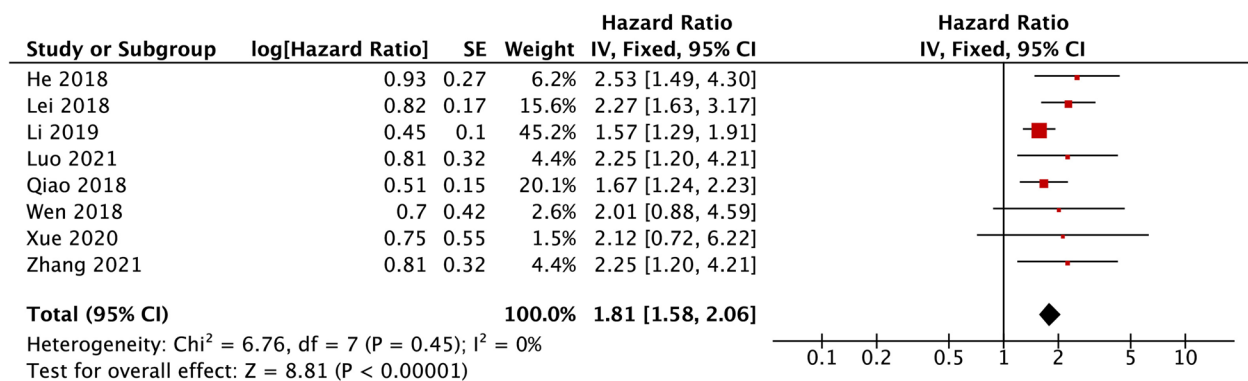


Fig. 3 The forest plot for the association between ABHD11-AS1 expression and OS. Abbreviations: CI, confidence interval

Table 2 Subgroup analysis of the association between ABHD11-AS1 expression and OS

Variables	Total cases (n)	HR 95% CI	p	I ² (%)	Model
Cancer types					
Digestive system cancer	392	2.01 [1.66, 2.44]	<0.00001	0	Fixed
Respiratory system cancer	288	1.58 [1.31, 1.92]	<0.00001	0	Fixed
Others	162	2.16 [1.31, 3.56]	0.002	0	Fixed
Sample sizes (n)					
≥100	527	1.71 [1.48, 1.98]	<0.00001	44	Fixed
<100	315	2.29 [1.70, 3.10]	<0.00001	0	Fixed

Abbreviations: n number, HR hazard ratio, CI confidence interval

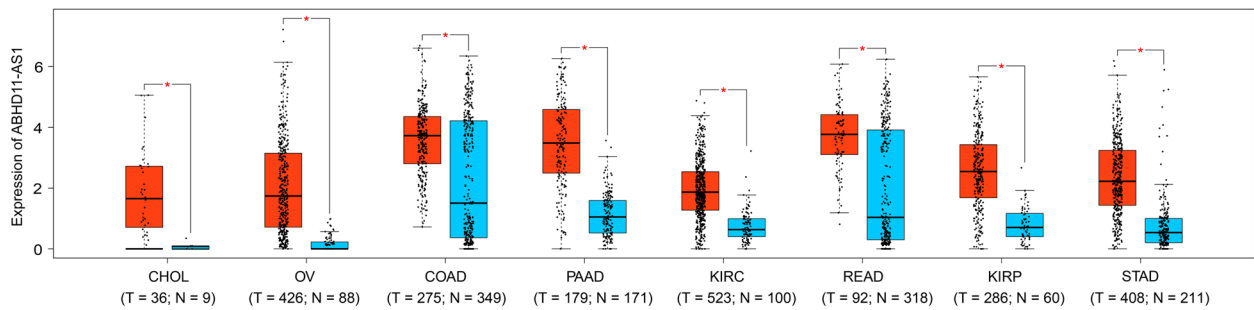


Fig. 4 The expression of ABHD11-AS1 in eight kinds of tumor tissues (red) vs. normal tissues (blue). **** $p < 0.01$. Abbreviations: T, tumor tissue; N, normal tissue; CHOL, cholangio carcinoma; OV, ovarian serous cystadenocarcinoma; COAD, colon adenocarcinoma; PAAD, pancreatic adenocarcinoma; KIRC, kidney renal clear cell carcinoma; READ, rectum adenocarcinoma; KIRP, kidney renal papillary cell carcinoma; STAD, stomach adenocarcinoma

kidney renal clear cell carcinoma (KIRC), rectum adenocarcinoma (READ), kidney renal papillary cell carcinoma (KIRP), stomach adenocarcinoma (STAD).

The results from UCSC Xena database indicated that the overexpression of ABHD11-AS1 predicted poor PFI in six kinds of cancers, such as adrenocortical carcinoma (ACC), glioblastoma multiforme (GBM), brain lower grade glioma (LGG), mesothelioma (MESO), PAAD, and uveal melanoma (UVM) (Fig. 5A - F). Besides, high ABHD11-AS1 expression predicted unfavorable DFI in four kinds of cancers like uterine carcinosarcoma (UCS), PAAD, liver hepatocellular carcinoma (LIHC), and lung squamous cell carcinoma (LUSC) (Fig. 5G - J).

In terms of DSS, data from seven cancers, such as ACC, breast invasive carcinoma (BRCA), GBM, LGG, LIHC, PAAD, and LUSC, demonstrated that ABHD11-AS1 expression was significantly associated with poor DSS (Fig. 5K - Q). Similar to the results of our meta-analysis, high expression of ABHD11-AS1 was strongly associated with poor OS in seven cancers, including LUSC, LGG, acute myeloid leukemia (LAML), head and neck squamous cell carcinoma (HNSC), GBM, ACC, and PAAD (Fig. 5R - X).

Discussion

Recently, accumulating findings have highlighted that *lncRNAs* play an essential role in tumorigenesis and are expected to become new predictive biomarkers and ideal

targets for cancer therapy [43, 44]. Therefore, in order to accelerate progress toward early detection and even eliminating malignancy, lots of studies, including this meta-analysis, have endeavored to ascertain the association of *lncRNAs* with diverse malignancies [45–49]. ABHD11-AS1, a novel *lncRNA*, has been documented to participate in the development and progression of considerable carcinomas [11]. However, the underlying biological mechanisms of ABHD11-AS1 in various malignancies are extremely sophisticated.

In cervical cancer, ABHD11-AS1 exerted its carcinogenic influence by facilitating malignant behaviors of tumor cell, such as inhibiting cell apoptosis as well as promoting cell migration, proliferation, and invasion via upregulating MARK2 and competitively binding to miR-330-5p and miR-1254 [50, 51], which was further confirmed by Yang's study as well [52]. Besides, numerous previous studies found that ABHD11-AS1 could promote papillary thyroid cancer development and progression by miR-1301-3p/STAT3, miR-199a-5p/SLC1A5, and miR-29a/EPS15L1 axis [14, 15, 21]. Meanwhile, mechanical outcomes discovered that ABHD11-AS1 could also regulate the miR-1254-WNT11, ITGA5/FAK/PI3K/Akt, and miR-133a/SOX4 axis to boost colorectal cancer progression and invasion, which was directly associated with unfavorable prognosis [17, 39, 34]. For gastric cancer, Xin et al. latterly revealed that augmented

(See figure on next page.)

Fig. 5 The prognostic value of ABHD11-AS1 based on UCSC Xena database analysis. The Kaplan–Meier curves of PFI in (A) adrenocortical carcinoma, (B) glioblastoma multiforme, (C) brain lower grade glioma, (D) mesothelioma, (E) pancreatic adenocarcinoma, (F) uveal melanoma; The Kaplan–Meier curves of DFI in (G) uterine carcinosarcoma, (H) pancreatic adenocarcinoma, (I) liver hepatocellular carcinoma, (J) lung squamous cell carcinoma; The Kaplan–Meier curves of DSS in (K) adrenocortical carcinoma, (L) breast invasive carcinoma, (M) glioblastoma multiforme, (N) brain lower grade glioma, (O) liver hepatocellular carcinoma, (P) pancreatic adenocarcinoma, (Q) lung squamous cell carcinoma; The Kaplan–Meier curves of OS in (R) lung squamous cell carcinoma, (S) brain lower grade glioma, (T) acute myeloid leukemia, (U) head and neck squamous cell carcinoma, (V) glioblastoma multiforme, (W) adrenocortical carcinoma, (X) pancreatic adenocarcinoma. Abbreviations: PFI, progression free interval; DFI, disease free interval; DSS, disease specific survival; OS, overall survival

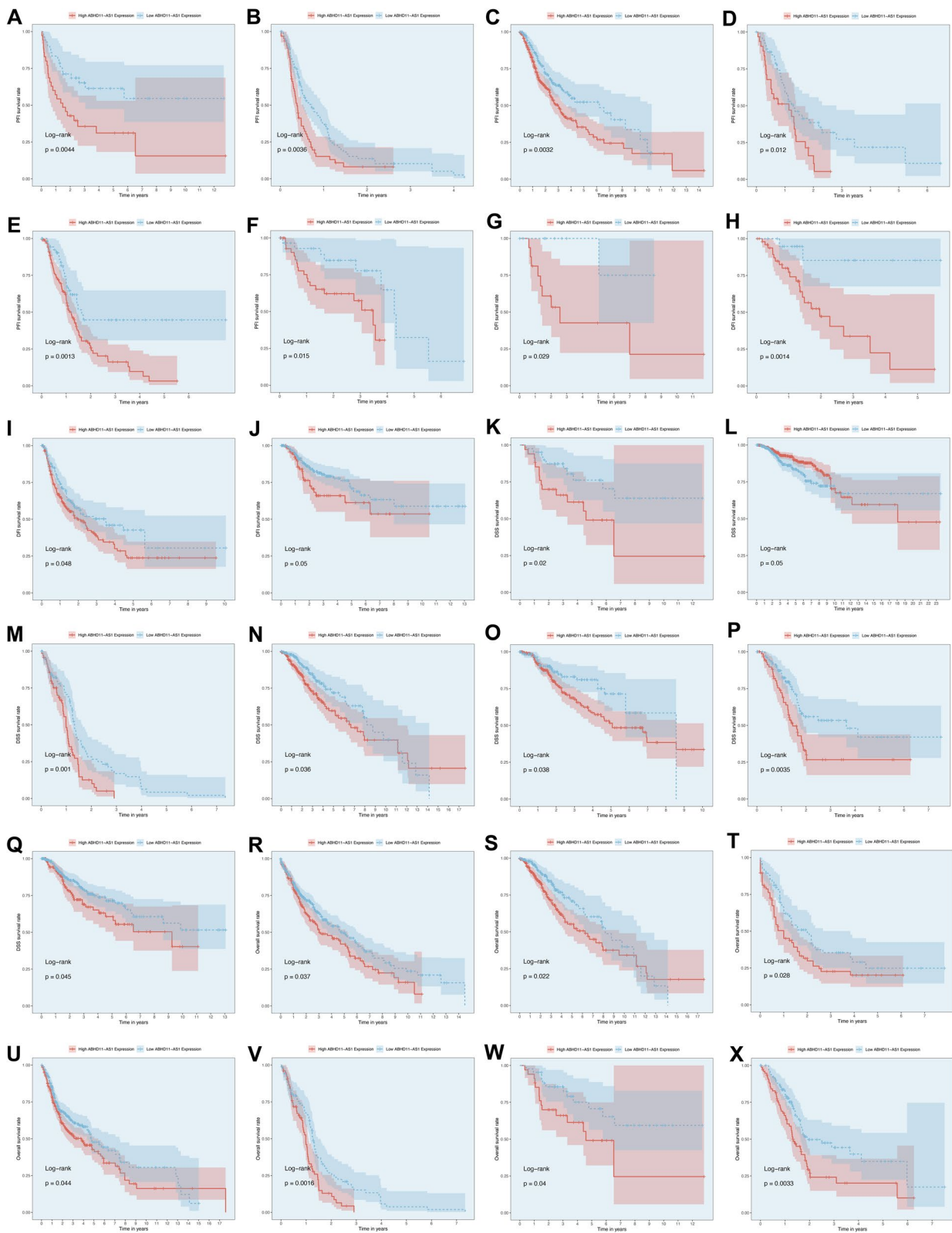


Fig. 5 (See legend on previous page.)

expression of ABHD11-AS1 significantly repressed cell apoptosis but enhanced cell proliferation by modulating the miR-361-3p/PDPK1 signaling pathway [22]. Notably, high ABHD11-AS1 expression was examined in non-small cell lung cancer, and it stimulated cell colony formation, migration, and invasion, along with proliferation by strengthening the expression of STAT1 and STAT3 [38]. Taken together, emerging evidence proved that ABHD11-AS1 acted as a vital role in diverse malignancy progression. Whereas there was no meta-analysis to quantitatively determine the association between ABHD11-AS1 and malignancy prognosis currently.

This meta-analysis enrolled 14 clinical studies comprising eight kinds of malignancies and 1215 cancer cases. It illustrated that high ABHD11-AS1 expression was closely associated with LNM (OR=2.73, 95%CI [1.97, 3.77], $I^2=0%$, $p<0.00001$), advanced tumor stage (OR=3.14, 95%CI [2.34, 4.21], $I^2=39%$, $p<0.00001$), and unfavorable OS (HR=1.81, 95%CI [1.58, 2.06], $I^2=0%$, $p<0.00001$). Notwithstanding, there was no evidence revealing that ABHD11-AS1 expression was related to tumor sizes, the gender of patients, and the age of patients. Additionally, both sensitivity and subgroup analyses verified the robustness and reliability of the results of this study. Simultaneously, elevated ABHD11-AS1 expression was further determined in eight kinds of malignancies such as CHOL, OV, COAD, PAAD, KIRC, READ, KIRP, and STAD based on validation of the GEPIA2021 database. Furthermore, DSS, DFI, and PFI were important indicators for evaluating malignancy prognosis, but merely one study [41] mentioned them. To assess the prognostic value of ABHD11-AS1 completely, we also added OS, DSS, DFI, and PFI indicators by analysing UCSC Xena database. The pooled results indicated that patients with high ABHD11-AS1 expression had worse prognosis. Briefly, these results suggested that ABHD11-AS1 may be explored as a promising biomarker for early detection of cancer and prediction of prognosis in malignant tumors.

However, several limitations in this meta-analysis ought to be noticed. First, of the 14 included studies, solely eight provided the information on OS based on Kaplan–Meier curves, which made us unable to extract the HR and 95%CI data directly. Although we adopted the Engauge Digitizer 4.1 software following the introduction of Tierney et al. [26], to obtain the corresponding data indirectly, some subjective factors might inevitably affect the data. Second, there were many high-quality studies investigating the value of ABHD11-AS1 in various malignancies outside China, such as Iran [53], the United States [11], and India [12], but they were excluded after screening with stringent inclusion criteria since they were either review or database-based study. Ultimately, only 14 publications

performed in China were included into this meta-analysis. Hence, the pooled results might lack representativeness to other regions worldwide. Third, most of the included studies adopted different cut-off values on tumor sizes and the age of patients. For example, four studies [16, 21, 37, 40] took 45 years old as the cut-off value of the age of patients, while three [35, 36, 42] and two studies [22, 23] regarded 60 and 65 years old as the cut-off values, respectively. Thereby, we included the data of 45 years old to this meta-analysis when assessing the association of ABHD11-AS1 with the age of patients, which, to some extent, might insufficiently pool the results due to relatively small sample sizes. Therefore, large-scale multicenter studies with uniform study protocols from different countries are needed to further validate the conclusions.

Conclusions

Current evidence supports that elevated ABHD11-AS1 expression predicts LNM, advanced tumor stage, and unfavorable OS, PFI, DFI, and DSS in malignancies, but there is no significant or clinical correlation with tumor sizes, patient gender, and patient age. ABHD11-AS1 may be considered as a novel biomarker for screening cancers and predicting malignancy prognosis.

Abbreviations

<i>LncRNAs</i>	<i>Long non-coding RNAs</i>
ABHD11-AS1	ABHD11 antisense RNA 1
OS	Overall survival
LNM	Lymph node metastasis
PFI	Progression free interval
DFI	Disease free interval
DSS	Disease specific survival
PFS	Progression free survival
HR	Hazard ratio
CI	Confidence interval
NOS	Newcastle–Ottawa scale
GEPIA	Gene Expression Profiling Interactive Analysis
ORs	Odds ratios
CHOL	Cholangio carcinoma
OV	Ovarian serous cystadenocarcinoma
COAD	Colon adenocarcinoma
PAAD	Pancreatic adenocarcinoma
KIRC	Kidney renal clear cell carcinoma
READ	Rectum adenocarcinoma
KIRP	Kidney renal papillary cell carcinoma
STAD	Stomach adenocarcinoma
LIHC	Liver hepatocellular carcinoma
UCS	Uterine carcinosarcoma
LUSC	Lung squamous cell carcinoma
ACC	Adrenocortical carcinoma
BRCA	Breast invasive carcinoma
GBM	Glioblastoma multiforme
LGG	Brain lower grade glioma
HNSC	Head and neck squamous cell carcinoma
LAML	Acute myeloid leukemia
MESO	Mesothelioma
UVM	Uveal melanoma

Acknowledgements

Not applicable.

Authors' contributions

Conceptualization: Guangyao Lin, Jing Wang Data curation: Guangyao Lin, Tao Ye, Jing Wang Formal analysis: Guangyao Lin, Jing Wang Investigation: Tao Ye, Jing Wang Methodology: Guangyao Lin, Tao Ye, Jing Wang Resources: Guangyao Lin, Tao Ye, Jing Wang Software: Guangyao Lin Supervision: Jing Wang Validation: Guangyao Lin, Jing Wang Writing – original draft: Guangyao Lin Writing – review & editing: Guangyao Lin, Jing Wang All the authors contributed to and approved the final manuscript.

Funding

Not applicable.

Availability of data and materials

Data were taken from publicly available publications and as such can be widely accessed. The datasets used during the current study are available from the corresponding author on reasonable request.

Data availability

No datasets were generated or analysed during the current study.

Declarations**Ethics approval and consent to participate**

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

¹Department of Gynecology, Longhua Hospital, Shanghai University of Traditional Chinese Medicine, Shanghai 200030, China. ²Department of Gynecology, The Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University, Luzhou, Sichuan 646000, China. ³Department of Obstetrics, The Affiliated Traditional Chinese Medicine Hospital, Southwest Medical University, Luzhou, Sichuan 646000, China.

Received: 1 March 2024 Accepted: 27 August 2024

Published online: 02 September 2024

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