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Research

Elevated CEP72 expression facilitates the progression of hepatocellular carcinoma and is associated with unfavorable outcomes

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Received: 4 January 2025 / Accepted: 30 April 2025
Published online: 27 May 2025

Abstract

Introduction The specific roles and mechanisms underlying the involvement of centrosomal protein 72 (CEP72) in hepatocellular carcinoma (HCC) development are not fully understood. Thus, this study aimed to explore the influence of CEP72 on the prognosis of HCC and elucidate the underlying mechanisms involved.

Material and methods CEP72 expression was verified through the use of various databases, including the TCGA, GEO, CCLE, and HPA databases. Moreover, to validate the prognostic importance of CEP72 in HCC, we conducted the Kaplan–Meier survival analyses using the GEPIA database. The connection between CEP72 and hsa-miR-139-5p was established using RT–qPCR and Western blotting. To further evaluate the role of CEP72 and hsa-miR-139-5p in tumor regulation, we conducted the CCK8 assay, transwell migration assay, and invasion assay.

Results The abnormal upregulation of CEP72 in HCC tissues was identified through the use of the TCGA, GEO, CCLE, and HPA databases. Furthermore, we observed a notable correlation between elevated CEP72 expression and an unfavorable prognosis in individuals diagnosed with HCC. Furthermore, in vitro experiments further demonstrated that CEP72 expression enhanced the proliferation, migration, and invasion of HCC cells. Finally, we explored the influence of miRNAs on CEP72 expression by analyzing CEP72 expression patterns, establishing correlations, and conducting survival analysis. Interestingly, our findings confirmed that hsa-miR-139-5p was the upstream pathway regulator of CEP72 expression.

Conclusion Based on our findings, CEP72 is a prognostic biomarker for HCC, and the miRNA-mediated expression of CEP72 may affect the prognosis of HCC patients.

Keywords Hepatocellular carcinoma · CEP72 · Hsa-miR-139-5p · Prognosis

1 Introduction

Liver cancer is a prevalent malignancy globally, with approximately 906,000 new cases reported annually. It is also the third leading cause of cancer-related deaths, accounting for approximately 830,000 fatalities each year [1]. HCC, the predominant type of primary liver cancer, accounts for approximately 80% to 90% of all HCC cases [2]. Surgical resection remains

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s12672-025-02540-0.

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the primary treatment option for early-stage HCC. Liver transplantation is another surgical option for patients with both limited tumor burden and underlying liver disease. For patients who are not eligible for surgery, locoregional therapies are employed. Transarterial chemoembolization (TACE) and radiofrequency ablation (RFA) are commonly used techniques. TACE delivers chemotherapeutic agents directly to the tumor blood supply, while RFA uses thermal energy to destroy cancer cells. In advanced cases, systemic therapies are available. Targeted therapies, such as sorafenib and lenvatinib, inhibit specific molecular pathways involved in cancer growth [3]. Clinical trials exploring novel treatments are also underway. Despite these advances, challenges in HCC treatment persist, including drug resistance and tumor recurrence. Therefore, ongoing research is critical to develop more effective therapies to improve patient outcomes.

Physiologically, the centrosome is a spindle pole component that controls cell division, polarity, growth, and migration [4]. The presence of structural and numeric centrosome aberrations may lead to chromosomal segregation errors and promote tumor progression. This process is widely regarded as a driver of chromosomal instability, leading to a significant acceleration in tumor growth and advancement [5, 6]. Research has demonstrated the significant involvement of centrosomal protein (CEP) in both the development and progression of tumors. For example, CEP55 (a sensitive component of the CEP family) interacts with SPAG5 and exerts its oncogenic activity through the PI3K/AKT pathway in HCC [7]. Centrosomal proteins, called centrosome P4.1-associated proteins (CPAPs), are important for centrosome functions such as regulating centriole elongation and microtubule polymerization during cell division. CPAP overexpression promotes angiogenesis and metastasis in HCC through the upregulation of several STAT3 target genes, including CD44 [8]. The CEP72 protein is necessary for maintaining microtubule-organizing activity and centrosome structural integrity [9]. One study showed that CEP72 may act as a putative oncogene to inhibit the mitotic function of BRCA1 to ensure chromosomal instability in colorectal cancer [10]. Studies have shown that the CEP72 genotype (rs924607) is correlated with prolonged neuropathy in childhood, and higher CEP72 expression is correlated with poor prognosis and recurrence-free survival in patients with cancer [11–13]. The researcher used RNA sequencing to analyze the total RNA of HCC and adjacent peritumor tissues from 30 HCC patients. They found that the top-ranked gene, CEP72, is one of eight signature genes for HCC [14]. However, how CEP72 affects the development of human HCC is unclear. Therefore, we performed a comprehensive bioinformatic analysis and analysis of CEP72 expression profiles to explore the role of CEP72 in prognosis prediction and its potential impact on HCC.

2 Methods

2.1 Data source and expression analysis

The difference in CEP72 expression between normal tissues and HCC tissues was assessed using various databases, including UALCAN (https://ualcan.path.uab.edu/), the Gene Expression Omnibus (GEO) database (https://www.ncbi.nlm.nih.gov/geo/), the CCLE database (https://sites.broadinstitute.org/ccle), and the Human Protein Atlas (HPA) database (https://www.proteinatlas.org/). Moreover, we used the StarBase database (https://rnasysu.com/encori/) to examine miR-139-5p expression.

2.2 Survival analysis

We used Kaplan—Meier Plotter, a powerful network dataset (https://kmplot.com/analysis/index.php?p=service), and the GEPIA database (http://gepia.cancer-pku.cn/index.html) to assess the impact of genes and miRNAs on survival in patients with HCC.

2.3 Prediction of potential upstream miRNAs for CEP72

The prediction of CEP72-targeting miRNAs in HCC was conducted through the miRWalk (http://129.206.7.150/), TargetScan (https://www.targetscan.org/vert_80/), and StarBASE databases.

2.4 Cell culture, transfection, and western blotting

Human liver carcinoma cell lines (SK-HEP-1, Huh7, and HepG2) and a normal liver cell line (LO2) were obtained from the Cell Bank at the Shanghai Institute of Cell Biology, Chinese Academy of Sciences, Shanghai, China. SK-HEP-1 cells were maintained in DMEM supplemented with 10% fetal bovine serum (FBS) at 37.0 °C in a 5% CO_2 atmosphere. The transfection samples, including si-NC, si-1-CEP72, si-2-CEP72, si-3-CEP72, hsa-miR-139-5p inhibitor, and inhibitor NC, were prepared using the liposome transfection reagent Lipofectamine 3000 as per the manufacturer's instructions.

Following transfection, the cells were cultured in DMEM supplemented with FBS for 48 h to assess transgene expression. Supplementary Table 1 provides a list of primer sequences. We used rabbit polyclonal anti-CEP72 (China, Proteintech, 19,928-1-AP) and rabbit anti-GAPDH (China, Proteintech, 10,494-1-AP) for Western blot analysis at dilutions of 1:2,500 and 1:5,000, respectively.

2.5 RT-quantitative PCR

Total RNA was extracted with the FastPure Cell Total RNA Isolation Kit (Promega, China) according to the manufacturer's instructions. Reverse transcription of 1 μg of RNA to cDNA was performed. For real-time PCR analysis, the ChamQ Universal SYBR qPCR master mix kit from Vazyme Biotech Co., Ltd., in China was utilized following the manufacturer's guidelines. A LightCycler 96 real-time PCR system (Roche Diagnostics GmbH, Mannheim, Germany) was used to quantify the RNA expression levels of the target genes, with the GAPDH and U6 levels serving as internal controls.

2.6 Dual-luciferase reporter assay

The predicted 3′-untranslated region (3′-UTR) fragment of CEP72 targeted by hsa-miR-139-5p was cloned into either pMIR-CEP72-WT plasmids (designated "CEP72-WT") or pMIR-CEP72-Mut plasmids (IGEbio) containing a site-directed mutation in the hsa-miR-139-5p-binding region of the CEP72 3′-UTR (termed "CEP72-MUT"). Following this, cells were co-transfected with the respective plasmid constructs (WT or MUT) alongside hsa-miR-139-5p-mimics-NC or hsa-miR-139-5p mimics.

2.7 Cell proliferation assay

For the cell proliferation assay, 1×10^3 cells were uniformly distributed on 96-well plates, with 5 replications per well. Cell proliferation was monitored using a CCK-8 assay, and absorbance readings were taken at 0, 24, 48, 72, and 96 h.

2.8 Transwell assays

For the migration and invasion assays, we used transwell inserts (3422, Corning, USA) with an 8-μm pore size. To conduct invasion assays, we coated a 24-well permeable support plate with Matrigel matrix (354234, Corning, USA) and incubated it at 37.0 °C for one hour to solidify. In the transwell invasion assay, 1×10^5 cells were seeded in the upper chamber of the transwell inserts in serum-free medium, and the lower chamber was filled with medium containing 10% FBS. For the migration assays, the inserts were seeded with 5×10^4 cells suspended in serum-free medium, while the lower chamber was filled with 500 μL of medium supplemented with 10% FBS. After incubating at 37.0 °C for 48 h, the membrane was subjected to crystal violet staining, and subsequently, the migrated and invaded cells were quantified using ImageJ software.

2.9 Statistical analysis

An automated online database was utilized for statistical analysis in this study. Statistical significance levels were established as follows: $^*P < 0.05$, $^{**}P < 0.01$, and $^{***}P < 0.001$. A P value of *0.05 was considered to indicate statistical significance. Each experiment was independently replicated a minimum of three times.

3 Results

3.1 CEP72 was significantly upregulated in HCC

Analysis of gene expression data from the TCGA database indicated elevated levels of CEP72 expression in HCC tissues relative to normal tissues (Fig. 1A). Consistent with these findings, the expression patterns of CEP72 in the GSE101728, GSE29721, GSE54236, and GSE101685 datasets agreed with those in the TCGA dataset (Fig. 1B–E). Furthermore, high CEP72 expression was observed in the majority of HCC cell lines in the CCLE database (Fig. 1F). To confirm the increased expression of CEP72 at the protein level in HCC, immunohistochemistry (IHC) data were downloaded from the HPA database (Fig. 1G) analyzed using a semi-quantitative scoring method, with staining intensities defined as follows: 0 (no cellular labeling), 1 (weak intensity), 2 (moderate intensity), and 3 (strong intensity).. Taken together, these results offer robust evidence supporting the upregulation of CEP72 in HCC.

3.2 CEP72 expression was related to patient outcomes but not to clinicopathological parameters

CEP72 expression levels in the TCGA database were analyzed to evaluate their associations with the clinicopathological characteristics of HCC patients. According to our findings, no significant correlation was detected between CEP72 expression and patient characteristics, such as sex, age, pathological stage, or tumor grade (Fig. 2A–D). Furthermore, the analysis of Kaplan—Meier curves and data from the GEPIA database demonstrated a significant association between decreased CEP72 expression and improved disease-free survival (DFS) (Fig. 2F, 2H) as well as overall survival (OS) (Fig. 2E, 2G). Through the above analyses, higher CEP72 would typically indicate a better prognosis of HCC.

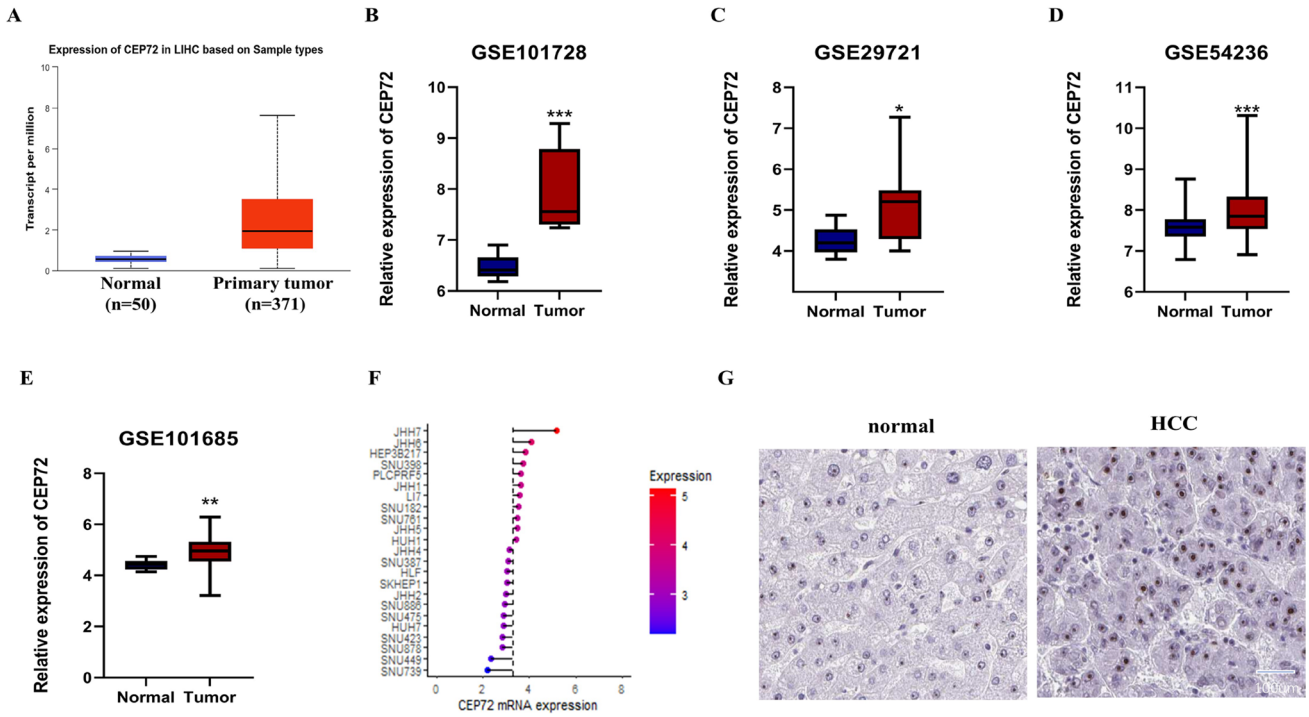

Fig. 1 Expression analysis of CEP72 in the databases. Relative expression levels of CEP72 in 371 HCC tissues and 50 normal tissues from the TCGA database **A**. Relative expression levels of CEP72 in the GSE101728 (n = 7, t = 7), GSE29721 (n = 10, t = 10), GSE54236 (n = 80, t = 81), and GSE101685 (n = 8, t = 24) datasets **B**–**E**. Relative expression levels of CEP72 in HCC cell lines from the CCLE database **F**. Immunohistochemical analysis of CEP72 in HCC samples from the HPA database **G**

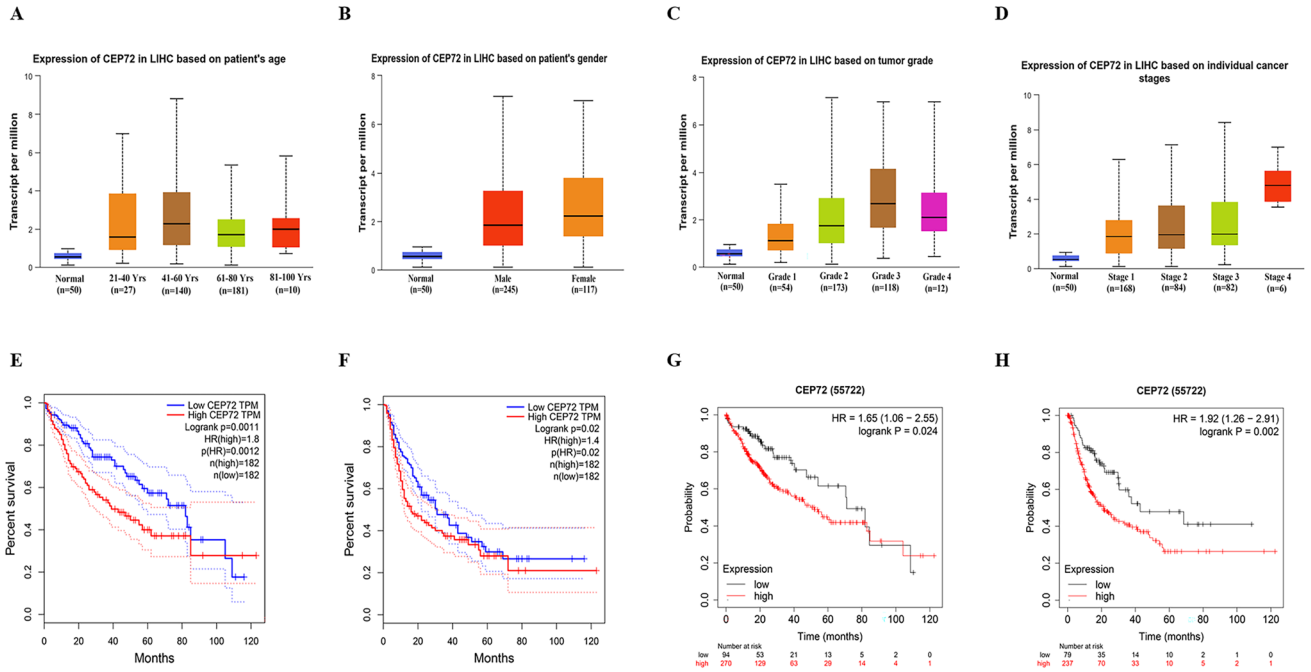

Fig. 2 The correlation between CEP72 expression and clinicopathologic parameters and outcomes was assessed. Associations of CEP72 expression with clinicopathologic parameters from the TCGA database, including age **A**, sex **B**, grade **C**, and stage **D**. The associations of CEP72 expression with OS and DFS in HCC patients were determined by Kaplan–Meier survival analysis **G**–**H** and GEPIA survival analysis **E**–**F**

3.3 The promotion of HCC cell proliferation, migration, and invasion was attributed to CEP72

To explore the potential impact of CEP72 on the promotion of HCC cell proliferation, migration, and invasion, cells were transfected with si-1-CEP72, si-2-CEP72, and si-3-CEP72, with si-NC serving as the control group. Si-1-CEP72, si-2-CEP72, and si-3-CEP72 effectively inhibited CEP72 mRNA expression (Fig. 3A). To determine the extent of CEP72 knockdown in the SK-HEP-1 cell line, we used Western blot analysis. Si-1, si-2, and si-3 significantly reduced the protein level of CEP72 (Fig. 3B). CCK8 assays demonstrated significant inhibition of HCC cell proliferation upon CEP72 knockdown (Fig. 3C and Fig. S1A). To evaluate the potential effect of CEP72 on HCC migration and invasion, SK-HEP-1 and HepG2 cells were subjected to a transwell assay. The findings demonstrated a notable decrease in the migratory ability (Fig. 3D–E and Fig. S1B–C) and invasive potential (Fig. 3F–G and Fig.S1D-E)) of HCC cells upon inhibition of CEP72. These findings supported that CEP72 promoted HCC cell proliferation, migration, and invasion.

3.4 hsa-miR-139-5p, a possible upstream miRNA regulator of CEP72, could suppress the progression of HCC

Noncoding RNAs have been recognized for their significant involvement in gene expression modulation. The differential miRNA expression profiles in the GSE115016 dataset were compared between HCC tissues and paracancerous normal tissues via microarray analysis. We analyzed the differentially expressed miRNAs in the GSE115016 dataset (Fig. 4A). Then, the miRWalk, TargetScan, and StarBase databases were used to predict potential miRNA regulators of CEP72. We determined the intersection of the regulatory factors upstream of the GSE115016 dataset and CEP72 via the miRWalk, TargetScan, and StarBASE databases (Fig. 4B). Our study identified hsa-miR-139-5p as a promising miRNA candidate involved in the regulation of CEP72. To explore the link between CEP72 and hsa-miR-139-5p, we carried out a correlation analysis and observed a noteworthy inverse correlation between the two (Fig. 4C). Subsequently, we examined the expression profiles of hsa-miR-139-5p in HCC and observed substantial downregulation in both HCC tissues and cell lines (SK-HEP-1, Huh7, and HepG2) compared to the normal liver cell line (LO2) (Fig. 4D–E). Furthermore, we evaluated the expression patterns and prognostic relevance of hsa-miR-139-5p in individuals with HCC (Fig. 4F). Interestingly, our results revealed a noteworthy association between increased levels of hsa-miR-139-5p and an adverse prognosis in patients with HCC. These results support the idea that hsa-miR-139-5p may act as a regulatory miRNA that targets the CEP72 gene upstream.

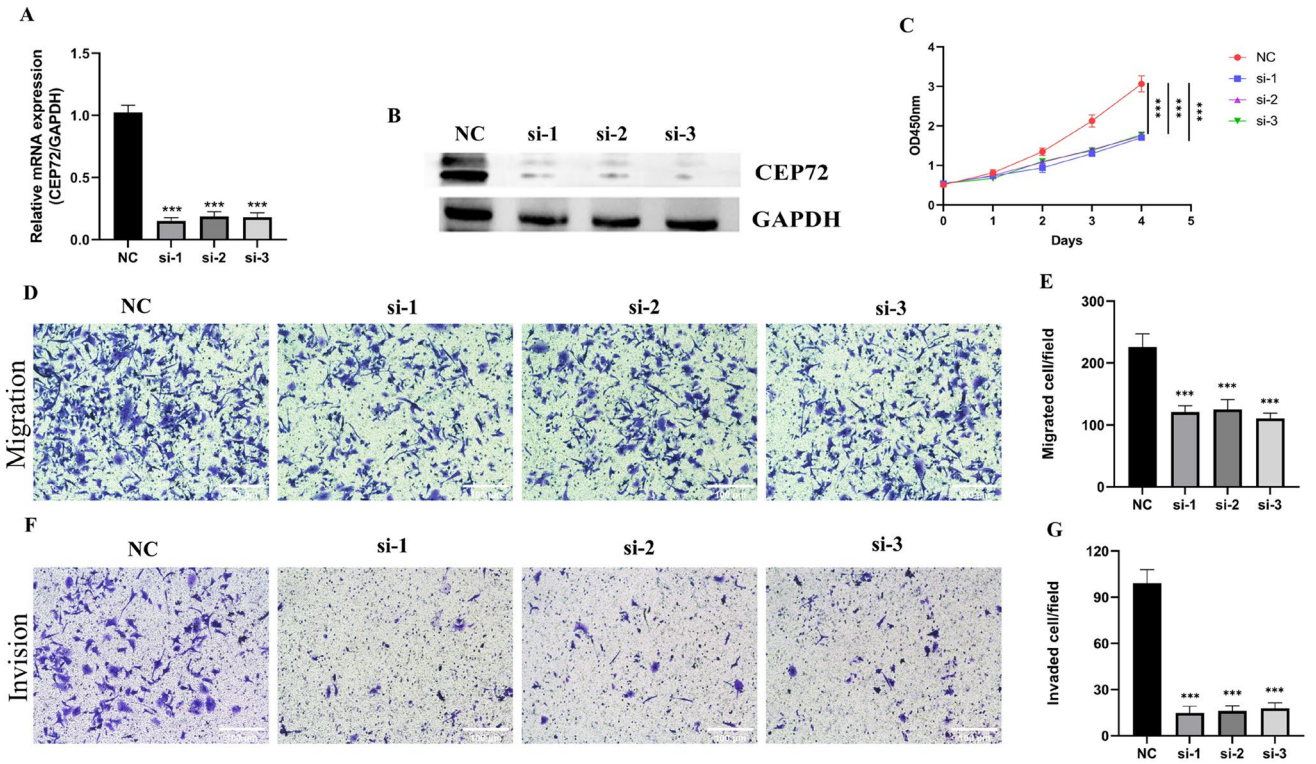

Fig. 3 Downregulation of CEP72 led to decreased proliferation, migration, and invasion in HCC cells. RT—qPCR analysis of CEP72 mRNA (**A**) and Western blot analysis (**B**) in HCC cells (SK-HEP-1) transfected with si-NC, si-1, si-2, or si-3. The effect of CEP72 knockdown on HCC cell proliferation, as determined by the CCK-8 assay (**C**). Cell migration was assessed through Transwell assays **D**–**E** and invasion **F**–**G** of cancer cells after the downregulation of CEP72. Representative images of six random fields per well are shown (100 × magnification)

To test this hypothesis, hsa-miR-139-5p was initially suppressed in the HCC cell line SK-HEP-1. Subsequently, the impact of the hsa-miR-139-5p inhibitor on the expression of CEP72 RNA was assessed. A considerable reduction in hsa-miR-139-5p expression was detected in the cohort treated with the hsa-miR-139-5p inhibitor (Fig. 5A). To validate hsa-miR-139-5p/CEP72 interaction, StarBASE-predicted binding sites (Fig. 5B) were tested via dual-luciferase reporter assay. SK-HEP-1 cells co-transfected with CEP72-WT and hsa-miR-139-5p mimics/NC showed reduced luciferase activity and CEP72-MUT abolished this suppression (Fig. 5C). Additionally, we conducted a comparative analysis of the protein level of CEP72 in the presence or absence of the hsa-miR-139-5p inhibitor, revealing a marked increase in the CEP72 protein level in the hsa-miR-139-5p inhibitor group (Fig. 5D). These experimental findings suggest that hsa-miR-139-5p effectively targets CEP72. To investigate the potential regulatory role of hsa-miR-139-5p in HCC proliferation, migration, and invasion, we conducted CCK-8 and Transwell assays, specifically in SK-HEP-1 cells. Our results demonstrated that the inhibition of hsa-miR-139-5p significantly enhanced the proliferation (Fig. 5E), migration (Fig. 5F–G), and invasion (Fig. 5H–I) of SK-HEP-1 cells. However, overexpression of hsa-miR-139-5p reversed the above phenomenon (Fig. S2A–E). These findings underscore the potential of hsa-miR-139-5p as a regulator of crucial aspects of HCC progression.

4 Discussion

In our study, the role of CEP72 in HCC was comprehensively investigated using RNA expression data from the TCGA, CCLE, and GEO databases. Our analysis revealed an increase in CEP72 in HCC tumor tissues, indicating worse prognostic outcomes for patients with higher expression levels of this gene. This upregulation of CEP72 in HCC was further confirmed through IHC data from the HPA database. Moreover, we carried out functional experiments, such as CCK8, migration, and invasion assays, to investigate how inhibiting CEP72 affects the growth, movement, and invasion of HCC cells. According to these findings, inhibiting CEP72 dramatically reduced the migratory potential, invasive capacity, and growth rate of HCC cells. These findings provide additional support for previous studies suggesting that the upregulation of CEP72 in HCC patients is associated with unfavorable outcomes.

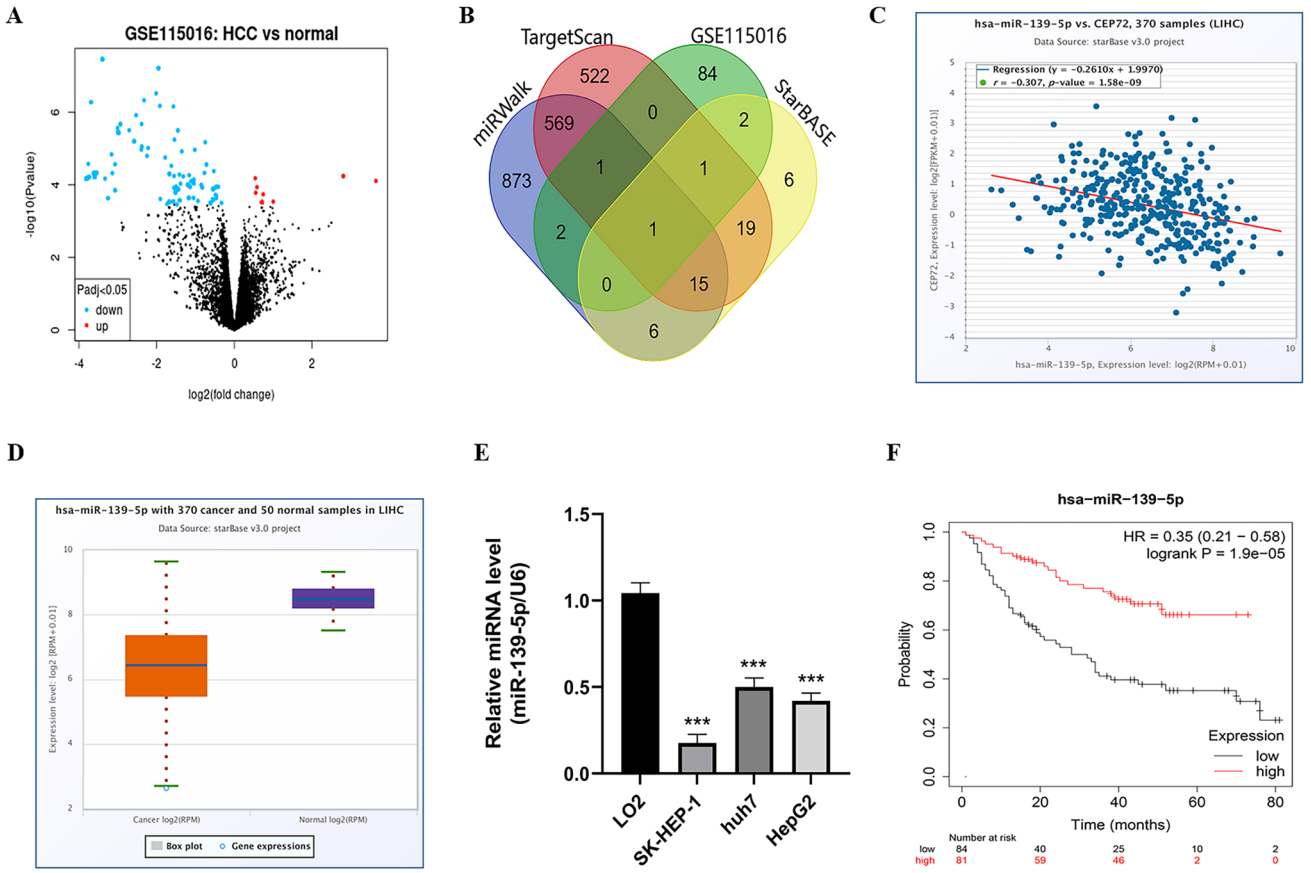

Fig. 4 Identification of potential upstream miRNA targets associated with CEP72 in HCC. The volcano map displays the differential analysis of miRNA expression between normal tissue and HCC tissue from the GSE115016 dataset (**A**). A diagram showing the shared regulatory miRNAs linked with CEP72 (**B**). The StarBASE database was used to examine the correlation between the anticipated hsa-miR-139-5p and CEP72 in HCC (**C**). The StarBase database was used to compare the expression of hsa-miR-139-5p between HCC and normal tissue samples (**D**). RT—qPCR was used to assess the expression of hsa-miR-139-5p in LO2 and HCC cell lines (SK-HEP-1, Huh7, and HepG2) (**E**). The prognostic value of hsa-miR-139-5p in HCC was assessed using Kaplan—Meier Plotter (**F**)

The global health concern of HCC persists, characterized by a poor prognosis and elevated mortality rates. For HCC patients, cancer-related deaths are mainly due to late diagnosis, metastasis, and rapid progression [15]. Survival rates can be significantly improved by early diagnosis and effective treatment. Therefore, there is an urgent need for early diagnosis and prognostic markers for HCC patients [16]. Emerging oncogenomic evidence positions CEP72 as a multifaceted regulator across malignancies. Pan-cancer analyses (TCGA Pan-Cancer Atlas) reveal consistent CEP72 upregulation in invasive subtypes of breast, lung adenocarcinoma, and colorectal cancers. CEP72 is increasingly believed to affect human cancers, including HCC. Lüddecke et al. suggested that CEP72 could inhibit the mitotic function of BRCA1 and lead to chromosomal instability, leading to cancer progression in colorectal cancer [10]. However, the understanding of CEP72 in HCC is still insufficient. While CEP72 copy number variations predict response in vitro, clinical validation remains elusive due to spatial heterogeneity in tumor biopsies.

There are three types of ncRNAs: miRNAs, lncRNAs, and circRNAs. They regulate gene expression by interacting through ceRNAs [17–20]. We analyzed the GSE115016 dataset and several prediction programs, including miRWalk, StarBASE, and TargetScan, to explore upstream regulatory miRNAs for CEP72. Consequently, by conducting correlation analysis, examining expression patterns, and analyzing survival data, hsa-miR-139-5p emerged as the top candidate for regulating CEP72. To validate this discovery, we performed a transfection experiment and employed RT—PCR and western blot assays to measure the mRNA and protein levels of CEP72, respectively. The findings revealed that the suppression of hsa-miR-139-5p expression resulted in a notable decrease in the mRNA and protein levels of CEP72. Importantly, prior research has demonstrated the regulatory impact of hsa-miR-139-5p on the proliferation and migration abilities of cancer cells [21–23]. Chi et al. reported that the interaction of hsa-miR-139-5p with YTHDF1 promoted the proliferation of HCC cells [24]. Montero-Conde et al. reported a reduction in cancer cell migration and proliferation upon exogenous expression

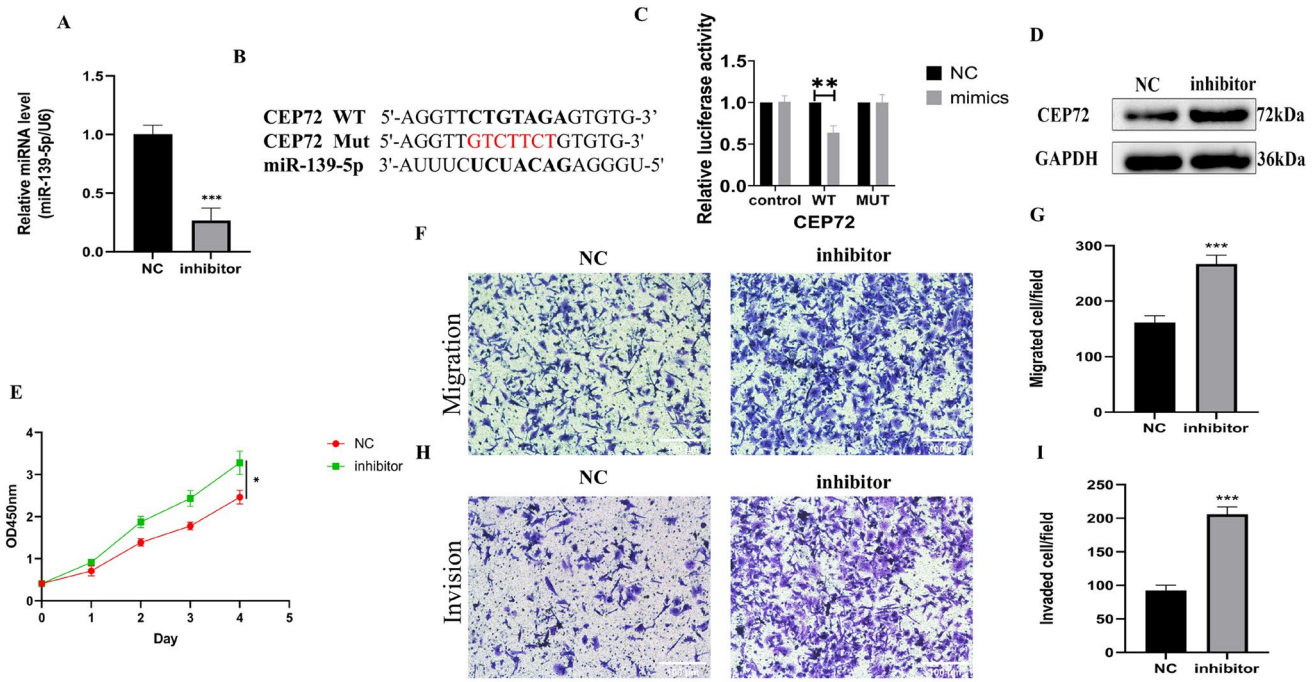

Fig. 5 The connection between hsa-miR-139-5p and CEP72 and HCC progression. The relative expression of hsa-miR-139-5p was analyzed via RT—qPCR in HCC cells (SK-HEP-1) after transfection with either the hsa-miR-139-5p inhibitor or NC (**A**). Bioinformatic analysis of hsa-miR-139-5p/CEP72 binding sites (**B**). SK-HEP-1 cells were co-transfected with hsa-miR-139-5p mimics or NC alongside CEP72-WT/MUT, and luciferase activity was then assayed (**C**). Western blot analysis of the CEP72 protein level in SK-HEP-1 cells treated with hsa-miR-139-5p inhibitor or NC inhibitor (**D**). The effect of hsa-miR-139-5p knockdown on HCC cell proliferation, as determined by a CCK-8 assay **E**. Transwell assays were performed to determine the migratory (**F**–**G**) and invasive **H**–**I** properties of cancer cells after the downregulation of hsa-miR-139-5p. Representative images of six random fields per well are shown (100 × magnification)

of hsa-miR-139-5p [25]. An intriguing discovery from our research was the increased advancement capabilities of HCC cells upon suppression of hsa-miR-139-5p expression, which corroborates the aforementioned studies.

5 Conclusions

In summary, our extensive analysis involving bioinformatics and laboratory experiments revealed the promising prognostic significance of CEP72. Additionally, we identified the important underlying mechanism in HCC progress, namely hsa-miR-139-5p/CEP72 axis. We believe that HCC may be treated efficiently and effectively by blocking CEP72.

Acknowledgments The study was supported by National Natural Science Foundation of China (NSFC No. 82360495).

Author contributions Conceptualization, project administration, data curation, and writing-original draft preparation,Qiuxia Wei; Conceptualization, project administration, data curation, and writing-review and editing, Nengren Tan. All authors have read and agreed to the published version of the manuscript.

Funding There is not funding support.

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

Declarations

Ethics approval and consent to participate Not applicable.

Competing interests The authors declare no competing interests.

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