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In smokers, the axis NCAPG/hsa-let-7b-5p/TMPO-AS1 promotes lung adenocarcinoma

Prerna Vats, Sakshi Nirmal, Rajeev Nema

Department of Biosciences Manipal University Jaipur, Dehmi Kalan, Jaipur-Ajmer Expressway, Jaipur, Rajasthan, India

ABSTRACT

Background: Smoking is linked to high morbidity and mortality rates of lung cancer, emphasizing the need for a better understanding of prognosis-related mRNA/miRNA/lncRNA-ceRNA networks.

Materials and methods: The study utilized databases like OncoMX, The University of ALabama at Birmingham CANcer data (UALCAN), OncoDB, ENCORI, Kaplan-Meier (KM) Plotter, miRNet, CancerMIRNome, TISIDB, and TIMER2.0 to analyze *NCAPG*/miRNA and LncRNA expression in lung cancer tumors and healthy tissues.

Results: The *NCAPG* gene is overexpressed in lung cancer cells. High *NCAPG* expression is associated with adenocarcinoma patients with a log fold change of 8.7 in case of tumor *vs*. normal samples (t = 515, n = 59). Overexpression of *NCAPG* indicates poor overall survivability in lung adenocarcinoma (LUAD) patients [hazard ratio (HR) = 1.6, confidence interval (CI) = 1.34–1.9, p = 9.9e–08] and those with a smoking history (HR = 1.44, CI = 1.11-1.87, p = 0.0062), but not significantly associated with lung squamous cell carcinoma (LUSC). miRNA hsa-let-7b-5p negatively correlates (R = –0.348) with *NCAPG* expression, with its down expression associated with poor survivability (HR = 0.71), while lncRNA TMPO-AS1 positively correlates (R = 0.575) with the *NCAPG* axis, with its overexpression associated with poor survivability (HR = 2.16).

Conclusion: Elevated levels of *NCAPG* and TMPO-AS1 in lung adenocarcinoma patients lead to aggressive growth and poor prognosis. miRNA hsa-let-7b-5p, a key miRNA, may inhibit these factors, potentially improving patient prognosis. Further research and clinical trials are needed to validate this targeted therapy.

Keywords: lung adenocarcinoma; *NCAPG*; ceRNA network; prognosis; smoking

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Introduction

Molecular targeting has revolutionized therapy for non-small cell lung cancer (NSCLC), but it only benefits patients with related driver gene mutations, which still face challenges with acquired drug resistance [1]. NSCLC is a major cause of cancer-related death, with most patients diagnosed with two main subtypes: lung adenocarcinoma (LUAD) and lung squamous cell carcinoma (LUSC). The survival rate of lung cancer remains low due to its high metastatic potential and chemotherapeutic resistance [2]. Treatment varies depending on the type of cancer and diagnosis stage, with targeted therapy being recommended for advanced-stage treatment. Several genes, including *EGFR*, *ALK*, *KRAS*, *ROS1*, *BRAF*, *NTRK1/2/3*, *MET*, and *RET*, have indicated their genetic alteration status as predictive biomarkers, yet approximately 30% of patients lack these genetic alterations [3]. A small group of diagnostic biomarkers are used every day in clinical practice. These are p40 and thyroid transcrip-

Address for correspondence: Dr Rajeev Nema, Assistant Professor Department of Biosciences, Manipal University Jaipur, Dehmi Kalan, Jaipur-Ajmer Expressway, Jaipur, Rajasthan, 303007, India; e-mail: rajeev.nema@jaipur.manipal.edu

tion factor 1 (TTF-1) for immunohistochemistry (IHC), cytokeratin 19 fragment (CYFRA 21-1), and carcinoembryonic antigen (CEA) for blood and serum testing [4]. The addition of novel markers is required to improve specificity and sensitivity. To classify the patient's risk and decide the treatment strategy, prognostic prediction is important. Various cancers highly express NCAPG, a mitosis-associated chromosomal condensation protein, affecting tumor cell proliferation, invasion, metastasis, and apoptosis [5]. Tumor size, histological grading, Tumor–Node–Metastasis (TNM) stage, total survival rate, and other factors strongly correlate with its expression. We need to conduct further exploration to deepen our understanding of cancer pathogenesis, discover new biomarkers, and gain insights into personalized cancer treatment. The levels of NCAPG, hsa-let-7b-5p (micro ribonucleic acid — miRNA), and TMPO-AS1 (long non-coding ribonucleic acid — lncRNA) were looked at in lung adenocarcinoma and healthy tissue in this study. The goal was to understand how cancer controls the expression of *NCAPG* genes through their regulatory mechanism or competitive endogenous RNA (ceRNA) network. The ceRNA network consists of the coding (mRNAs) and the non-coding regions of DNA (miRNAs and lncRNAs) and monitors proper gene expression. Any dysregulation in their levels will affect this regulatory mechanism/ sponge the expression of *NCAPG* genes.

Materials and methods

Expression analysis NCAPG

The study used The University of ALabama at Birmingham CANcer data (UALCAN) [6] database to study the gene expression of *NCAPG* across The Cancer Genome Atlas (TCGA) cancers. We conducted the differential expression analysis of *NCAPG* using the UALCAN, OncoDB and Encyclopedia of RNA Interactomes (ENCORI) [7] databases, evaluating the expression level between tumor and normal in both LUAD and LUSC.

Survival analysis

We utilized the Kaplan-Meier Plotter [8] for survival analysis on lung cancer datasets, focusing on the gene symbol *NCAPG*, a key factor in the disease. The analysis included survival analysis of three parameters: overall survival (OS), first progression (FP), and post progression survival (PPS), along with the histological subtypes adenocarcinoma and squamous cell carcinoma, as well as their associations with smoker patients, inclusive of both genders ("Gene symbol, Affy id: *NCAPG*, 218662_s_at").

The study focuses on ceRNA regulatory network analysis

The study used the ENCORI and miRNet [9] databases to analyze the miRNA/lncRNA network associated with *NCAPG*. The ENCORI databases verified the correlation between *NCAPG* and miRNA. We further evaluated the prognostic significance of *NCAPG*-associated miRNA using the Kaplan-Meier (KM) plotter. We used the Enrichr [10] and UALCAN databases to identify lncRNA associated with *NCAPG*, KM Plotter database was used to check the prognostic significance, and validated the co-relation values using ENCORI.

Tumor infiltration immune cells (TIICs) analysis

TISIDB, an interactive web application to analyze several immune cells correlated with *NCAPG,* was used. It provided valuable insights into TIICs in lung cancer subtypes, including CD8 T cells, CD4 T cells, T follicular helper cells, natural killer (NK) cells, mast cells, monocytes, neutrophils, eosinophils, resting dendritic cells, and plasma cells. Further, the correlation was examined between the strong associated immune cell and *NCAPG* using TIMER2.0.

Statistical analysis

We analyzed *NCAPG* gene expression using t-tests and online database models to compare tumors and tissues. We examined the relationship between *NCAPG* gene expression and prognosis. *NCAPG* performance, heterogeneity, and gene enrichment. A log-rank test was used to compare survival rates, and the significance level was $p < 0.05$.

Results

Prognostic value of NCAPG is shown in pan-cancer study

The OncoMX database showed that *NCAPG* was overexpressed in lung cancer with a fold change of

Table 1. Gene expression based Pan-Cancer Analysis

UniProtKB/ /Swissport AC	Gene symbol	Log2 F.C.	p-value	Adj. p-value	Significant	Expression trend	TCGA study	Source
Q9BPX3	*NCAPG*	–0.2	0.254	0.345	No	Down	Thyroid cancer	TCGA
		1.69	1.32E–21	1.08E–19	Yes	Up	Stomach cancer	
		3.78	3.04E–78	3.46E–75	Yes	Up	Liver cancer	
		3.53	1.02E–83	5.24E–81	Yes	Up	Uterine cancer	
		2.01	2.69E–16	1.64E–14	Yes	Up	Bladder cancer	
		1.29	1.15E–27	5.14E–26	Yes	Up	Head_and_neck cancer	
		3.48	1.03E–241	7.22E–239	Yes	Up	Lung cancer	
		1.88	5.32E–13	9.26E–11	Yes	Up	Esophageal cancer	
		1.18	1.52E–34	3.05E–33	Yes	Up	Colorectal cancer	
		1.68	7.52E–21	2.14E–19	Yes	Up	Prostate cancer	
		2.23	2.00E–70	9.66E–69	Yes	Up	Kidney cancer	
		3.06	1.02E–161	3.54E–159	Yes	Up	Breast cancer	

TCGA — The Cancer Genome Atlas

3.48, as listed in Table 1. Using the UALCAN databases for more research, it was found that *NCAPG* was overexpressed in LUSC and LUAD. This is shown in Figure 1A. Next, we analyzed the differential expression of *NCAPG* in LUAD and LUSC using the UALCAN database. The results showed that *NCAPG* expression was significantly overexpressed in both subtypes, with a fold change of 8.7 and 9.5, respectively, as shown in Figures 1B–C. Further, Figure 1D–G shows a consistent pattern of overexpression using two different databases, OncoDB and ENCORI, in both LUAD and LUSC.

Survival and prognostic value of NCAPG

The study then determined *NCAPG*'s prognostic significance using the KM Plotter database. Here, we first analyzed *NCAPG* gene with three different survival status (OS/FP/PPS), along with LUAD, LUSC, sex, and smoking parameters as shown in Table 2.

Based on the significant levels, we further analyzed selected parameters. And the results showed that high expression of *NCAPG* was significantly linked to a poor prognosis in lung cancer patients, with a low expression group having an overall survival (OS) of 92.97 months and a high expression group having an OS of 48 months [hazard ratio (HR) = 1.52, confidence interval (CI) = 1.35–1.72, p = 4.1e–12). Next, our analysis of LUAD and OS patients (HR = 1.6, CI = 1.34–1.9, p = 9.9e–08) suggested a significant association between *NCAPG* overexpression and poor prognosis. However, when we applied this to LUSC and OS patients, we found a less significant association (HR = 1.01, CI = 0.83–1.22, p = 0.93), with a low expression cohort (62.47 months) and a high expression cohort (51 months), as depicted in Figure 2A–C. Patients with LUAD who had smoked in the past had a much lower chance of survival (HR = 1.44, CI = 1.11–1.87, p = 0.0062), a low expression cohort (93) and a high expression cohort (71 months), compared to people with squamous cell carcinoma and a smoking history (HR = 1.22, CI = 0.82–1.75, p = 0.34), a low expression cohort (78) and a high expression cohort (64.1 months), as shown in Figure 2D–E.

Competitive endogenous RNA regulation network analysis

microRNAs and lncRNA influence mRNA dysregulation, which is associated with poor prognosis, tumor progression, and metastases. Here, we first analyzed the lncRNA association with NCAPG using the Enrichr database, selecting the top 5 lncRNAs for further analysis due to their significant association with a p value of less than 0.05 as mentioned in Supplementary File — Tab. S1. We looked more closely at these lncRNAs using the UALCAN database to see if there were any differences in how they were expressed between people with LUAD and people who were not affected by LUAD. Our results showed that only three of the lncRNAs were significantly over-

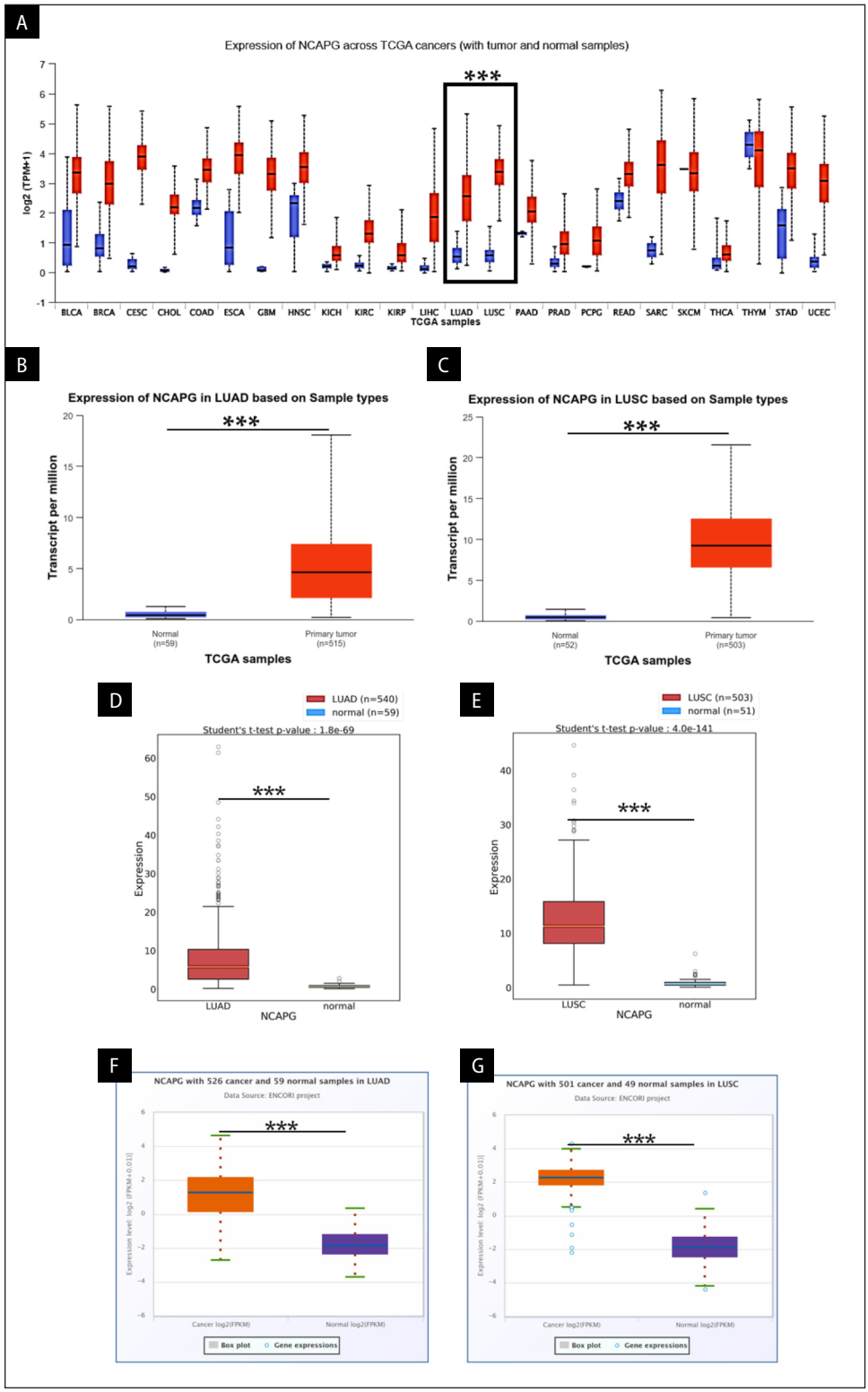

Figure 1. Expression pattern of *NCAPG* across Pan-Cancer. **A.** The University of ALabama at Birmingham CANcer data (UALCAN) database determined the expression profile of *NCAPG*, with red boxes indicating its expression level in cancer and blue boxes indicating its expression level in normal. Differential expression of *NCAPG* in lung cancer LUAD (normal n = 59, tumor n = 515) (**B**), LUSC (normal n = 52, tumor n = 503) **C**), ONCODB LUAD (normal n = 59, tumor n = 540) (**D**), and LUSC (normal n = 51, tumor n = 503) (**E**), ENCORI, LUAD (normal n = 59, tumor n = 526) (**F**), and LUSC (normal n = 49, tumor n = 501) (**G**)

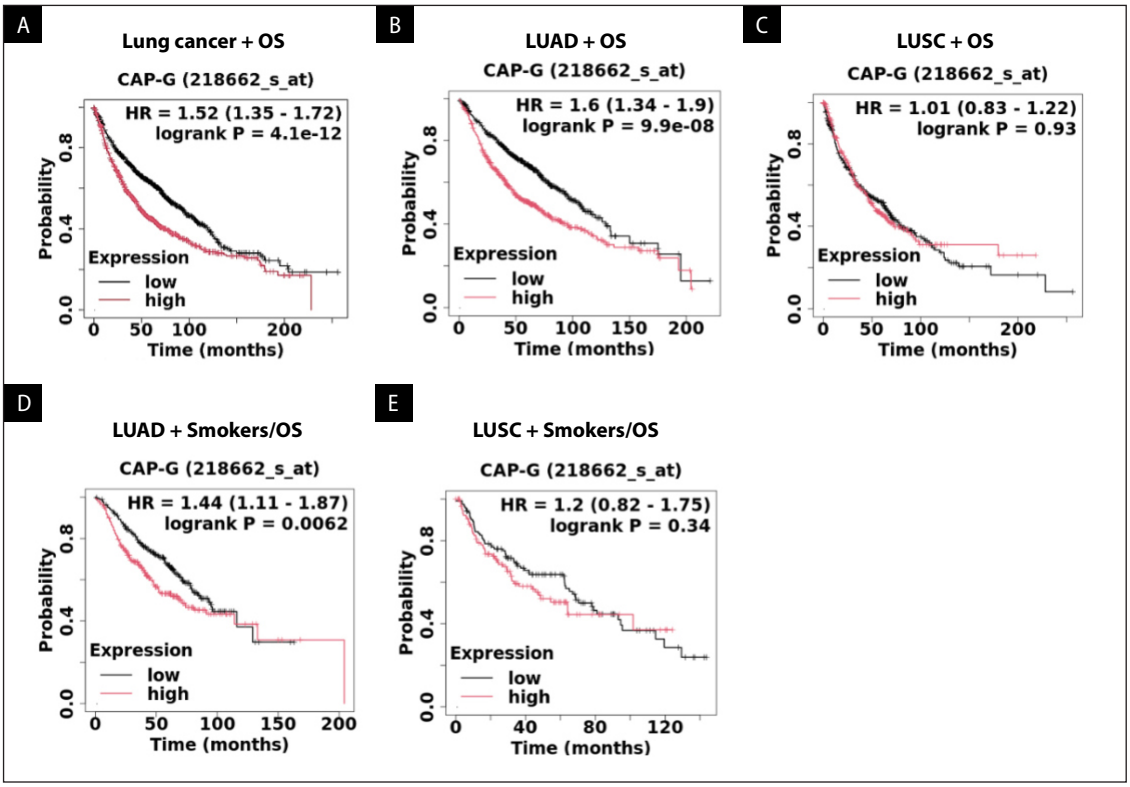

Figure 2. The study examine the prognostic role of mRNA expression of *NCAPG* in lung cancer patients using Kaplan-Meier survival curves for overall survival (OS) (n = 2166) (**A**), OS + Adenocarcinoma (LUAD) (n = 1161) (**B**), OS + Squamous cell carcinoma (LUSC) (n = 780) (**C**), OS + LUAD + Smoker (n = 546) (**D**), and OS + LUSC + Smoker (n = 244) (**E**)

Table 2. Survival analysis of *NCAPG/hsa-let-7b-5p/TMPO-AS1*

S.NO	Gene	Index	Patient number	Hazard ratio	CI	Log(P)	High expression cohort (months)	Low expression cohort (months)
1	*NCAPG*	OS	2166	1.52	1.35–1.72	4.1e–12	48	92.97
		FP	1252	1.99	1.68–2.37	1.8e–15	11	33.23
		PPS	477	1.13	0.91–1.38	0.26	11	15.83
		OS/LUAD	1161	1.6	1.34–1.90	9.9e–08	62	106
		FP/LUAD	906	1.99	1.62–2.45	1.5e–11	12	34.9
		PPS/LUAD	376	1.12	0.88–1.43	0.34	13.57	19
		OS/LUSC	780	1.01	0.83–1.22	0.93	51	62.47
		FP/LUSC	220	1.9	1.25–2.89	0.0021	9.37	35
		PPS/LUSC	51	1.22	0.68–2.19	0.49	7.82	9
		OS/LUAD/MALE	566	1.35	1.07–1.70	0.013	55	84
		OS/LUAD/FEMALE	537	1.93	1.46–2.56	3.3e–06	71	116
		OS/LUAD/SMOKER	546	1.44	1.11–1.87	0.0062	71	93
		OS/LUAD/NONSMOKER	192	1.62	0.88–2.96	0.12	56.5	76
		OS/LUAD/MALE/SMOKER	319	1.16	0.82–1.65	0.39	81	79
		OS/ LUAD/FEMALE/SMOKER	227	1.98	1.32–2.97	7e–04	49	116
2	*hsa-let-7b-5p*	OS/LUNG CANCER	504	0.71	0.53–0.95	0.021	54.4	42.17
3	*TMPO-AS1*	OS/ LUAD	672	2.16	1.69–2.76	4.1e–10	63	133.57
		OS/LUAD/SMOKER	231	2.34	1.41–3.88	0.00066	–	–

OS — overall survival; FP — free progression; PPS — post-progression survival; LUAD — lung adenocarcinoma; LUSC — lung squamous cell carcinoma

expressed in LUAD patients (p-values < 0.05) as listed in Supplementary File — Tab. S2. Next, we used the ENCORI database to look at the relationship between lncRNAs and NCAPG. We found that all the lncRNAs and NCAPG had a significant relationship, but TMPO-AS1 had a very strong positive relationship with the NCAPG gene (R = 0.575) in LUAD patients as shown in Supplementary File — Tab. S3 and Fig. 3A. Next, we used the KM plotter database to analyze TMPO-AS1 for survival status in LUAD patients and, surprisingly, we found that LUAD patients with high expression of TMPO-AS1 had a worse prognosis (HR = 2.16, CI = 1.69–2.76, p = 4.1e–10, Fc = 2.1). We further analyzed TMPO-AS1 with OS + LUAD + smoker patients (HR = 2.34, CI = 1.41–3.88, p = 0.00066), and we found that its overexpression was significantly associated with poor prognosis as shown in Figure 3B–C. Also, the expression profile analyzed using UALCAN database, as shown in Figure 3D, showed significant upregulation of TMPO-AS1 in tumor samples as compared to the normal.

To further investigate microRNAs, we created a TMPO-AS1-miRNA-NCAPG network us-

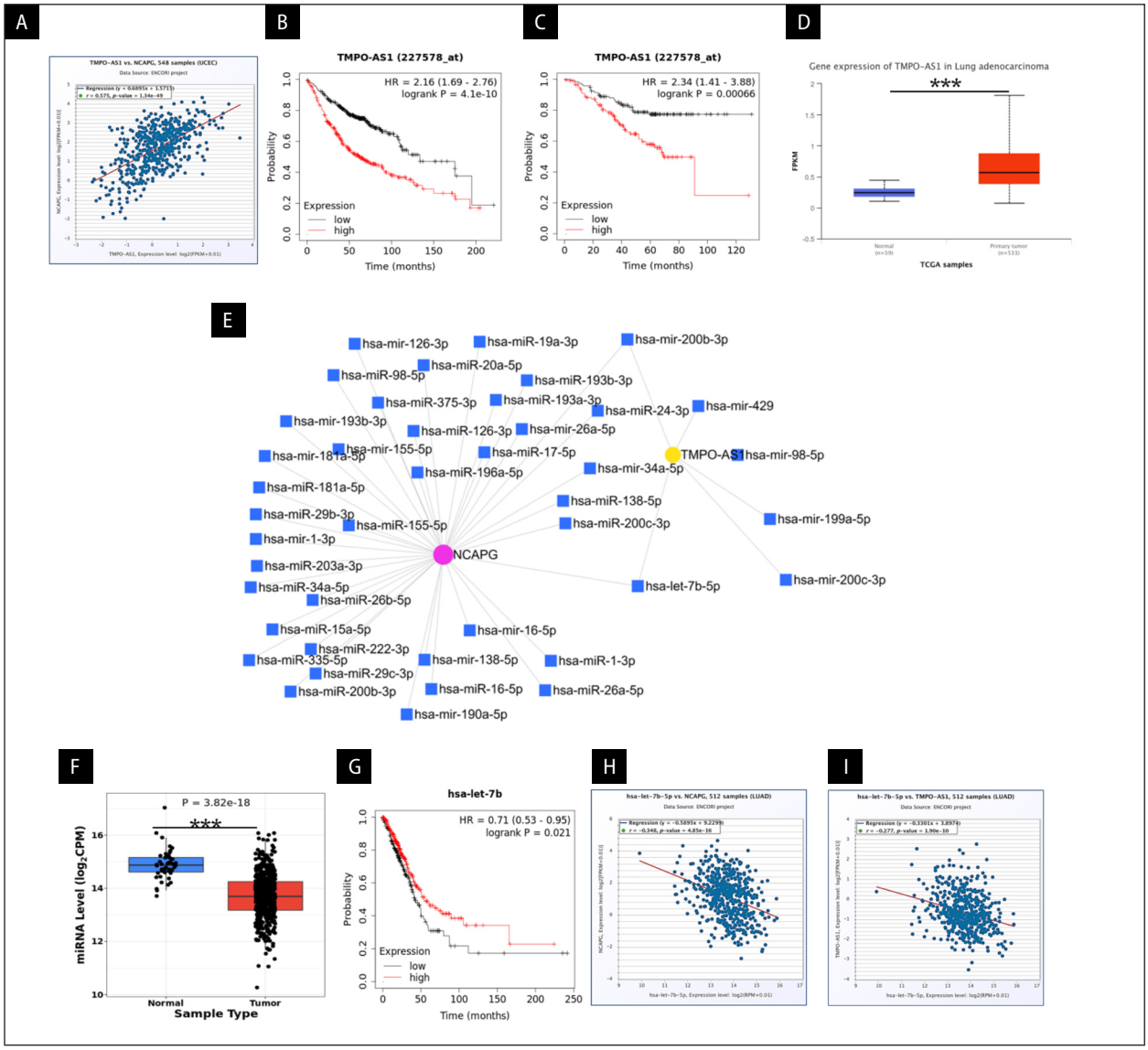

Figure 3. The study examines the correlation, survival status, and expression of miRNAs in tumor tissues from lung cancer patients, using various databases. **A.** Correlation between NCAPG and TMPO-AS1 using ENCORI Database. Survival analysis using KM plotter OS + LUAD (n = 1161) (**B**) and OS + LUAD + Smoker (n = 546) (**C**); **D.** Analyzes the differential expression of TMPO-AS1 in lung adenocarninoma patients using The University of ALabama at Birmingham CANcer data (UALCAN); **E.** Conducts a network analysis between *NCAPG*, miRNAs (hsa-let-7b-5p), and TMPO-AS1 using miRNet Database

ing the miRNet database, as shown in Figure 3E, and found that the miRNet topology, set to "concentric circle", was most suitable for miRNA associated with the NCAPG, showing hsa-let-7b-5p in proximity to NCAPG and TMPO-AS1. For prognostic purposes, the study used the CancerMIRNome and KM Plotter databases to determine miRNA expression and survival status. And we found that hsa-let-7b-5p is significantly down-expressed in lung cancer (p = 3.82e–18), and its down-regulation is also associated with poor survival (HR = 0.71, CI = 0.53-0.95, P = 0.021), as shown in Figure 3F–G. To add to that, we used the ENCORI database to find the relationships between hsa-let-7b-5p and NCAPG, along with hsa-let-7b-5p and TMPO-AS1. As shown in Figure 3H–I, hsa-let-7b-5p was negatively related to both NCAPG (R = –0.348) and TMPO-AS1 (R = –0.277) in LUAD. Based on the available data, we conclude that the TMPO-AS1/hsa-let-7b-5p/*NCAPG* feedback loop may contribute to the progression of lung adenocarcinoma in smokers.

Tumor infiltration of immune cells (TIICs) associated with NCAPG

The TISIDB database was used to analyze the close proximation of TIICs with the *NCAPG* gene expression in LUAD. A heatmap was retrieved containing various immune cells such as CD8 T cells, CD4 T cells, T follicular helper cells, NK cells, mast cells, monocytes, neutrophils, eosinophils, resting dendritic cells, and plasma cells. Interestingly, the data showed strong proximation between CD4 cells and *NCAPG* gene as highlighted in Figure 4A. The results suggested that an elevated CD4 immune cell infiltration could be associated with *NCAPG* overexpression, and correlation analysis using TISIDB (Rho = 0.632) and the TIMER 2.0 (R = 0.762) databases confirmed the strong positive association between NCAPG and CD4 + Th2 cells in lung adenocarcinoma (Fig. 4B–C). This association is crucial for determining cancer treatment efficacy and patient prognosis.

Discussion

Lung cancer is a complex disease with diverse cytogenetic and molecular abnormalities that impact clinical presentation, treatment decisions, and prognosis. The lack of robust predictive biomarkers and individual variations among patients hinder immunotherapy. This study aimed to determine the role of *NCAPG* in prognosis and elucidate its dysregulation in lung cancer. *NCAPG*, contributes to mitotic checkpoint alignment and chromosome partitioning during cell cleavage. The discovery of actionable oncogenic drivers, such as *EGFR*, *ALK*, *MET*, *KRAS*, *BRAF*, and *ROS1*, has revolutionized molecular targeted therapy for non-small cell lung cancer (NSCLC). However, these inhibitors only provide clinical benefits to patients with related driver gene mutations, who still face challenges with drug resistance [11]. Researchers used transcriptional sequencing and TCGA database analysis to identify NCAPG, a non-SMC subunit responsible for DNA supercoiling and chromosome segregation. NCAPG was the only differentially expressed gene that negatively correlated with the survival of NSCLC patients [12].

Researchers have found that several cancers, including breast, pancreatic ductal adenocarcinoma, prostate, and gastric cancers, overexpress *NCAPG* [13]. In addition, they have linked overexpression of the *NCAPG* protein to early tumor recurrence due to an elevated cell proliferation rate [14]. It can raise the expression of the MMP family in colon cancer through the PI3K/Akt pathway and lower the expression of *NCAPG* by lowering MMP2 and MMP9 [15, 16]non-SMC subunit in the concentrate I complex, might promote the proliferation of hepatocellular carcinoma (HCC. *NCAPG* expression is significantly higher in lung cancer tissue than in tissue surrounding the tumor [15, 17, 18]. Reducing *NCAPG* expression can stop lung cancer cells from growing, migrating, and invading [14]. This study examined the molecular mechanism behind *NCAPG* dysregulation in lung adenocarcinoma, as well as its potential as a prognostic biomarker for lung cancer smokers. A study that looked at the differences in *NCAPG* levels between lung adenocarcinoma and lung squamous cell carcinoma found a strong connection between *NCAPG* and lung squamous cell carcinoma. However, further survival analyses revealed a significant association between *NCAPG* overexpression and poor prognosis in LUAD patients, particularly those with a history of smoking. Aggressive lung adenocarcinoma states, including invasion, proliferation, and metastasis, are associated with *NCAPG*

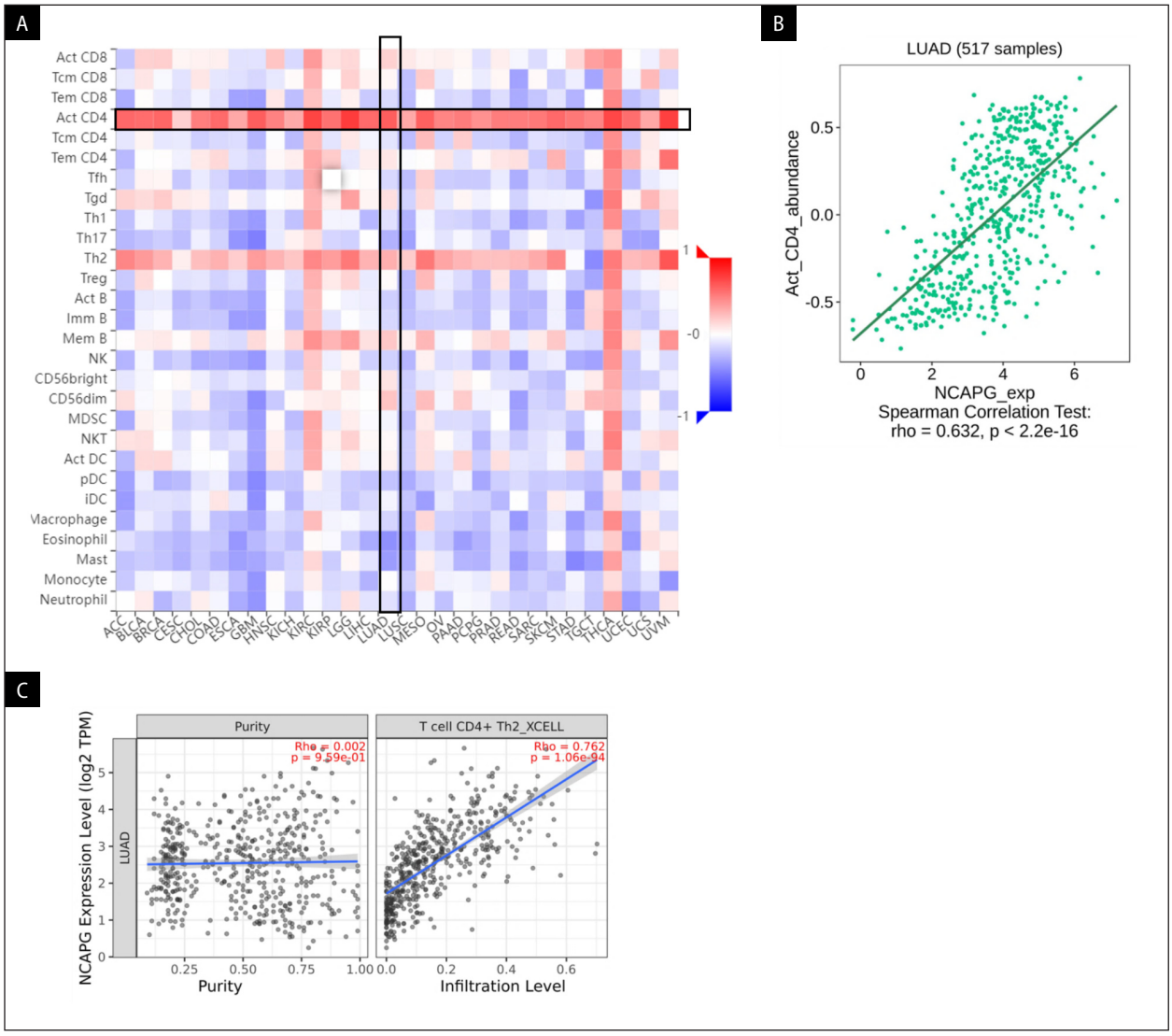

Figure 4. The study examines the tumor-infiltrating immuno cells. **A.** The Heat map shows the correlation between NCAPG and ACT_CD4 cells using TISIDB Database; **B.** The positive correlation of NCAPG and Act_CD4 cells using TISIDB Database; **C.** The positive correlation of NCAPG and Act_CD4 cells using (**B**) TIMER2.0 Database

overexpression. This knowledge provides a better understanding of how to target *NCAPG* in lung cancer treatments. The study analyzed the role of microRNAs and long non-coding RNAs in regulating *NCAPG* expression through the ceRNA network. Top five lncRNAs associated with *NCAPG* were studied and only TMPO-AS1 was found to be in strong proximation with the gene. Previous studies have shown TMPO-AS1 upregulation associated with poor prognosis in several cancer types [19–21]. Similarly, in this study upregulation of TMPO-AS1 was associated with a poor survival status in LUAD smoker patients. Several miRNAs were linked to *NCAPG*, with hsa-let-7b-5p being the key miRNA that negatively correlated with *NCAPG* expression. According to previous literature, the downregulation of hsa-let-7b-5p was responsible for poor survival status in various cancers [22–24]. Corroborating the previous studies, we found that downregulation of hsa-let-7b-5p was associated with a poor prognosis in lung cancer. TMPO-AS1, a long noncoding RNA, had a positive relationship with *NCAPG* expression and a negative relationship with hsa-let-7b-5p. This suggests that it may control *NCAPG* expression in LUAD. This ceRNA network dysregulation was associated with adverse clinical outcomes, emphasizing its significance in lung adenocarcinoma and smokers' prognosis. Targeting *NCAPG* in lung cancer treatments is crucial.

Conclusion

The study reveals that patients with lung adenocarcinoma and smoking history have a poor prognosis due to increased *NCAPG* expression, which is regulated by lncRNA TMPO-AS1 and negatively correlated with miRNA hsa-let-7b-5p. High levels of miRNA hsa-let-7b-5p may reduce *NCAPG* expression, potentially improving LUAD and smokers' prognosis. Targeting miRNA hsa-let-7b-5p could potentially inhibit tumor growth and improve patient outcomes.

Conflicts of interest

The authors declare that they have no competing interests.

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Ethics statement

This study does not require ethical approval.

Data availability statement

The data that support the findings of this in silico analysis are available from the corresponding author upon request.

Author contribution

R.N.: Conception, study design, critical reading, intellectual assessment of the manuscript, and preparation of the manuscript. P.V.: Conception, study design, preparation of the manuscript. S.N.: Conception, study design, preparation of the manuscript.

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